California Air Resources Board

PUBLIC MEETING AGENDA

March 17th, 2005

9:00 a.m.

Agenda Items to be heard;
05-3-1, 05-3-2, 05-3-3, 05-3-4

Visit our Web Site
@ www.arb.ca.gov

Includes
AcrobatTM
ReaderTM

PC and Mac
Compatible

ELECTRONIC BOARD BOOK

California Environmental Protection Agency

Air Resources Board

LOCATION:
Air Resources Board
Byron Sher Auditorium, Second Floor
1001 I Street
Sacramento, California 95814

PUBLIC MEETING AGENDA

This facility is accessible by public transit. For transit information, call (916) 321-BUSS, website: http://www.sacrt.com
(This facility is accessible to persons with disabilities.)

March 17, 2005
9:00 a.m.

Agenda Item #

05-3-1: Report to the Board on a Health Update: Ozone Exposure and Short-Term Mortality - Two Recent Studies in the United States and Europe

Staff will present the results of two recent studies that have found an association between short-term exposure to ozone (daily and weekly) and mortality. These studies, one conducted in the US and the other in Europe, showed that an increase in the ambient ozone concentration was associated with an increase in daily mortality, and more specifically with increases in cardiovascular and respiratory deaths.

05-3-2: Board Meeting to Consider (5) Research Proposals

1. *"Ventilation and Indoor Air Quality in New Homes," Indoor Environmental Engineering, Proposal No. 2575-247.*
2. *"Development and Demonstration of an Aerosol Tracer Technique Based on Neutron Activation Analysis for Studying Cyclical Deposition and Resuspension of Aerosol-Associated Toxic Contaminants," University of California, Los Angeles, Proposal No. 2579-247.*
3. *"The Development of Exhaust Speciation Profiles for Commercial Jet Engines," University of Michigan, Proposal No. 2565-246.*
4. *"Dairy Operations: An Evaluation and Comparison of Baseline and Potential Mitigation Practices for Emissions Reduction in the San Joaquin Valley," California State University, Fresno, Proposal No. 2582-247.*
5. *"Hourly In-Situ Quantification of Organic Aerosol Marker Compounds," University of California, Berkeley, Contract No. 03-324.*

05-3-3: Report to the Board on the Draft Report to the Legislature: Indoor Air Pollution in California

As required under legislation enacted in 2002 (AB 1173, Keeley), staff have prepared a comprehensive report on indoor air pollution in California. Current scientific studies show that indoor sources of pollutants contribute significantly to serious health impacts including asthma, cancer risk, premature death, increased heart and lung disease, irritant effects, and infectious disease. The quantifiable costs of indoor pollution in California are estimated at $45 billion. While there are a number of regulations and guidelines that address some aspect of indoor pollution, no state or federal agency has a clear mandate and authority to fully and directly reduce indoor air pollution. Alternatives to high-emitting indoor sources are available. Several mitigation approaches are suggested, including indoor source emission limitations and a comprehensive program to reduce indoor emissions.

05-3-4: Report to the Board on an Emergency Regulation: Availability of Stationary Engines (less than 175 hp) Meeting the 0.15 g/bhp-hr Particulate Matter (PM) Standard

Staff is currently investigating concerns related to the implementation of the Stationary Engine ATCM and may propose amendments to the ATCM for issues that have come up concerning new Stationary Diesel Engine availability at the Board's March meeting. This ATCM became effective December 8, 2004, and implementation began on January 1, 2005. Some engine and equipment distributors and dealers have expressed concerns over the availability of new engines that are less than 175 hp that meet a 0.15 g/bhp-hr PM standard. Staff's presentation will provide background information and staff's proposal.

TO SUBMIT WRITTEN COMMENTS ON AN AGENDA ITEM IN ADVANCE OF THE MEETING:

CONTACT THE CLERK OF THE BOARD, 1001 I Street, 23rd Floor, Sacramento, CA 95814 (916) 322-5594
FAX: (916) 322-3928
ARB Homepage: www.arb.ca.gov

To request special accommodation or language needs, please contact the following:

- TTY/TDD/Speech-to-Speech users may dial 7-1-1 for the California Relay Service.
- Assistance for Disability-related accommodations, please go to http://www.arb.ca.gov/html/ada/ada.htm or contact the Air Resources Board ADA Coordinator, at (916) 323-4916.
- Assistance in a language other than English, please go to http://www.arb.ca.gov/as/eeo/languageaccess.htm or contact the Air Resources Board Bilingual Coordinator, at (916) 324-5049.

OPEN SESSION TO PROVIDE AN OPPORTUNITY FOR MEMBERS OF THE PUBLIC TO ADDRESS THE BOARD ON SUBJECT MATTERS WITHIN THE JURISDICTION OF THE BOARD.

Although no formal Board action may be taken, the Board is allowing an opportunity to interested members of the public to address the Board on items of interest that are within the Board's jurisdiction, but that do not specifically appear on the agenda. Each person will be allowed a maximum of five minutes to ensure that everyone has a chance to speak.

THE AGENDA ITEMS LISTED ABOVE MAY BE CONSIDERED IN A DIFFERENT ORDER AT THE BOARD MEETING.

SMOKING IS NOT PERMITTED AT MEETINGS OF THE CALIFORNIA AIR RESOURCES BOARD

California Environmental Protection Agency

Air Resources Board

LOCATION:
Air Resources Board
Byron Sher Auditorium Second Floor
1001 I Street
Sacramento, California 95814

PUBLIC MEETING AGENDA

INDEX

This facility is accessible by public transit. For transit information, call (916) 321-BUSS, website: http://www.sacrt.com
(This facility is accessible to persons with disabilities.)

March 17, 2005
9:00 a.m.

		Pages
05-3-1	Report to the Board on a Health Update - Ozone Exposure and Short-Term Mortality - Two Recent Studies in the United States and Europe	---
05-3-2	Board Meeting to Consider (5) Research Proposals	---
05-3-3	Report to the Board on the Draft Report to the Legislature: Indoor Air Pollution in California	1 - 334
05-3-4	Report to the Board on an Emergency Regulation: Availability of Stationary Engines (less than 175 hp) Meeting the 0.15 g/bhp-hr Particulate Matter (PM) Standard	---

CALIFORNIA AIR RESOURCES BOARD

NOTICE OF PUBLIC MEETING TO CONSIDER A REPORT TO THE LEGISLATURE ON INDOOR AIR POLLUTION IN CALIFORNIA

The Air Resources Board (the Board or ARB) will conduct a public meeting at the time and place noted below to consider a Report to the Legislature on Indoor Air Pollution in California.

DATE: **March 17, 2005**

TIME: **9:00 AM**

PLACE: **Air Resources Board**
Byron Sher Auditorium
1001 I Street
Sacramento, CA 95814

This item will be considered at a regular meeting of the Board, which will commence at 9:00 a.m., March 17, 2005, and may continue at 8:30 a.m., March 18, 2005. This item may not be considered until March 18, 2005. Please consult the agenda for the meeting, which will be available at least 10 days before March 17, 2005, to determine the day on which this item will be considered.

If you have a disability-related accommodation need, please go to http://www.arb.ca.gov/html/ada/ada.htm for assistance or contact the ADA Coordinator at (916) 323-4916. If you are a person who needs assistance in a language other than English, please contact the Bilingual Coordinator at (916) 324-5049. TTY/TDD/Speech-to-Speech users may dial 7-1-1 for the California Relay Service.

The Air Resources Board (ARB) staff prepared the report to the Legislature on Indoor Air Pollution in California (Report) to satisfy requirements of Assembly Bill 1173 (stats. 2002, Chapter 987; Health and Safety Code Section 39930). As required by this legislation, the Report summarizes the best scientific information available on indoor air pollution, including: common indoor pollutants and their sources; the potential health impacts of indoor pollutants and associated costs; existing regulations and current industry practices; options for mitigation in schools, non-industrial workplaces, homes, and other indoor locations; and state and federal efforts related to control of biological and radiological substances.

In preparing the Report, the ARB consulted with the Department of Health Services, the Office of Environmental Health Hazard Assessment, the California Energy Commission, the Department of Industrial Relations (Cal/OSHA), other agencies, industry, and interested members of the public. Two public review periods and two public workshops

were held to receive comments from stakeholders on the draft Report. A University of California scientific peer review panel also reviewed the draft Report.

Indoor pollution causes substantial, avoidable health effects–ranging from irritant effects to asthma, cancer, and premature death–and costs Californians at least $45 billion each year. Exposure and the associated risk is substantial because there are numerous sources of pollutants in indoor environments, and because people spend most of their time indoors. Many agencies, professional groups, and organizations have taken actions to prevent or reduce indoor pollution, but these have been piecemeal and are not sufficiently effective in preventing indoor air pollution problems.

There are many actions that could be taken to significantly reduce indoor emissions and exposure. The Report discusses these actions and makes recommendations. A comprehensive risk reduction program is needed to effectively assure acceptable indoor air quality in California homes, schools, and public buildings. A program that stresses direct emission reductions but includes improved building codes and ventilation, increased public and professional education, and advisory guidelines is recommended. High priority source categories recommended for mitigation include ozone-generating indoor air cleaning devices, biological contaminants, building materials and furnishings, combustion appliances including woodstoves and fireplaces, environmental tobacco smoke, and radon. Other sources of concern, but of a lower priority, include architectural coatings, consumer products, household and office equipment, and pesticides. Special priority is recommended for measures that reduce children's exposures.

ARB staff will present the Report at the meeting. Copies of the report may be obtained from the Board's Public Information Office, 1001 "I" Street, 1st Floor, Visitors and Environmental Services Center, Sacramento, CA 95814, (916) 322-2990, on or after February 28, 2005. The report and additional information may also be obtained from ARB's internet site at http://www.arb.ca.gov/research/indoor/ab1173/ab1173.htm.

Interested members of the public may present comments relating to this matter orally or in writing at the meeting, and in writing or by e-mail before the meeting. To be considered by the Board, written comments not physically submitted at the meeting must be received **no later than 12:00 noon, Wednesday, March 16, 2005**, and addressed to the following:

Postal mail is to be sent to:

Clerk of the Board
Air Resources Board
1001 I Street
Sacramento, CA 95814

Electronic mail should be sent to indoorap@listserv.arb.ca.gov and received at the ARB **no later than 12:00 noon, March 16, 2005.**

Facsimile transmissions are to be transmitted to the Clerk of the Board at 916) 322-3928 and received at the ARB **no later than 12:00 noon, March 16, 2005**.

The Board requests, but does not require 30 copies of any written statement be submitted and that all written statements be filed at least 3 days prior to the hearing, so that ARB staff and Board members have time to fully consider each comment. Further inquiries regarding this matter should be directed to Mr. Richard Bode, Chief, Health and Exposure Assessment Branch, (916) 323-8413, or Ms. Peggy Jenkins, Manager, Indoor Exposure Assessment Section, (916) 323-1504.

CALIFORNIA AIR RESOURCES BOARD

Catherine Witherspoon
Executive Officer

Date: March 1, 2005

The energy challenge facing California is real. Every Californian needs to take immediate action to reduce energy consumption. For a list of simple ways you can reduce demand and cut your energy costs, see our Web-site at www.arb.ca.gov.

California Environmental Protection Agency

Air Resources Board

DRAFT FOR BOARD REVIEW

Report to the California Legislature

INDOOR AIR POLLUTION IN CALIFORNIA

A report submitted by:

California Air Resources Board

Pursuant to Health and Safety Code § 39930
(Assembly Bill 1173, Keeley, 2002)

February 2005

Arnold Schwarzenegger
Governor

ACKNOWLEDGEMENTS

This report was prepared with the able and dedicated support from Jacqueline Cummins, Marisa Bolander, and Jeania Delaney.

We appreciate the valuable input received from the following groups:

- Many government agency representatives who provided information and thoughtful comments on draft reports, especially Jed Waldman, Sandy McNeel, Janet Macher, and Feng Tsai, Department of Health Services; Richard Lam and Bob Blaisdell, Office of Environmental Health Hazard Assessment; Deborah Gold and Bob Nakamura, Cal/OSHA; Bill Pennington and Bruce Maeda, California Energy Commission; Dana Papke and Kathy Frevert, California Integrated Waste Management Board; Randy Segawa, Department of Pesticide Regulation; and many others.

- Bill Fisk, Lawrence Berkeley National Laboratory, for assistance in assessing the costs of indoor pollution.

- Stakeholders from the public and private sectors, who attended the public workshops and shared their experiences and suggestions concerning indoor air quality in California.

- Susan Lum, ARB, project website management, and Chris Jakober, for general technical assistance.

DISCLAIMER

The mention of commercial products, their source, or their use in connection with material presented in this report is not to be construed as actual or implied endorsement of such products by the State of California.

ALTERNATIVE FORMS OF REPORT

If you are a person with a disability and desire to obtain this document in an alternative format, please contact Jacqueline Cummins at (916) 445-0753 or jcummins@arb.ca.gov. TTY/TDD/Speech-to-speech users may dial 7-1-1 for the California Relay Service.

This report is also available electronically on ARB's website at:
http://www.arb.ca.gov/research/indoor/ab1173/ab1173.htm

AUTHORS

Principal Authors

Dorothy Shimer, M.S.
Staff Air Pollution Specialist
Thomas J. Phillips, M.S.
Staff Air Pollution Specialist
Peggy L. Jenkins, M.S., Manager
Indoor Exposure Assessment Section

Contributing Authors

Susan Lum, M.A.
Whitney Webber, M.S., candidate
Michael Robert, Ph.D. candidate
Lori Miyasato, Ph.D. candidate
Deborah Drechsler, Ph.D.
Nancy Hughett, M.S.
Jed Waldman, Ph.D.
Sandra McNeel, DVM

Report Reviewers

Peggy Jenkins, Manager, Indoor Exposure Assessment Section
Richard Bode, Chief, Health and Exposure Assessment Branch
Bart Croes, P.E., Chief, Research Division
Michael Scheible, Deputy Executive Officer
Catherine Witherspoon, Executive Officer

Scientific Peer Reviewers

Kirk R. Smith, Ph.D., MPH
School of Public Health, Environmental Health Sciences Division, University of California at Berkeley
S. Katharine Hammond, Ph.D.
School of Public Health, Environmental Health Sciences Division, University of California at Berkeley
William W. Nazaroff, Ph.D.
Civil and Environmental Engineering, University of California at Berkeley
Richard Sextro
Lawrence Berkeley National Laboratory, Indoor Environment Program

TABLE OF CONTENTS

ACKNOWLEDGEMENTS iii
DISCLAIMER iv
ACRONYMS viii
ABBREVIATIONS AND SYMBOLS x
EXECUTIVE SUMMARY 1
1. INTRODUCTION AND BACKGROUND 28
1.1 Indoor Pollution Poses a Significant Health Risk 28
1.2 People Spend Most of Their Time Indoors 30
1.3 Children's Health – Vulnerability Implies Increased Indoor Air Quality Impact 30
1.4 Environmental Justice Considerations Relative to Indoor Air Quality 31
2. HEALTH EFFECTS, SOURCES AND CONCENTRATIONS OF INDOOR AIR POLLUTANTS 33
2.1 Key Health Impacts 33
2.2 Traditional (Criteria) Pollutants 43
2.3 Toxic Air Contaminants and Other Indoor Air Pollutants 61
3. COSTS OF INDOOR AIR POLLUTION 109
3.1 Premature Death 109
3.2 Medical Costs 114
3.3 Productivity Costs of Indoor Air Pollution 120
3.4 Summary of Estimated Costs 121
3.5 Benefits of Improving Indoor Air Quality 122
4. EXISTING REGULATIONS, GUIDELINES, AND PRACTICES 125
4.1 Standards and Regulations 125
4.2 Indoor Air Quality Guidelines 129
4.3 Emission Limits 132
4.4 Building Design and Construction Standards and Guidelines 143
4.5 Building Operation and Maintenance Standards 151
5. METHODS TO PREVENT AND REDUCE INDOOR AIR POLLUTION 155
5.1 Source Control 155
5.2 Ventilation 156
5.3 Building Operation and Maintenance 159
5.4 Public and professional Education 161
5.5 Air Cleaning Devices 162
6. PRIORITIZATION OF SOURCES AND POLLUTANTS BASED ON EXPOSURE AND ADVERSE IMPACTS 165
6.1 High Ranked Source Categories 167
6.2 Medium Ranked Source Categories 169
7. OPTIONS TO MITIGATE INDOOR AIR POLLUTION 173
7.1 General Mitigation Options 173
7.2 Solutions for Schools 175
8. SUMMARY 180
9. REFERENCES 182
10. GLOSSARY 231

APPENDICES

I. Assembly Bill 1173 Text
II. Explanation of Indoor Cancer Risk Estimates
III. Background for Estimates of Indoor Formaldehyde Concentrations
IV. Responses to comments from the scientific peer review committee.
V. Responses to comments from the public.

LIST OF FIGURES

ES-1: Where Californians Spend Time 2
ES-2: Estimated Potential Cancer Burden from Air Toxics in California by Source 5
2.1: Estimated Potential Cancer Burden from Air Toxics in California by Source 40
2.2: Causes of CO Deaths in California 54
2.3: Annual California CO Cases 55
2.4: California Formaldehyde Levels 65
2.5: Statewide Indoor and Outdoor Concentrations of VOCs 74
5.1: Relationship of Ventilation and Indoor Air Pollutant Levels 157

LIST OF TABLES

ES-1. Sources and Potential Health Effects of Major Indoor Air Pollutants 3
ES-2. Summary of Estimated Costs of Some Indoor Air Pollution in California 12
ES-3.1. High Priority Source Categories for Mitigation 18
ES-3.2. Medium Priority Source Categories for Mitigation 22
1.1. Average Percent of Time Californians Spend in Major Locations 30
2.1. Sources and Potential Health Effects of Major Indoor Air Pollutants 34
2.2. Indoor Exposures and Exacerbation of Asthma 36
2.3. Indoor Exposures and Development of Asthma 37
2.4. Carcinogenic Status of Selected Indoor Air Pollutants 39
2.5. Summary of Recent Indoor PM Exposure Studies 52
2.6. Toxic Air Contaminants Emitted From Building and Finishing Materials 70
2.7. Personal, Indoor, and Outdoor VOC Concentrations from a Northern California Study 73
2.8. Current Estimated Attributable Risks Associated with ETS 78
2.9 Estimates of Current California Indoor Concentrations of Nicotine and RSP 81
2.10. Health Outcomes and Indoor Dampness 85
2.11. Presence of Indoor Mold and Health Outcomes 87
2.12. Comparison of Indoor-air, Personal-air, and House-dust Levels in U.S. 91
2.13. California Portable Classrooms Study Results for Lead in Floor Dust 93
2.14. Sources of Mercury in the Residential Environment 94
2.15. Recommended Airborne Concentrations of Mercury 95
3.1. Unit Costs for Health Effects 110
3.2. Estimated Valuation of Mortality Attributed to Indoor Air Pollution in California 112
3.3. Estimated Annual Medical Costs of Indoor Air Pollution in California 117
3.4. Fraction of Asthma Attributable to Mold or Dampness in Houses 119
3.5. Updated Estimates of Annual Cost of Asthma 120
3.6. Summary of Estimated Costs of Some Indoor Air Pollution in California 123
4.1. ARB Indoor Air Quality Guidelines 130
4.2. Minimum Design Ventilation Rates for Nonresidential, High-Rise Residential, and Hotel Buildings, 2001 Title 24 Standards 146
6.1. High Priority Source Categories for Mitigation 166
6.2. Medium Priority Source Categories for Mitigation 171

ACRONYMS

ACRONYM	DEFINITION
AAQS	ambient air quality standards
AB	Assembly Bill
ACM	asbestos containing material
AHERA	Asbestos Hazard Emergency Response Act
ANSI	American National Standards Institute
ARB	California Air Resources Board
ASHRAE	American Society of Heating, Refrigerating, and Air-conditioning Engineers
CADR	clean air delivery rate
Cal/EPA	California Environmental Protection Agency
Cal/OSHA	California Occupational Safety and Health Program, which consists of several agencies within the Department of Industrial Relations, including the Division of Occupational Safety and Health and the Occupational Safety and Health Standards Board
CCR	California Code of Regulations
CDC	U.S. Center for Disease Control
CDE	California Department of Education
CDFA	California Department of Food and Agriculture
CEC	California Energy Commission
CHPS	Collaborative for High Performance Schools
CIWMB	California Integrated Waste Management Board
COPD	chronic obstructive pulmonary disease
CPA	Composite Panel Association
CRI	Carpet and Rug Institute
DGS	California Department of General Services
DHS	California Department of Health Services
DOE	U.S. Department of Energy
DOF	California Department of Finance
EC	elemental carbon
EPA	U.S. Environmental Protection Agency
HAP	hazardous air pollutant
HEPA	high efficiency particulate arrestance
HSC	California Health and Safety Code
HVAC	heating, ventilating, and air conditioning
IAQ	indoor air quality
IDEC	indirect-direct evaporative cooling
IEQ	indoor environmental quality
IESNA	Illumination Engineering Society of North America
IREL	interim (Indoor) reference exposure level
LAUSD	Los Angeles Unified School District
LEED™	Leadership in Energy and Environmental Design
MCL	maximum contaminant level
MDI	methyl diisocyanate
NHEXAS	National Human Exposure Assessment Survey
NO_2	nitrogen dioxide
NO_3	particle nitrate
NO_X	oxides of nitrogen

NOEL	no observable effect level
O&M	operation and maintenance
OEHHA	California Office of Environmental Health Hazard Assessment
OP	organophosphate pesticide
OR	odds ratio
OSHA	U.S. Occupational Safety and Health Administration
PACM	presumed asbestos containing material
PAH	polycyclic aromatic hydrocarbon
PBDE	polybrominated diphenyl ethers
PBO	piperonyl butoxide
PCB	polychlorinated biphenyls
PCS	Portable Classrooms Study
PEL	permissible exposure limit
PM	particulate matter
PM10	particulate matter smaller than 10 microns in aerodynamic diameter
PM2.5	particulate matter smaller than 2.5 microns in aerodynamic diameter
PTEAM	Paritcle Total Expousre Assessment Methodology
REL	reference exposure limit
RH	relative humidity
RSP	respirable suspended particulate
SB	Senate Bill
SBS	sick building syndrome
SCSA	State and Consumer Services Agency
SO_2	sulfur dioxide
TVOC	total volatile organic compounds
U.S. EPA	United States Environmental Protection Agency
USD	Unified School District
VOC	volatile organic compound
WHO	World Health Organization

ABBREVIATIONS AND SYMBOLS

TERM	DEFINITION
°C	degrees Celsius
cfm	cubic feet per minute
CFU	colony forming unit
cm^2	square centimeter
CO	carbon monoxide
CO_2	carbon dioxide
dBA	decibel (referenced to 1 ampere)
°F	degrees Fahrenheit
kg	kilogram (one thousand grams)
l/min	liters per minute (flow rate)
m^2	square meter
m^3	cubic meter
µg	microgram (one-millionth of a gram)
µg/g	micrograms per gram (concentration)
$\mu g/cm^2$	micrograms per square centimeter (surface density)
$\mu g/m^3$	micrograms per cubic meter (concentration)
mg	milligrams (one-thousandth of a gram)
mg/kg	milligrams per kilogram (concentration)
ml	milliliter (one-millionth of a liter)
ng	nanogram (one-billionth of a gram)
ng/g	nanograms per gram (concentration)
No.	number
%	percent
pCi/l	picoCurie per liter (a measure of radioactivity)
PM2.5	particulate matter with aerodynamic diameter less than 2.5 microns
PM10	particulate matter with aerodynamic diameter less than 10 microns
ppb	parts per billion (such as one grain of sand in a billion grains of sand)
ppm	parts per million (such as one grain of sand in a million grains of sand)
§	section
T	temperature
tpd	tons per day

EXECUTIVE SUMMARY

I. INTRODUCTION

The California Air Resources Board (ARB) staff prepared this report to the Legislature on indoor air quality in response to requirements of Assembly Bill 1173 (Keeley, 2002; California Health and Safety Code [HSC] Section [§] 39930). This report summarizes the best scientific information available on indoor air pollution, including: information on common indoor pollutants and their sources; the potential health impacts of indoor pollutants, and associated costs; existing regulations and practices; options for mitigation in schools, homes, and non-industrial workplaces; and other information specified in the legislation. Stakeholder input was obtained from relevant state agencies, industries, interest groups, and the public. The report was also reviewed by a panel of University of California scientists with expertise in various aspects of indoor air quality and air pollution exposure.

Indoor Air Pollution Poses Substantial Health Risks

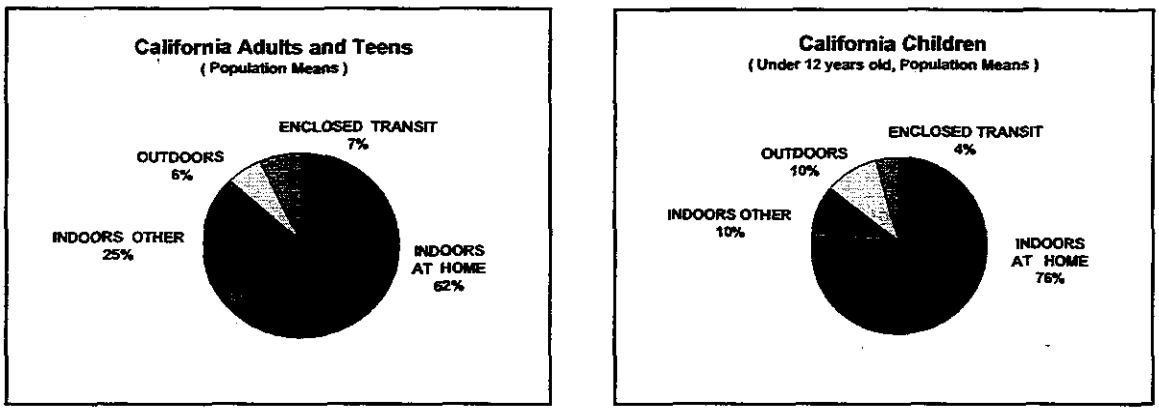
Available scientific information indicates that indoor air pollution poses substantial health risks in many indoor environments. In comparative risk projects that ranked environmental health problems in order of the risk they pose to health and the environment, both the California and U.S. Environmental Protection Agencies ranked indoor pollutants and sources in the high-risk categories. Outdoor pollution emissions from motor vehicles and stationary (industrial) sources were also ranked high. Indoor pollution ranked high relative to other environmental problems because there are numerous sources of pollutants indoors, indoor air concentrations of some pollutants often occur at levels that create significant health risks, and people spend most of their time indoors. While regulation of outdoor sources such as motor vehicles and industrial facilities is very extensive and has notably reduced pollutant levels in California, indoor pollution sources have not been addressed in a comprehensive manner. If such an effort were established, significant gains could be achieved in public health protection from reductions in indoor source emissions and from other measures that might be taken to reduce indoor concentrations and exposures.

Why Indoor Sources Have Such a Significant Impact

The total quantity of air pollutants emitted indoors is less than that emitted by outdoor sources. However, once emitted, indoor air pollutants are diluted much more slowly, due to the partial trapping effect of the building shell. Additionally, indoor emissions occur in closer proximity to people: Californians, like others from industrialized nations, spend most of their time indoors. As shown in Figure ES-1, California adults spend an average of 87 percent of their time indoors, and children under 12 years of age spend about 86 percent of their time indoors. Most of the time spent indoors is spent in the home. However, working adults spend about 25 percent of their time at other indoor locations such as office buildings, stores, and restaurants, primarily for work, and children spend about 21 percent of their time in school on a school day. Senior individuals spend a great deal of time in their homes. Because of these time budgets, the trapping effect of buildings, and people's proximity to indoor sources of emissions, there is a much higher likelihood that people will be exposed to indoor pollutants than outdoor pollutants. Investigators have calculated that pollutants emitted indoors have a 1000-fold greater chance of being inhaled than do those emitted outdoors (Smith, 1988; Bennet *et al.*, 2002; Lai *et al.*, 2000).

Homes and schools are thus critical exposure microenvironments, especially for children and seniors. These groups are more sensitive to the adverse effects of some pollutants, and spend most of their time indoors. The passenger compartments of cars and buses also are key exposure environments: studies have shown very high levels of vehicle exhaust pollutants inside cars and school buses as they travel along California roadways. However, these environments differ significantly from building environments and are more closely associated with outdoor pollution, and are not considered further in this report.

Figure ES-1: Where Californians Spend Time
(Jenkins *et al.*, 1992a; Phillips *et al.*, 1991)



Children Are Especially Vulnerable to Poor Indoor Air Quality

Children may be especially vulnerable to poor indoor air quality due to several factors. Children's physiology and developing bodies make them more susceptible to chemicals that affect development and lung function. Their immune systems are not fully developed, and their growing organs are more easily harmed. Additionally, infants and children inhale more air relative to their size than do adults at a given level of activity, so that they inhale a larger dose of pollutants than do adults in the same environment. Children also tend to be more active than adults. These factors, combined with elevated indoor concentrations of pollutants, can lead to higher exposure and risk for children than adults.

II. HEALTH EFFECTS OF INDOOR POLLUTANTS

Indoor air pollution can cause a variety of impacts on human health, from irritant effects to respiratory disease, cancer, and premature death. Indoor air pollutants can be elevated to levels that may result in adverse health effects. The major indoor pollutants that can have a substantial impact on Californians' health are listed in Table ES-1, along with their sources and associated health impacts. The health impacts of greatest significance include asthma, cancer, premature death, respiratory disease and symptoms, and irritant effects.

Modern society includes many trade-offs, often characterized through risk/benefit analyses. Transportation, building materials, appliances, consumer products, plastics, and pesticides

Table ES-1. Sources and Potential Health Effects of Major Indoor Air Pollutants

POLLUTANT	MAJOR INDOOR SOURCES	POTENTIAL HEALTH EFFECTS ASSOCIATED WITH ONE OR MORE OF THE POLLUTANTS LISTED*
Asbestos	Building materials in older homes released during renovation, naturally occurring in some soils	Lung cancer, asbestosis, mesothelioma
Biological Agents (bacteria, fungi, viruses, house dust mites, animal dander; cockroaches, microbial VOCs)	House and floor dust; pets; bedding; poorly maintained air- conditioners, humidifiers, dehumidifiers; moist structures or furnishings; insect infestation; building occupants	Allergic reactions; asthma; eye, nose, and throat irritation; humidifier fever, influenza, and other infectious diseases
Carbon Monoxide	Unvented or malfunctioning gas and propane appliances, wood stoves, fireplaces, tobacco smoke, motor vehicles in attached garages	Headache; nausea; angina; impaired vision and mental functioning; fatal at high concentrations
Endocrine Disruptors (phthalates; DDT, chlordane, heptachlor, o-phenylphenol; PBDEs)	Plastics; pesticides; flame retardants	Mimic or block natural effects of hormones (estrogen and others); developmental abnormalities
Environmental Tobacco Smoke (ETS)	Cigarettes, cigars, and pipes	Respiratory irritation, bronchitis and pneumonia in children; asthma in preschool children; lung cancer; heart disease; aggravated asthma, decreased lung function
Formaldehyde, Other Aldehydes	Composite wood products such as plywood and particleboard; furnishings; wallpaper; durable press fabrics; paints; combustion appliances; tobacco smoke	Cancer; eye, nose, and throat irritation; headache; allergic reactions; aggravated asthma, decreased lung function
Lead	Lead paint chips, contaminated soil	Learning impairment
Nitrogen Dioxide	Unvented or malfunctioning gas appliances, other combustion appliances	Aggravated asthma, decreased lung function; eye, nose, and throat irritation; increased respiratory disease in children
Organic Chemicals (benzene, chloroform, para-dichlorobenzene, methylene chloride, perchloroethylene, phthalates, styrene, others)	Solvents; glues; cleaning agents; pesticides; building materials; paints; treated water; moth repellents; dry-cleaned clothing; air fresheners;	Cancer; eye, nose, throat irritation; aggravated asthma, decreased lung function; headaches; at high levels: loss of coordination; damage to liver, kidney and brain
Ozone	Infiltration of outdoor air, ozone generating air "purifiers", office machines	Lung inflammation, aggravated asthma, cough, wheeze, chest pain
Particulate Matter	Cigarettes, wood stoves, fireplaces, cooking, candles, aerosol sprays, house dust	Increased mortality and hospital admissions; lung cancer; eye, nose, throat irritation; increased susceptibility to sinus and respiratory infections; bronchitis; aggravated asthma, decreased lung function
Polycyclic Aromatic Hydrocarbons (PAH)	Cigarette smoke, cooking, woodburning	Cancer; gene mutation
Radon	Uranium-bearing soil under buildings, ground-water, construction materials	Lung cancer (especially in smokers)

* Please note that when multiple pollutants are listed in a group, each pollutant may not cause all of the health effects listed in the third column.

impart obvious benefits to society. However, it is noted that the use of many beneficial or desirable products at times have a down side – the emission of a variety of chemicals that can have an adverse impact on human health. The impact on health depends on the toxicological properties of the chemical and the exposure and absorbed dose an individual may receive.

Asthma

Asthma is a chronic inflammatory lung disease that results in constriction of the airways. Its prevalence has increased dramatically both in California and throughout the country over the past few decades. According to 2001 data, 11.9 percent of Californians, or 3.9 million people, have asthma (CHIS, 2003). Children have been especially affected; in California, asthma prevalence is greatest among 12 to 17 year olds.

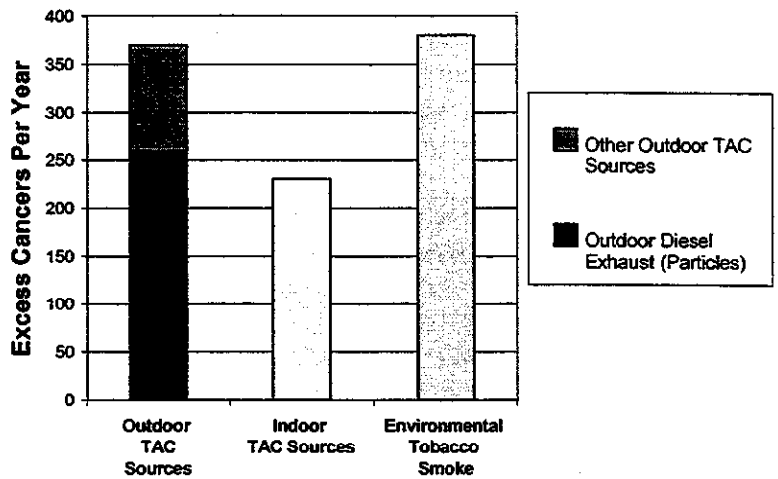
Indoor air pollutants exacerbate asthma symptoms, resulting in breathing difficulties. A recent Institute of Medicine (National Academy) report, *Clearing the Air: Asthma and Indoor Exposures* (IOM, 2000), identified new associations between indoor air pollutants and asthma, in addition to the traditional indoor asthma triggers such as cat and dog dander, house dust mites, and environmental tobacco smoke (ETS). The scientists found sufficient evidence of an association between exacerbation of asthma and exposure to high levels of nitrogen dioxide (NO_2) and other nitrogen species (NO_X), and mold. They found limited or suggestive evidence of an association of asthma exacerbation with exposure to formaldehyde and fragrances. A more recent review of indoor pollution studies further identified several links between asthma symptoms and specific volatile organic chemicals (VOCs), especially formaldehyde (Delfino, 2002). Studies of workplace asthma have further demonstrated an association between asthma symptoms and VOCs, primarily from cleaning products (Rosenman *et al.*, 2003). Several studies also have found an association of increased outdoor ozone levels with exacerbation of asthma, and one study recently linked ozone with the development of asthma in children who are active outdoors. Similar effects would be expected with exposure to ozone indoors.

Cancer

A substantial number of common indoor pollutants have been classified as carcinogens. Examples include formaldehyde, benzo(a)pyrene and other polycyclic aromatic hydrocarbons (PAHs), tobacco smoke, benzene, chlorinated solvents such as tetrachloroethylene, and radon gas. Several studies have measured indoor concentrations of carcinogenic chemicals in California homes. Results have shown that carcinogens, especially formaldehyde, are routinely found in most homes, often at higher concentrations than concurrent outdoor levels, due to the presence of indoor sources. These concentrations result in extended indoor exposures, which translate to a significant increase in cancer risk attributable to indoor pollutants, primarily those emitted from building materials and consumer products. As shown in Figure ES-2, ARB staff estimate that about 230 excess cancer cases may occur annually in California due to exposures from the limited number of indoor toxic air contaminants that can be quantified from residential and consumer sources. This estimate approaches the estimated cancer burden from outdoor diesel exhaust particles, which is responsible for much of the excess cancer burden associated with breathing ambient air in California. This indoor cancer estimate also equals about two-thirds of the total burden from excess cancer resulting from outdoor air pollutant emissions.

Exposure to environmental tobacco smoke (ETS) makes a significant contribution to the cancer burden from air pollution as well. OEHHA has recently estimated about 400 excess cancers from ETS for the year 2004, which translates to about 380 excess cases for the year 2000, shown in Figure ES-2 for comparability (fully discussed in Sections 2.1.2, 2.3.3, and 3.1.3). Those ETS risk levels are similar to the total outdoor burden. Despite workplace restrictions and other positive trends, the risk from ETS will remain significant, because some individuals, especially children in households with smokers, are still exposed to substantial levels of ETS.

Figure ES-2:
Estimated Potential Cancer Burden from Air Toxics in California by Source



Radon, a radioactive gas, enters indoor environments from uranium-containing soil and rock under and near buildings, and from some domestic water obtained from groundwater and wells, and decays into radioactive radionuclides often called radon daughters. Only an estimated 0.8 percent of California residences have annual radon levels above 4 picoCuries per liter (pCi/l), U.S. EPA's recommended mitigation level. However, due to its potency and synergism with tobacco smoke, a preliminary estimate extrapolated from national data shows that radon may contribute to 1500 excess lung cancer deaths per year in California. However, the risk from radon cannot be fully differentiated from that of exposure to tobacco smoke (NRC, 1999a), and reducing exposure to tobacco smoke is the most effective measure to reduce the risk of lung cancer (NRC, 1999a; Mendez *et al.*, 1998). Additionally, this may be an overestimate, because exposure to tobacco smoke is notably lower in California than in the rest of the nation, and recent measurements indicate somewhat lower radon levels in the Sierra Nevada foothills than previously measured, indicating that statewide levels may be lower as well. In light of these factors, the actual radon risk in California is uncertain. Because elevated radon is found in just a few areas in California, and can vary from building to building, radon mitigation is not recommended in existing buildings until adequate testing has been conducted in each building, and preventive measures are recommended in new buildings only in areas where radon soil levels are elevated.

Irritant Effects

Many indoor pollutants cause eye, nose, throat, and respiratory tract irritation. Aldehydes, as well as some other VOCs and oxidants, are known mucous membrane irritants. Formaldehyde is the most commonly identified irritant. Acute effects of irritant chemicals can include respiratory and eye irritation, headache, difficulty breathing, and nausea. Some of these effects, particularly respiratory symptoms and eye, nose, and throat irritation can also be experienced with chronic exposure. Terpenes, such as pinene and limonene, frequently used in cleaning products for their favorable odor characteristics and solvent properties, react with indoor oxidants to produce formaldehyde and ultrafine PM. Further research is needed to understand

the extent and duration of exposure to terpene reaction products, and the potential health effects of those exposures.

Irritant chemicals and other factors are suspected of causing or contributing to episodes of Sick Building Syndrome (SBS), in which a large number of building occupants experience irritant and neurological effects while they are in a building. The specific causes of SBS have not yet been firmly identified; however, SBS episodes can affect a high number of workers, have been well documented, and have resulted in high costs to some businesses due to reduced productivity and, in some cases, legal settlements. The most common symptoms include eye irritation, congested nose, headache, fatigue, difficulty concentrating, and skin rash.

Premature Death and Increased Disease

Several pollutants for which ambient air quality standards have been established occur at elevated levels indoors due to emissions from indoor sources. In other cases, indoor sources increase the high levels of exposure that occur when high levels of polluted ambient air enters the indoor space. Ambient particulate matter (PM) has been associated with premature death and serious respiratory and cardiovascular effects in numerous studies. Carbon monoxide (CO) can cause death with high exposures of relatively short duration, and lower levels can cause flu-like symptoms and other health effects. Nitrogen dioxide (NO_2) can harm the lungs and other mucous membranes, cause respiratory disease, and exacerbate asthma. Ozone can have similar effects at elevated levels; however, indoor levels are typically lower than outdoor levels. Indoor sources of these pollutants sometimes cause indoor concentrations that exceed health-based ambient air quality standards established for outdoor air.

Particulate matter

Particulate matter (PM) is a complex mixture of very small particles and other non-gaseous materials suspended in the air. Indoor particle sources include combustion devices such as woodstoves and fireplaces, and activities such as smoking, cooking, candle burning, and vacuuming, all of which can produce PM with harmful components similar to those found in outdoor air. Indoor particles also include fibrous materials, pollen, mold spores and fragments, and tracked-in soil particles. Pollens and mold can trigger allergies and asthma. Tracked-in particles and some particles from combustion sources become trapped in carpets and have been shown to include a mix of toxic components such as polycyclic aromatic hydrocarbons (PAHs) and lead.

A large number of major epidemiologic studies have consistently shown a strong association between outdoor (ambient) PM concentrations and increased mortality from cardiovascular and respiratory disease. They also have shown increased morbidity effects with increased PM levels, including increased hospitalizations and emergency room visits due to respiratory problems such as asthma, chronic obstructive pulmonary disease (COPD), chronic bronchitis, and pneumonia; increased respiratory symptoms such as cough and wheeze; decreased lung function and reduced lung function growth in children; and increased cardiovascular disease such as congestive heart failure, stroke, and ischemic heart disease.

The studies documenting these effects measured outdoor particles, which are composed of a mix of particles from combustion sources, soil, and particles formed through chemical reactions in the atmosphere. Because a substantial portion of PM from indoor sources is similar to outdoor PM components, indoor PM emissions are highly likely to be significant contributors to

the adverse impacts seen in the epidemiology studies, and they may also contribute to those effects beyond the levels quantified in the epidemiology studies.

Reducing outdoor PM concentrations to the level of the current California ambient air quality standard for PM would result in significant reductions in adverse health effects, including approximately 6,500 deaths and 17,000 serious, non-fatal illnesses each year in California (ARB/OEHHA, 2002). Although current studies have not directly addressed the potential impacts of indoor PM on health, if consistent with outdoor PM, the impacts of PM of indoor origin are likely to have very large impacts on public health, potentially resulting in thousands of additional cases of serious illness and disease each year.

Carbon monoxide

Carbon monoxide (CO) is a colorless, odorless gas that causes flu-like symptoms (headache, nausea, lethargy) and inability to concentrate, at lower exposure levels over periods of time. At very high levels, CO can cause unconsciousness and even death. CO is a product of incomplete combustion, emitted from sources such as vehicle exhaust, gas and propane stoves and furnaces, woodstoves, kerosene heaters, and cigarettes.

Very high levels of CO occur relatively infrequently indoors. However, a California study of death certificates showed that about 30 – 40 deaths occurred in California each year, on average, due to unintentional CO poisoning (Girman *et al.*, 1998; Liu *et al.*, 1993a, 2000). About two-thirds of those deaths were attributable to indoor sources. The indoor sources most implicated in past CO poisonings were combustion appliances, such as malfunctioning or poorly tuned gas or propane furnaces and stoves, and the improper use of charcoal grills and hibachis indoors (contrary to warnings). Motor vehicles, such as those unwisely left running in a garage, also have taken a substantial toll. The relevant literature also indicates that other CO health effects occur: hundreds of emergency room visits and thousands of misdiagnosed flu-like illnesses due to non-fatal CO poisoning are estimated to occur each year.

Nitrogen dioxide and associated acids

Nitrogen dioxide (NO_2) is a red to dark brown gas with a pungent acrid odor. Adverse health effects attributable to NO_2 include exacerbation of asthma (especially in children), respiratory symptoms and infection, lung damage, and lung disease after long periods of exposure. Indoor sources of NO_2 include gas and propane appliances, wood burning stoves and fireplaces, kerosene heaters, charcoal grills, and motor vehicles. Indoor levels can be especially elevated from the use of older wall furnaces, when their exhaust is not vented to the outdoors, and from gas stoves, because people often do not use the exhaust hoods above them, or the exhaust is not vented to the outdoors. Several nitrogen compounds related to NO_2 also are found in indoor environments, including nitrous acid (HONO) and nitric oxide (NO). Nitrogen dioxide is the only nitrogen oxide regulated as a pollutant in outdoor air.

Ozone

Ozone is a serious respiratory irritant and a main component of smog. Outdoor ozone is the primary source of indoor concentrations of ozone in most indoor environments, but ozone also is directly emitted indoors from some types of "air cleaners", and from poorly maintained laser printers and other types of office equipment. Indoor levels typically range from 20 to 80 percent of outdoor levels, but have been shown to reach levels that exceed a Stage 1 smog alert level when some types of ozone-generating "air cleaners" are used. Breathing elevated

concentrations of ozone can be harmful to health, especially for active people, including children. Whether inhaled indoors or outdoors, ozone can irritate the respiratory tract, which can cause coughing, wheezing, and pain on deep breathing. Ozone also can exacerbate asthma, particularly those with concurrent allergen exposure. Ozone masks the odor of other indoor pollutants by deadening the sense of smell. It also reacts with certain indoor pollutants to produce toxic by-products, including formaldehyde and ultrafine particles.

Toxic Air Contaminants and Other Indoor Air Pollutants

Other pollutants can occur at elevated levels indoors due to emissions from indoor sources. Some have been identified by the ARB as toxic air contaminants (TACs), air pollutants other than traditional (criteria) pollutants that can contribute to an increase in death or serious illness.

- **Volatile organic compounds (VOCs)** – As mentioned above, VOCs such as formaldehyde and chlorinated solvents are common in indoor air, and can exacerbate asthma and cause cancer and irritant effects. Some of these chemicals also have reproductive, developmental, and neurological effects at very high levels encountered infrequently in non-industrial workplaces. Indoor levels of formaldehyde, a pungent smelling gas, nearly always exceed chronic health-based guideline levels and acceptable cancer risk levels. Formaldehyde is emitted from numerous indoor sources including building materials (especially pressed wood products), composite wood furnishings, personal care products, cosmetics, permanent pressed clothing, combustion sources, and some new carpet pads and adhesives.

- **Environmental tobacco smoke (ETS)** – ETS causes cancer, heart disease, asthma episodes, middle ear infections in children, sudden infant death syndrome (SIDS) and other adverse effects. ARB and OEHHA (2004/2005) currently estimate that exposure to ETS results in about 400 excess lung cancer deaths, 1700-5500 heart disease deaths, and more than 31,000 asthma episodes per year in children (these estimates are currently under scientific peer review). These estimates would be expected to decrease over time, due to the substantial reductions in smoking and ETS exposure in California. Nonetheless, despite decreases in the percent of smokers in the population and the statewide prohibition of smoking in workplaces, some individuals, especially children, are still exposed to elevated levels of ETS in the homes and vehicles of smokers.

- **Polycyclic aromatic hydrocarbons (PAHs)** – PAHs, emitted from combustion sources such as cigarettes, woodstoves and fireplaces, include a number of known or suspected carcinogens. They have been found to adsorb onto particles in the air and deposit onto carpets, from which they can be resuspended during vacuuming or other activity.

- **Radon daughters and asbestos** are other known lung carcinogens found indoors in some California environments. Both radon and asbestos are naturally-occurring, each emanating from specific types of soils. Radon levels in California are typically lower than mitigation guideline levels. Nonetheless, as discussed above, a preliminary estimate indicates that radon may contribute to 1500 excess lung cancer deaths in California per year. However the risk from radon cannot be fully separated from that of tobacco smoke, and reduction of exposure to tobacco smoke remains the primary mitigation approach. Indoor asbestos is elevated only infrequently, typically during remodeling of older buildings. Naturally-occurring asbestos has been measured recently in a few areas of California, and has resulted in some mitigation actions.

- **Pesticides and metals** – Dust from surfaces and carpets in homes and schools have been shown to contain numerous residues of pesticides, lead, mercury and other long-lasting contaminants that have originated from outdoor activities, cigarettes, fireplaces, and other sources. This is of special concern for very young children, who spend time on the floor, and put their hands in their mouths, because ingestion is often the primary route of exposure. Pesticides are widely used, and some can cause adverse developmental and neurological effects at elevated exposure levels. Many pesticides registered for use today are short lived, yet some are persistent in the environment, lasting 20 or 30 years or more. Recent studies indicate that some pesticides may be more persistent in indoor environments, because they do not experience the effects of weather and sunlight.

Biological Contaminants

Biological contaminants include substances of plant, animal, or microbial origin, such as bacteria, viruses, mold, pollen, house dust mites, animal dander; biological toxins such as endotoxins and mycotoxins, and microbial volatile organic chemicals. These biological agents are abundant in both indoor and outdoor environments, but are considered contaminants when found in undesired locations or at elevated concentrations. Excessive exposure to these contaminants can be associated with mucous membrane irritation (which may cause symptoms such as itchy eyes, runny nose or sore throat) or hypersensitivity reactions such as asthma attacks or allergy symptoms in sensitive individuals. Some individuals in persistently damp buildings report a variety of symptoms such as headache, memory difficulties, vomiting, and diarrhea; some researchers postulate that exposure to biological toxins may induce such symptoms. In a recent Institute of Medicine report (IOM, 2004), scientists found a number of symptoms and illnesses associated with dampness in buildings and with indoor mold, although the scientific evidence was not yet considered sufficient to confirm a causal relationship.

Many communicable diseases are primarily transmitted from person to person in indoor air. Common viral infections such as influenza, measles and chicken pox, as well as emerging diseases such as Severe Acute Respiratory Syndrome (SARS), are spread through inhalation of virus-contaminated droplets produced when an infected person coughs or sneezes, or from touching a surface or object contaminated with infectious droplets and then touching one's mouth, nose, or eyes (CDC, 2004). Tuberculosis is a notorious infectious disease that is transmitted in closely occupied spaces. Building-related illness (BRI) refers to an illness for which the specific cause can be identified within the building, such as bacteria in ventilation systems causing Legionnaires' disease, or humidifier fever. The usual causes of BRI include viruses, bacteria, and fungi. BRI impacts can be substantial, and are of increasing interest as the role of buildings in promoting diseases of biological contaminants becomes better understood.

III. INDOOR CONCENTRATIONS AND PERSONAL EXPOSURES

Indoor concentrations of many pollutants sometimes exceed health-based guideline levels or standards. Some pollutants, like formaldehyde, nearly always exceed recommended levels. Studies conducted by the ARB, the U.S. EPA, and others also have shown that indoor levels of VOCs and some other pollutants are often higher than outdoor levels.

More importantly, people's "personal exposures" to pollutants, especially to VOCs, are often greater than both indoor and outdoor pollutant levels. Personal exposures to some pollutants are elevated because people spend time very near sources of pollutants, such as when using a gas stove, cleaning solutions, or personal care products. During such activities, the product

emissions are most concentrated very near the person. Pollutants become more diluted in the air as distance from the source increases. Consequently, for VOCs and many other pollutants, personal exposure levels are most closely correlated with indoor concentrations.

Indoor – Outdoor Relationships

There is continuous air exchange between indoor environments and the outdoors. Outdoor emissions readily infiltrate into indoor environments, and indoor emissions seep outdoors and can contribute to outdoor air pollution. For example, ozone formed outdoors and fine particles and other emissions from nearby motor vehicles typically penetrate indoor environments to varying degrees, depending on the rate of air exchange, degree of filtration, and other factors. For residential buildings, the main entry routes of outdoor air are open windows and doors, gaps in the building shell, and devices such as swamp coolers that move outdoor air indoors. For large public and commercial buildings, the main entry route is through the mechanical heating, ventilating, and air conditioning (HVAC) systems, which actively move outdoor air indoors and typically filter some of the particles from the air. Any pollutants in the air just outside the building may thus be brought into the indoor space. Indoor pollutant levels can be much higher than those outdoors when indoor sources are present and the air exchange rate is low.

Similarly, indoor pollutants can flow through windows and penetrate small gaps in the building shell to contribute to the local outdoor burden of pollution. Emissions from certain sources used indoors, such as paints, consumer products, and gas and woodburning appliances contribute to local outdoor pollution levels, either through direct emissions or, in the case of reactive volatile organic chemicals, through chemical reactions.

Environmental Justice Considerations

ARB adopted an environmental justice policy in 2001. This policy requires the fair treatment of all people regardless of gender, ethnicity, and socioeconomic status. The limited research available indicates that some segments of the population may be disproportionately exposed to indoor pollutants. In California, African Americans, American Indians, and Alaska natives experience a higher prevalence of lifetime asthma (Meng *et al.*, 2003). However, in general, the prevalence of asthma appears to be more strongly correlated with lower socioeconomic status than with race and ethnicity (IOM, 1993). Dust mites, cockroaches, and mold are important asthma triggers that are more likely to be present in locations where lower income individuals most often live. Additionally, research indicates that blood lead levels are higher for poor and minority children in central cities. Formaldehyde levels have been highest in mobile homes, which are more often occupied by lower income families.

The ARB has taken steps to address some of these issues. Special air monitoring studies have been conducted at schools in some communities, and a large asthma study is underway. Fact sheets for public outreach have been published in English and Spanish. Efforts are underway to limit formaldehyde emissions from composite wood products through an Air Toxics Control Measure. Pursuit of indoor mitigation measures can further help reduce any disparities in exposure and health impact that may exist among different groups of the population.

Non-industrial Workplaces

Non-industrial workplaces often provide unique situations for exposure to indoor air pollutants. Products and activities associated with non-industrial workplaces such as beauty salons, hospitals, dry cleaners, medical laboratories, jails, photocopy centers, aircraft cabins, indoor

sports arenas, retail shops, copy shops, and other workplaces can lead to elevated levels of air pollutants and indoor environmental concerns. Despite Cal/OSHA's regulations for pollutant levels and ventilation requirements, some workers in these environments experience adverse health effects related to indoor environmental quality. Additionally, many of these locations are regularly visited by members of the public.

Cal/OSHA has various regulations to address ventilation and personal exposures, but most indoor air quality complaints arise in non-industrial workplaces, where preventive controls are not normally utilized. A review of Cal/OSHA inspection data indicates ventilation requirements are often not met. From 1995 through 2001, citations for ventilation violations were issued during 519 on-site inspections. Of these citations, the majority (260) were in services, with 114 in schools and 33 in health care. A substantial number of citations were issued for other indoor air quality violations as well, but there are inconsistencies in recordkeeping, making it difficult to analyze for indoor air qualiy concerns. However, from 1997 through 1999, 849 workplace inspections were coded as indoor air investigations, and Cal/OSHA has indicated that the actual number of indoor air quality related inspections may be more than twice that number.

IV. COSTS OF INDOOR POLLUTION

Indoor air pollution takes a significant toll on Californians' lives and has significant economic costs. Exposure to indoor pollutants results in premature death and increased disease, increased expenditures for health care, decreased worker productivity, and decreased learning by school children. Table ES-2 shows estimates of the costs of indoor air pollution in California that are currently quantifiable. It includes the valuation of health (cost of premature death), an estimate of the increased expenditures for health care, and an estimate of some of the costs associated with reduced worker productivity. Because of the limited amount of information available for accurately estimating indoor pollution costs and the broad range of effects and resultant costs, there is considerable uncertainty in the cost estimates shown. Most importantly, the costs of many known or suspected indoor pollution impacts cannot currently be quantified due to lack of cost data and/or sufficiently quantified population exposure data. For example, the costs for the impacts of biological contaminants and indoor PM-related illness and disease are likely to be very high, potentially in the billions of dollars, but are not yet quantifiable. Additionally, while ETS has been well-studied and its impacts and costs can be reasonably quantified, the impacts and costs for some other toxic indoor pollutants have been less studied and cannot be quantified at this time.

The combined cost of both fatal and non-fatal impacts due to indoor air pollution in California homes, schools, and non-industrial workplaces is substantial: it is estimated to be **$45 billion per year.** As shown in Table ES-2 and discussed in Section 3 of this report, the annual valuation of premature deaths attributable to indoor air pollution is estimated to total about $36 billion, with most stemming from ETS and radon. OEHHA's most recent ETS risk estimates are currently under review by the Scientific Review Panel, and may change somewhat before this report becomes final. The actual total valuation of premature deaths is likely to be higher than the $36 billion presented here because these estimates do not include the impacts of other pollutants that may increase the risk of premature death, such as other carcinogens emitted from materials and products, and PM from cooking, wood smoke, and other indoor combustion sources.

Table ES-2. Summary of Estimated Costs of Some Indoor Air Pollution in California

Health End Point	Health Valuation: Premature Death[1-3] ($ Billions/yr)	Medical Cost[2-4] ($ Billions/yr)	Lost Productivity Cost[2,3] ($ Billions/yr)	Total Cost ($ Billions/yr)
CO: poisoning	0.15	<0.001	NA	0.15
VOCs: cancer	0.73	0.011	NA	0.74
ETS: lung cancer	2.4	0.025	NA	2.4
ETS: heart disease	23	0.055	NA	23
ETS: asthma episodes	NA	0.020[5]	NA	0.020
ETS: low birth weight	NA	0.19	NA	0.19
ETS: otitis media	NA	0.019[5]	NA	0.019
Radon: lung cancer	9.5	0.097	NA	9.6
Mold and moisture: asthma and allergies	0.031	0.19[5]	NA	0.22
Sick building syndrome	NA	NA	8.5	8.5
TOTAL[6]	**36**	**0.6**	**8.5**	**45**

1. From Table 3.2.
2. Estimates are based on average or mid-point of incidence rates of death and disease from previous tables, and estimates of productivity discussed in the text. Values are rounded to two significant figures.
3. Original data were adjusted to year 2000 dollars and year 2000 population, except where noted otherwise in previous tables.
4. From Table 3.3.
5. Includes indirect costs such as lost work days, lost school days, and travel expenses.
6. Totals are rounded to two significant figures. These totals are likely low because conservative cost estimates were used, and quantitative information is not readily available for many known impacts of indoor air pollution, such as for indoor PM and many indirect costs of health effects.

The quantifiable medical costs (direct and some indirect) due to indoor air pollution total more than $0.6 billion per year, with a large portion of the costs attributable to mold and other moisture-related allergens. These cost estimates for morbidity do not include the potential losses due to other impacts such as those from other indoor allergens, the long-term effects of CO poisoning, reduced student performance, lost earnings opportunity, unpaid caregivers, and human suffering. Finally, the cost of reduced worker productivity due to indoor air pollution

(primarily sick building syndrome) that could be prevented is estimated to be about $8.5 billion per year.

V. EXISTING REGULATIONS, GUIDELINES AND PRACTICES

Despite the significant health effects and potential economic impacts caused by indoor sources of pollution, there are few government standards restricting emissions from common sources of indoor pollutants, and there is no comprehensive program to protect air quality within residences, schools, or public and private buildings. A variety of agencies and organizations have established standards and guidelines that can be applied to limited aspects of indoor environments to assist in the assessment and control of health hazards from air pollutants. Foremost among these are workplace standards; however, those standards are designed for 8-hour exposures of healthy adults, are not as protective as standards set for ambient air, and are not designed to be protective of the more sensitive subgroups of the population, such as children. Other standards are applicable to indoor air quality, but only in a limited way. For example, the ambient air quality standards and emission control regulations indirectly improve IAQ by improving ambient air quality, and Assembly Bill 13 (1995) prohibits cigarette smoking, in workplaces. Although many of these programs have resulted directly or indirectly in improvements in indoor air quality, they do not ensure healthful indoor air quality.

- **Workplace Standards.** The California Occupational Safety and Health Program (Cal/OSHA) in the Department of Industrial Relations (DIR) has authority to develop, promulgate, and enforce air pollutant exposure limits, ventilation regulations, and other standards for the workplace that directly impact indoor air quality. The California Occupational Safety and Health Standards Board is the unit within the Cal/OSHA program with authority to adopt standards and regulations to protect workers. Some of the Cal/OSHA standards and regulations that impact indoor air quality are the following:

  - ✓ **Permissible Exposure Limits**. The Standards Board sets permissible exposure limits (PELs) and other limits for airborne contaminants. The PELs are 8-hour exposure limits generally protective of the health of most workers. However, they are not designed to protect vulnerable members of the population such as infants, the elderly, or individuals with pre-existing heart or respiratory disease. Additionally, they are not intended to be protective for exposures greater than eight hours per day, five days a week, and PELs are not available for all indoor air contaminants.

  - ✓ **Ventilation**. Cal/OSHA requires employers to maintain and operate mechanical ventilation systems to provide at least the quantity of outdoor air required by the State Building Code at the time the building permit was issued.

  - ✓ **Mold, moisture**. Cal/OSHA requires that workplaces be maintained in a sanitary condition, and that employers correct all types of water intrusion or leakage, to reduce the potential for mold growth.

- **Ventilation design requirements.** Minimum ventilation levels for the design quantity of outdoor air in new non-residential buildings, such as offices and public buildings, have been established by the California Energy Commission for different types of buildings and different types of rooms (e.g., conference rooms vs. offices). The Commission also sets energy efficiency standards for residences, which has resulted in reduced infiltration of outdoor air, or "tightening" of new homes compared to older homes. This has implications for

indoor air quality, and the Commission is funding research to assess the need for revisions to the standard to assure healthful IAQ in homes.

- **Anti-smoking law**. Cigarette smoking, a major source of indoor pollution, is prohibited in nearly all public buildings in California. A statewide, smoke-free workplace law passed in 1995 (AB 13) eliminated smoking in nearly all California indoor workplaces–including restaurants, bars and gaming clubs–and spurred a reduction in smoking by the California population. The ban has been very successful in reducing worker exposure to cigarette smoke. In 1999, 93 percent of California's indoor workers reported working in a smoke-free environment, compared to only 45 percent in 1990 (Gilpin *et al.*, 2001). The prohibition of workplace smoking, along with the Department of Health Services Tobacco Control Program, have both had far reaching benefits. In 1994, 63 percent of Californians with children did not allow smoking in the house; by 2001, 78 percent did not allow it (Gilpin *et al.*, 2001). Additionally, smoking rates among California adults declined from 26 percent to 17 percent between 1984 and 2001 (BRFSS, 2001).

- **State and national ambient air quality standards** (AAQS) and control programs, established by the ARB and U.S. EPA, respectively, are developed to protect the general public from the harmful effects of "traditional pollutants" in outdoor air, for specified averaging times (exposure times). California's AAQS are often more protective than the national AAQS. Currently, the State AAQS are under review to ensure that they are protective of sensitive populations, especially infants and children (ARB/OEHHA, 2000). In the absence of indoor air quality standards or guidelines, the AAQS serve as useful guideline levels for those pollutants indoors, because they are based on specified averaging times and incorporate a margin of safety. Both the California and federal AAQS are listed at http://www.arb.ca.gov/research/aaqs/aaqs.htm.

- **Consumer product standards**. The federal Consumer Product Safety Commission (CPSC) has jurisdiction over consumer products, except for pesticides, cosmetics, tobacco and cigarettes, food, drugs, automobiles, and a few others. CPSC has authority to ban a product, establish mandatory safety standards, recall products for repair or replacement, require warning labels, or develop voluntary standards in conjunction with manufacturers. However, CPSC is primarily focused on addressing safety issues, and most often uses voluntary processes and labeling requirements.

  The ARB also regulates consumer products, for the purpose of reducing smog in California. An additional benefit is a reduction in the amount of certain types of VOCs that are released in homes and institutions. In addition to reducing the reactive VOC content of products, ARB has prohibited the use of several toxic air contaminants in 13 categories of products, resulting in reduced indoor emissions of those substances. Reducing reactive VOC emissions and toxic pollutants from cleaning compounds, polishes, floor finishes, cosmetics, personal care products, disinfectants, aerosol paints, and automotive specialty products has likely reduced personal exposures to those VOCs as well.

- **Local woodburning ordinances.** Several communities in California have recently implemented woodburning ordinances or policies to reduce smoke emissions in their communities. For example, in the San Francisco Bay area, 24 cities have ordinances that prohibit conventional fireplaces in new construction. The mountain town of Truckee has a more aggressive policy that states that existing unapproved wood burning appliances must be removed from all properties by July 15, 2006. The San Joaquin Valley Air Pollution Control District issues daily advisories on restrictions for residential wood burning.

- **Guidelines and Public Education.**

  - ✓ OEHHA has developed **acute and chronic reference exposure levels (RELs)** as guidelines to prevent harm from toxic air pollution, under the Air Toxics "Hot Spots" Information and Assessment Act of 1987 (HSC Section 44300 *et seq)*. Although established to identify healthful limits for outdoor air near industrial sources, RELs have been used for indoor pollutants as indicators of potential adverse health effects other than cancer. OEHHA has established chronic RELs for 79 air pollutants to define healthful levels for exposures that can last 12 years or more (OEHHA, 2003a), and acute RELs for 51 chemicals to define healthful levels for exposures of one hour (OEHHA, 2000a). OEHHA also has developed an 8-hour, interim indoor REL (IREL) for formaldehyde of 27 ppb, specifically for indoor application. This IREL identifies the level below which effects such as eye, nose, and throat irritation would not be expected to occur during typical daytime (8-hour) occupancy of buildings.

  - ✓ **ARB's Indoor Air Quality Guidelines** have been developed to advise the public regarding the health effects and indoor sources of key indoor pollutants, and what the public can do to reduce their exposures. Some AAQS are used as recommended maximum exposure levels in ARB's *Combustion Pollutants Guideline*. ARB's guidelines for formaldehyde and chlorinated solvents recommend achieving as low a level of those pollutants as possible indoors, because they are carcinogenic, and there are no known levels that are absolutely safe.

  - ✓ **DHS and other agencies** have developed various guidelines that can be applied to improve indoor environments. DHS published guidelines for reducing VOCs in new office buildings in 1996, played a key role in the development of Section 01350 emissions limits for materials used in state buildings and schools, and has been directed to develop guidelines to prevent and remedy mold problems in buildings. The California Energy Commission spearheaded the formation of the Collaborative for High Performance Schools (CHPS), which has developed *Best Practices Manual* that include guidance for selecting building materials with reduced indoor pollutant emissions. The U.S. EPA has developed its *IAQ Tools for Schools Program* to provide guidance for assuring healthful indoor air quality in schools. All of these and ARB's indoor air quality guidelines are available at no charge on the Internet.

  - ✓ **Industry and professional groups** have developed numerous guidelines for improving indoor air quality. Examples include the building ventilation requirements of the American Society of Heating, Refrigerating, and Air-conditioning Engineers' (ASHRAE), the product emissions criteria of the Carpet and Rug Institute's (CRI) Green Label and Green Label Plus Programs, and voluntary formaldehyde emission limits of the Composite Panel Association and the Hardwood Plywood and Veneer Association. The industry and professional guidelines vary in their degree of IAQ protection, but are widely used and generally have helped reduce some indoor pollutants over the years.

VI. METHODS TO PREVENT AND REDUCE INDOOR AIR POLLUTION

There are a number of methods that can be used to prevent or reduce indoor air pollution. The most effective approach is to remove or reduce indoor emissions by using building materials, consumer products, and appliances that emit little or no air pollution. Ventilation (including proper exhaust ducting) and public education also are important components of a strong indoor air quality improvement program. Air cleaning devices (air filters and air cleaners) can be helpful

in certain situations; however, their effectiveness is often limited, and some air cleaners actually release ozone into the indoor environment, adding to the indoor pollutant burden.

> **Minimizing indoor emissions is generally more effective than removing them after emission has occurred.**
>
> *T.J. Kelly, Battelle, Indoor Air Quality Symposium: Risk Reduction in the 21st Century, Sacramento, May, 2000*

- **Source control** is the most reliable approach to prevent indoor pollution because it does not depend on building maintenance or other human actions to assure healthful indoor air (NRC, 1981). Source control includes source substitution, source removal, and source modification. Reduction at the source is most effectively achieved through use of low- or zero-emitting appliances, products or materials, or reformulation of chemical products. For example, indoor formaldehyde levels can be greatly reduced by using building materials that emit little or no formaldehyde, instead of materials made with urea-formaldehyde resins. Low emission product designs or reformulations can usually be accomplished by the manufacturer, with minimal impact on the consumer, often with only minor increased costs.

- **Ventilation** is a standard engineering approach to assuring good indoor air quality and comfort. Ventilation removes and dilutes indoor contaminants, removes moisture from the air which helps to prevent mold growth, and removes body effluents such as carbon dioxide that lead to a stuffy environment. Natural ventilation, through open windows and doors, is the primary ventilation route for residences, while mechanical ventilation, using HVAC systems, is most common in commercial buildings. Adequate and effective ventilation, and ducting of exhaust from combustion appliances, are necessary for acceptable indoor air quality, even when known air contaminants are minimized. However, ventilation is not a complete solution to indoor pollution: ventilation consumes energy, and some pollutants, such as formaldehyde emitted from building materials, require years to off-gas and are not completely removed by ventilation.

- **Proper operation and maintenance of buildings** is critical to achieving and maintaining healthful air quality in buildings. Ventilation systems should be maintained as intended and filters replaced routinely to prevent soiling and the growth of mold and bacteria in the ventilation system and in the occupied space. Roof leaks that are not repaired promptly can lead to moisture intrusion and mold growth. Regular cleaning of indoor spaces with proper cleaning methods can reduce biological contaminants, such as those associated with insects and pollen, as well as persistent chemicals. Inattention to proper operation and maintenance will not only lead to poor indoor air quality, but can also prove more costly in the long term due to increased costs to remedy the larger problems that result.

- **Public education** is a key step for reducing Californians' exposures to many indoor air pollutants. People's choices and activities have a major impact on their exposures to air pollution. The use of various consumer products, and activities such as cigarette smoking and cooking can result in significant indoor releases of pollutants. However, public education alone is not a complete solution. Some groups of the population cannot respond appropriately to take needed action. For example, children cannot read or understand all written information that is provided; elderly people living in group settings cannot control the products used in their facility; and low-income families may not be able to afford safer alternatives.

- **Air cleaning devices** can also help improve indoor air quality; however, their effectiveness is often limited. Air cleaning devices include both central air filters and portable air cleaning appliances. Air filters are a normal component of mechanical HVAC systems in public and commercial buildings. High efficiency particulate arrestor (HEPA) filters, though not commonly used in commercial buildings, are most effective at removing particles from outdoor air as it is brought indoors. Air cleaning appliances are usually portable units used indoors to remove particles from the indoor air, although a few remove gases, and some remove both particles and gases. Mechanical air cleaners typically draw air through a filter while electronic air cleaners remove pollutants with the use of an electric charge. Electrostatic precipitators (ESPs) and ionizers are the two major types of electronic air cleaners on the market.

  The proper air cleaner may help control airborne particles in some situations; however, the limited scientific evidence available has not documented any health benefits from air cleaners. Additionally, ESPs and ionizers can produce ozone as a by-product; thus proper use and maintenance are critical to prevent harmful levels from developing when using these devices.

  Air cleaners that intentionally generate ozone should not be used indoors (ARB, 2005; DHS, 1998; ALA, 1997). Independent studies by the U.S. EPA, the Consumers Union, and others have shown that ozone-generating air cleaners do not effectively destroy microbes, remove odor sources, or reduce indoor pollutants enough to provide any health benefits. These devices can emit very high amounts of ozone – several times the state ambient air quality standard – but they are currently unregulated.

Ozone-generating Air Cleaners

"These machines are insidious. Marketed as a strong defense against indoor air pollution, they emit ozone, the same chemical that the ARB and USEPA (U.S. Environmental Protection Agency) have been trying to eliminate from our air for decades."

Barbara Riordan, interim ARB Chairperson. California Air Resources Board, Press Release 05-02, Sacramento, January 2005.

VII. PRIORITIZATION OF INDOOR SOURCES BASED ON EXPOSURE AND ADVERSE IMPACTS

Reduction of public exposure to the many indoor air pollutants is most effectively achieved by reducing pollution at the source. Tables ES-3.1 and ES-3.2 suggest a prioritization scheme for implementation of mitigation measures, by source categories. The source categories have been ranked into two groups–high and medium priority–and are listed alphabetically within each ranking group. The primary factors considered in prioritizing the source categories include the extent of the population's exposure to the sources and their emissions, the relative reduction in health impacts that could be achieved with further action beyond any already undertaken, ease of mitigation, and trends in emissions from and use of source categories. A quantitative prioritization was not undertaken because such an effort is well beyond the scope of this report. Such an effort would be an appropriate step prior to taking action under a comprehensive program to address indoor sources. Additionally, the preliminary indoor air pollution cost estimates provided in this report were considered, but were not weighted heavily in the prioritization because they primarily reflect the availability of cost information and the length of time a given pollutant, such as ETS and radon, has been studied, not necessarily the actual extent of exposure and risk in California.

Table ES-3.1. High Priority Source Categories for Mitigation [1]

SOURCES OF POLLUTANTS[2] (listed alphabetically)	EXAMPLES OF POLLUTANTS[3] EMITTED	POTENTIAL APPROACH TO MITIGATION[4]	DIRECT STATE AUTHORITY TO TAKE IAQ MITIGATION ACTIONS
Air Cleaners (ozone-generating)	Ozone	Emission limitations	No
Biological Contaminants (mold, pollen, bacteria, viruses, house dust mites, cockroaches)	Particles, allergens, asthma triggers, toxins	Requirements for habitable spaces; require certification of mold assessors and mitigators	Limited
Building Materials & Furnishings (particle board, plywood, paneling, flooring, caulk, adhesives, new carpet assembly, furniture)	Formaldehyde, acetaldehyde, benzene derivatives, acrylates, naphthalene, phenol, some other VOCs	Emission limitations, product use restrictions, market incentives	Limited (some indirect)
Combustion Appliances (gas & propane stoves, ovens, furnaces, heaters; woodstoves and fireplaces)	Carbon monoxide, nitrogen oxides, particles, soot, polycyclic aromatic hydrocarbons	Emission limitations, active exhaust ventilation, safety devices, product use restrictions, product re-design, improved venting	No
Environmental Tobacco Smoke (cigarettes, cigars)	Particles, polycyclic aromatic hydrocarbons, benzene, carbon monoxide, some other VOCs	Focused parent education; reduce smoking in homes and vehicles	Yes, workplaces No, private homes and vehicles
Radon (soil, rock, ground water, building materials containing radium)	Radionuclides, radon gas	Screening measurements, building codes	Limited

1. Individual sources may be higher or lower than the source category ranking.
2. All of the examples of pollutant sources may not emit all of the pollutants listed in the corresponding box in column two.
3. Air pollutants may be identified as Toxic Air Contaminants (TACs) by the California Air Resources Board, and/or identified as Proposition 65 chemicals; or, criteria (traditional) air pollutants.
4. Public education, economic incentives, and non-regulatory approaches should also be used where appropriate. The actual approach taken would be determined after extensive discussions with the relevant industries, in consideration of costs, feasibility, and effectiveness.

Tables ES-3.1 and ES-3.2 also suggest potential approaches for mitigating the pollutants and sources listed. Emission reductions should be accomplished at the manufacturing, distribution, or construction stage. Alternatives or mitigation options are currently available for most of the sources listed. Emission limitations achieved at the manufacturing stage, such as reducing toxic contaminants in building materials, would be effectively invisible to the consumer and assure exposure reduction. For example, low-emitting carpets, no-formaldehyde furniture, and non-toxic cleaning products are currently sold in the marketplace. Alternative products or formulations must be recommended with care, however: substitutes should not result in increased emissions of, or exposures to, other toxic pollutants.

Finally, Tables ES-3.1 and ES-3.2 include a column indicating whether direct authority exists at the state level to take the mitigation actions listed in column three. For most source categories, there is no state agency with clear, direct authority to take the mitigation actions indicated. In some categories, one or more agencies has limited authority to address a small portion of the sources included. For example, if needed, Cal/OSHA could impose product use restrictions or require other actions to reduce worker exposure to institutional cleaning product emissions. However, neither Cal/OSHA nor any other state agency has direct authority to restrict pollutant emissions from cleaning products for the purpose of reducing indoor air concentrations and exposures. In other cases of limited authority, the benefits to indoor air are incidental results from actions taken under the agency's primary authority, or mitigation actions required to avoid negative impacts from regulations.

The specific rationale for the ranking of each category is briefly discussed below. Note that some individual sources within the group may have a higher or lower priority. A more detailed assessment would be needed to prioritize specific products within these larger categories.

High-Ranked Source Categories

- **Air cleaning devices or "air purifiers" that generate** ozone can produce unhealthy levels of ozone in indoor environments. Some devices marketed as air cleaners purposely release ozone, which can result in ozone concentrations several times the State standard, and directly harm sensitive occupants. Additionally, these air cleaners are ineffective at cleaning the indoor air. Many effective alternatives are available in the marketplace. Additionally, ionizers and electrostatic precipitators emit ozone to varying degrees as a by-product of their function, and such emissions may need to be limited. The market for air cleaners has increased substantially in recent years, and is expected to continue to expand (Freedonia, 2004).

- **Biological contaminants** are a high priority because of their ubiquitous presence and their widespread health and fiscal effects. Animal dander, pollen, house dust mites, and cockroaches cause millions of sensitive individuals to experience allergy symptoms and asthma attacks. Indoor mold has been an increasing problem in recent years, costing substantial sums of money for remediation and lawsuit settlements. Bacteria such as *Legionella* cause both serious and mild illness. Disease transmission can occur because infectious agents are emitted into the indoor environment. Infectious disease transmission is increased in indoor environments with crowded or dirty conditions and insufficient outdoor airflow. Mitigation actions for mold and some other biologicals might include required annual inspections and remediation in public buildings, group homes, and rental units, and in private homes at the time of sale. Certification requirements for mold assessors and remediators and other IAQ consultants would help assure the quality of inspections and remediation.

- **Building materials and furnishings** are a high priority for mitigation because they often emit multiple toxic air pollutants, especially when new, and have a high loading level in indoor environments, resulting in high exposure levels for occupants. A substantial percent of the population is exposed to such emissions due to the continued high rate of new building construction in California and the increasing number of home renovations undertaken by homeowners. Emission limits for pollutants emitted from building materials and furnishings (formaldehyde being the most predominant) would benefit all indoor environments and has potential for significant health benefits due to reduced incidence of asthma exacerbation, cancer, and eye, nose and throat irritation.

  Low-emitting alternatives are available. For example, non-wood alternatives and composite wood products made with phenol-formaldehyde resin have much lower formaldehyde emissions than composite wood products made with urea-formaldehyde resin, and could be substituted for some applications. In cabinets and furniture, all surfaces of these products can be coated or laminated to substantially reduce formaldehyde emissions. Building materials are currently available that meet Section 01350 emission requirements for formaldehyde and other chemicals of concern. A list of products for use in school construction projects that meet these requirements is available at http://www.chps.net/manual/lem_overvw.htm. These alternatives materials are available and should be required in public buildings, group homes, schools, and other buildings.

- **Combustion appliances** are also a high priority for mitigation. They can emit carbon monoxide, nitrogen dioxide, polycyclic aromatic hydrocarbons, particles, and other pollutants. These pollutants can have severe acute health effects including respiratory effects and exacerbation of asthma, and contribute to cancer risk. Reduced exposure to pollutants from gas and propane appliances, whether it be through emission limitations, active exhaust ventilation, or both, could have immediate widespread benefits for occupants in environments with such appliances. Precedence for mitigation of appliance emissions has been set in the state's low-income weatherization program.

  Statewide measures to reduce emissions from woodstoves and fireplaces both indoors and outdoors also are highly desirable. Such measures could have a major impact on improving both community-wide indoor and outdoor air quality in many areas of the state. Emission limitations, product re-design, product use restrictions, and improved venting can be used for reducing this type of pollution. A number of local government entities have recently approved regulations restricting the use of woodstoves and fireplaces: in the San Francisco Bay area, 24 cities have ordinances that prohibit conventional fireplaces in new construction. The mountain town of Truckee has a more aggressive policy that requires that existing unapproved wood burning appliances be removed by July 15, 2006. The San Joaquin Valley implemented a daily advisory for restrictions on residential fireplace or wood stove use on January 1, 2004 (http://www.valleyair.org/BurnPrograms/wood_burning.htm). Woodsmoke especially impacts those with asthma and other respiratory disease.

- **Environmental tobacco smoke** has been greatly reduced in California, primarily due to legislation that bans smoking at the workplace. However, children's exposures remain a special concern, because they can be highly exposed when smoking occurs in their home or in vehicles driven by family or friends who smoke. Actions to reduce children's exposure–such as an increased focus of public education on smoking parents, and reduction of smoking inside vehicles and homes with children–remain a high priority.

- **Radon** is ranked as a high priority due to its high estimated lung cancer risk. However, indoor levels in California are generally below the recommended mitigation level, and the need for mitigation is very building- and area-specific. The risk from radon is strongly associated with smoking (NRC, 1999a). Mitigation might include revised building codes and requirements for testing and implementation of construction changes, if needed, upon the sale of a home or building in areas with elevated radon levels. The California Department of Health Services (DHS) has established a list of certified providers of radon services.

Medium Ranked Source Categories

The pollutant source categories included in Table ES-3.2 are lower in priority than those above, but nonetheless include some sources that warrant consideration for mitigation.

- **Architectural coatings,** such as paints and lacquers, are available in "low VOC" versions due to formulation changes targeted toward reducing outdoor ozone. However, they are not directly regulated by the state. ARB develops Suggested Control Measures and provides guidance and technical assistance to air quality management districts in the state, 22 of which have adopted rules to reduce VOC emissions from coatings. Like building materials, architectural coatings are widely used and have a high loading in indoor environments when used, due to the large surface areas they typically cover. Additionally, some components of coatings can be harmful, but may not necessarily be addressed through reactive VOC reductions. However, because reductions have been achieved in districts that cover 95 percent of the California population, this source category is ranked as a medium priority.

- **Consumer products and personal care products** have been regulated by ARB to reduce emissions of reactive VOCs in order to reduce outdoor smog formation. Reactive VOCs, and some toxic air contaminants, have been reduced substantially through reformulation of a number of product categories. For example, in reducing VOC content to comply with ARB regulations, manufacturers often use water-based technologies and use VOC exempt compounds such as acetone. To prevent increased use of TACs, ARB has prohibited the use of perchloroethylene, methylene chloride, and trichloroethylene in 13 categories including general purpose degreasers, brake cleaners, all spray paints, all aerosol adhesives and adhesive removers. Additionally, antiperspirants and deodorants are not allowed to contain any compounds identified as TACs. The Board recently approved a rule to remove *para*-dichlorobenzene from solid air fresheners and toilet/urinal care products.

  However, despite the breadth of products addressed under ARB's consumer products regulations, not all types of consumer products have been regulated. Also, due to the nature of some products (household cleansers, air fresheners, stain removers, etc.), the user is in close proximity to the release of chemicals during use, and can experience high pollutant exposure when using the product. Thus, there is an apparent need to reduce emissions from consumer products to prevent high personal exposures and risks, and to address types of products not currently regulated under ARB's programs. Chemical reformulations, emission limitations, content limits, and/or product use restrictions of consumer products are mitigation approaches that could result in further significant risk reductions, especially for product users. Because of the ARB's progress to date with chemically formulated products, some of the highest emitting consumer products have been reformulated, and therefore this category is ranked medium rather than high.

Table ES-3.2 Medium Priority Source Categories for Mitigation [1]

SOURCES OF POLLUTANTS[2] (listed alphabetically)	EXAMPLES OF POLLUTANTS[3] EMITTED	POTENTIAL APPROACH TO MITIGATION[4]	DIRECT STATE AUTHORITY TO TAKE IAQ MITIGATION ACTIONS
Architectural Coatings (e.g., paints, sealants, lacquers, varnishes)	Formaldehyde, acetaldehyde, ethylene glycol, metals, others	Emission limitations, chemical reformulations, use restrictions to reduce TACs & nonreactive VOCs with health impacts;	No
Consumer Products (e,g.,household and institutional cleaners, furniture- and floor-care products, air fresheners, stain removers, detergents) **Personal Care Products** (e,g.,products used for hair and skin care)	Methylene chloride, para-dichlorobenzene, perchloroethylene, toluene, benzene, naphthalene, formaldehyde, acetaldehyde, metals, others	Emission limitations, chemical reformulations, and product use restrictions to reduce TACs and nonreactive VOCs with health impacts; labeling program	Limited (some indirect)
Household & Office Equipment and Appliances (computers, photocopy machines, vacuum cleaners)	Particles, styrene, some other VOCs, phthalates, ozone, PBDE	Emission limitations, local exhaust requirements	No
Pesticides (insecticides, herbicides etc, used indoors and outdoors; track-in, drift.)	Chlorpyrifos, diazinon, permethrin, DDT, dieldrin	Formulation and application changes for indoor use	Limited

1. Individual sources may be higher or lower than the source category ranking.
2. All of the examples of pollutant sources may not emit all of the pollutants listed in the corresponding box in column two.
3. Air pollutants may be identified as Toxic Air Contaminants (TACs) by the California Air Resources Board, and/or identified as Proposition 65 chemicals; or, criteria (traditional) air pollutants.
4. Public education, economic incentives, and non-regulatory approaches should also be used where appropriate. The actual approach taken would be determined after extensive discussions with the relevant industries, in consideration of costs, feasibility, and effectiveness..

- **Household appliances and office equipment** such as computers, copy machines, and vacuum cleaners can emit a variety of pollutants such as particles, ozone, various VOCs of concern, and PBDEs. In most cases, these pollutants are emitted directly into the living or working area, and thus are of concern. Additionally, more and more office equipment is being purchased for use in the home, increasing the number of people potentially exposed. Emissions from each type of appliance could be addressed through emission limitations and/or requirements for local exhaust of the emissions. Because emissions information on many specific sources in this category is outdated or lacking, mitigation efforts for these products are a medium priority.

- **Pesticides** are used indoors and around the perimeter of buildings to control household pests such as ants, spiders, and cockroaches. Pesticides residues may be more persistent indoors than outdoors due to the lack of natural degradation forces such as ultraviolet light, high temperatures, wind, and rain. Pesticides can be tracked in from outdoor application and drift into the home after outdoor spray application. In rural areas indoor concentrations may be greater due to increased use of pesticides for agricultural purposes. Levels of pesticides have been measured in both air samples and house dust samples. Continued research and intervention by the U.S. EPA and DPR are needed to assure the least toxic pesticides are registered for indoor use and the formulations prohibit excessive human exposure. Most importantly, the implementation of integrated pest management approaches should be expanded to reduce the need for pesticide application. The U.S. EPA banned the use of chlorpyrifos and diazinon in indoor environments in 2000 and 2001, respectively. The California Department of Pesticide Registration (DPR) also governs the use of pesticides.

VIII. OPTIONS TO MITIGATE INDOOR AIR POLLUTION

This report has shown that there are many sources of indoor air pollution that produce substantial adverse health effects, result in lost productivity, and require considerable expenditures for health care. Despite these facts, there is no systematic program to improve indoor air quality, there are relatively few regulations or standards to specifically address indoor air quality problems, and few resources focused on effectively addressing problems and promoting improvements. Current efforts to address indoor pollution are not commensurate with the scope of the risk to health it poses to Californians.

General Mitigation Options

Ambient (outdoor) air quality is protected through a comprehensive system. In California and under federal law, ambient air quality standards are established for traditional (criteria) pollutants and must be attained. Under other state authority, pollutants identified as toxic air contaminants must be reduced to the maximum extent feasible. The approach used to reduce toxic air contaminants in ambient air, in which source emissions are reduced without setting enforceable air quality levels, seems most applicable to indoor air, together with increased attention to proper building operation and maintenance. Action to reduce indoor emissions and exposures would assure reduction of exposure and risk from key sources, and should be a major component of a new effort to address indoor air. Other approaches including public education, product testing and labeling, improved building codes, and setting of maximum exposure guideline levels, should also be part of the mitigation program. The following elements of an indoor air pollution reduction program are recommended for consideration:

1. **Create a management system for indoor air quality** that establishes a comprehensive program for assessing indoor health problems, identifying the actions needed to reduce the most significant problems, and setting guidelines, emission limits, or other requirements that will be effective in reducing the health impacts of indoor sources. As discussed in Sections 4 and 6 of this report, many agencies' actions affect indoor air quality, and a few have limited authority over some aspect of indoor air quality, but no state (or federal) agency has the authority or mandate to conduct a comprehensive indoor air pollution mitigation program. Such a program is needed, and should be fully coordinated with activities of other agencies whose actions affect indoor air.

2. **Establish emission limits,** when needed, for indoor pollutant sources that pose excessive risks due to their indoor emissions. These might include air cleaners, building materials, furnishings, combustion appliances, and others. While ventilation authority exists in the Energy Commission and Cal/OSHA, no state agency has a direct mandate to establish emission limits for indoor sources for the purpose of reducing indoor exposure and risk. Establishment of such limits would better protect public health, and may reduce (but not eliminate) the amount of ventilation needed under certain circumstances in some buildings, thus saving energy. Compliance could be accomplished by requiring emissions testing through an independent laboratory certified by the state, and submittal of the data to the lead agency.

3. **Require manufacturers to submit building materials, furnishings, combustion appliances, consumer products, and other significant sources for emissions testing** by an independent laboratory certified by the state, and to report those results to the state and to the public. Also, require results to be transmitted to the public via product labeling or accompanying materials in language consumers can understand. Implementation of a required test program could prove to be an effective approach, at least for reducing indoor pollutant levels in new buildings. A prototype emissions testing program has already been developed for reducing toxic VOCs from building materials and furnishings, and is designed to protect human health state sustainable building projects (Section 01350, State of California, 2002). A partial list of products that meet this specification is available at http://ciwmb.ca.gov/GreenBuilding/Specs/EastEnd/. However, there is currently no requirement for state agencies or others to use these guideline emission specifications, and only limited incentive for them to do so. Other national and international emissions test protocols that are widely used also are available.

4. **Make children's health in schools, homes, and care institutions the top priority.** Implement the recommendations for schools in Section 7.2 of this report. In schools and public daycare centers, require the use of building materials that are certified to be low-emitting, and require that school HVAC sytems be quiet (under 45 decibels) and well maintained. Increase efforts to reduce children's exposure to environmental tobacco smoke. Increased education and outreach efforts to smoking parents and caretakers are needed to inform them of the health dangers of second-hand smoke, and the actions they should be taking to protect children under their care from these dangers.

5. **Develop indoor air quality guidelines** for homes, schools, offices, and institutional living quarters. These would largely identify "Best Practices" for the design, construction, operation and maintenance of public, commercial, school, and institutional buildings. In some cases, they might include the identification of healthful levels or "bright lines" for some pollutants to be used as goals for mitigation activities and "best practices", but would not have an associated compliance program. They should also include performance measures for buildings and appliances, and valid certification requirements for professionals directly involved in indoor air quality-related occupations. Full commissioning should be required for all new public, commercial, and institutional residential buildings, to assure that they are constructed and operate as intended, and that they provide acceptable indoor air quality. ASHRAE Guidelines (1993, 1996) provide basic guidance for building commissioning, but state requirements are needed.

6. **Amend building codes to address indoor air quality, with a focus on assuring adequate ventilation under all circumstances.** For example, unvented cook stoves, ovens, and combustion appliances should not be allowed in residences. They should be

vented to the outdoors, such as through direct venting or an automatic (but quiet) exhaust fan that is activated when the appliance is turned on. Similarly, building codes should be established and enforced to prevent mold problems, residential ventilation issues, and others.

7. **Fund an outreach and education program focused on professionals,** including health professionals, teachers, school facility managers, and others who must be able to identify and remedy indoor air quality problems. Such individuals have many obligations, yet play a key role through their occupation in initial identification, prevention, and mitigation of indoor air quality problems. Most need more in-depth information and training on indoor air quality than they typically have had. Training and technical assistance should be provided for the private sector to develop the skills and services needed for high-quality building commissioning, operation, and mainenance.

8. **Conduct more research on indoor air quality**. Several high priority areas are specifically identified in this report for further research. Because of the known serious health impacts of ambient PM and recent studies showing high emissions of PM from indoor sources, research on the health effects of indoor PM are a high priority. The health effects of terpene-ozone reaction products and the extent of people's exposures to them, as well as other indoor chemical reaction products, also are key areas warranting focused research. There are many new chemicals introduced into the product mix each year, yet few have had full health and exposure studies completed. The effects of more recently identified indoor chemicals, such as PBDEs, warrant further investigation. Improved methods and protocols to detect indoor dampness and hidden mold growth are also needed. Synergistic and cumulative health effects are suspected for a number of indoor pollutants with similar structures or properties, yet little research has been conducted in this area. Finally, mitigation approaches assumed to be effective have sometimes been found to be much less effective than anticipated; the effectiveness of recommended or required mitigation measures should be confirmed through appropriately designed studies to assure that the necessary reductions in exposure and risk will be achieved.

9. **Fund an Innovative Clean Air Technology program (ICAT) for indoor air quality** to foster the development and commercialization of legitimate, cost effective technologies that can improve IAQ. For example, improved low-noise ventilation technologies, improved air monitors and assessment tools, and effective low-noise air cleaners are needed. ARB's current ICAT program, focused on improving outdoor air quality through improved technology, has been very successful in bringing new technologies to commercialization in California, adding new options for reducing air pollution while also bringing jobs and investment into the state. An indoor air quality ICAT program would be expected to do the same.

All of these suggested mitigation options are feasible if appropriate mandates and resources are provided. The feasibility of individual measures, such as emission limits for a specific type of product, cannot be determined without substantial additional information. As discussed in this report, alternative products or formulations are already available for some of the indoor sources of current concern. However, prior to taking any regulatory action, a more detailed assessment of the specific remedies available, including technological and economic feasibility, would be needed. Additionally, like ARB's current regulatory programs, any emission limitations or other mitigation measures should be developed with continuous discussion and review by stakeholders, the public, and other state agencies.

Mitigating Indoor Pollution in Schools: An Urgent Need

The Air Resources Board and Department of Health Services recently completed a statewide study of kindergarten through 12th grade public schools entitled "Environmental Health Conditions in California's Portable Classrooms" (ARB/DHS, 2003). Results showed there are a number of serious, widespread environmental health problems in California schools that need to be addressed. These problems were found in both portable (relocatable) and traditional (site-built) classrooms. Government standards and guidelines that are designed to protect children in classrooms and other buildings are essentially lacking; thus, results were compared to the most relevant environmental health guidelines and standards available, primarily from professional societies and government agencies.

Problems in Schools

The primary problems found include:

- Inadequate ventilation with outdoor air during 40 percent of class hours, and seriously deficient ventilation 10 percent of the time. This is often due to teachers turning off HVAC systems because of excessive noise.

- Formaldehyde air concentrations exceeded guideline levels for preventing acute eye, nose, and throat irritation in about 4 percent of the classrooms; nearly all classrooms exceeded guidelines for preventing long-term health effects, including cancer.

- Obvious mold in about 3 percent of classrooms, and water stains and other potential mold indicators in about one-third of classrooms, due to inadequate maintenance.

- Noise levels in all classrooms exceeded 35 decibels, a voluntary standard for classrooms; one-half of the classrooms also exceeded 55 decibels, the level used for outdoor nuisance regulations. Excess noise was primarily attributable to noisy ventilation systems.

Recommendations to Address the Problems Identified

Recommendations to address the problems identified in the study were developed in consultation with state agencies, industries, school officials, and other interested stakeholders. Actions are needed at all levels. A total of 16 recommendations are discussed in the November, 2003 Report to the Legislature. These are presented in two groups in the report: Group 1 includes high priority, high benefit actions that can be achieved at relatively low cost and should be accomplished in the near term, while Group 2 recommendations will require a longer timeframe and/or more substantial resources to accomplish. The recommendations fall into four general approaches needed to remedy and prevent the problems seen. These include:

- Direct and assist schools to comply with state regulations, especially Cal/OSHA's workplace regulations related to ventilation, moisture intrusion, and other aspects of building operation and maintenance. Schools should conduct a self-assessment and implement an indoor air quality management program, like that in U.S. EPA's *IAQ Tools for Schools Program.*

- Develop and promote "Best Practices" for design, construction, operation, and maintenance of school facilities. The CHPS manuals provide comprehensive guidance at no charge.

- Improve support (both funding and training) for school facilities and staff. Stable, long-term funding mechanisms are needed to assure adequate and timely operation and maintenance.

Postponed maintenance often results in greater costs. Focused training programs for administrators, facility managers, and teachers are needed: those closest to the classroom are often not aware of current "best practices" for operation and maintenance of classrooms.

- Establish guidelines and standards for school environmental health that are protective of children. Noise, lighting, and chemical contaminant levels appropriate for school children need to be identified.

Some actions have already been taken to begin to address these problems; however, they constitute only a first step toward realizing actual improvements in school conditions. Only a small percentage of schools and districts have actively pursued the many tools that are readily available to them to improve the school environment. The CHPS' *Best Practices Manuals*, U.S. EPA's *IAQ Tools for Schools Kits*, and the LAUSD's "Safe School Inspection Guidebook" are all available on the Internet free of charge, yet the number of California schools utilizing these tools is small. A proactive effort to implement the recommendations of the report is needed.

The complete *Report to the Legislature on Environmental Health Conditions in California's Portable Classrooms* is available at http://www.arb.ca.gov/research/indoor/pcs/pcs.htm.

IX. SUMMARY

Indoor pollution causes substantial, avoidable illness and health impacts – ranging from irritant effects to asthma, cancer, and premature death – and costs Californians billions of dollars each year. Because there are numerous sources of pollutants in indoor environments, and because people spend most of their time indoors, exposure and the associated risk is substantial. Many agencies, professional groups, and organizations have taken actions to reduce indoor pollution, but these have been piecemeal and are not sufficiently effective in addressing the problem.

There are many actions that could be taken to significantly reduce indoor emissions and exposure. If experience in controlling sources of outdoor pollution is repeated relative to indoor sources, many of these measures will be low cost and will provide substantial health benefits. A focused risk reduction program is needed to effectively assure acceptable indoor air quality in California homes, schools, and public buildings. A program that stresses direct emission reductions and includes improved building codes and ventilation, increased public and professional education, product labeling, and advisory standards is recommended. Indoor air cleaning devices (especially ozone generators), biological contaminants, building materials and furnishings, combustion appliances including woodstoves and fireplaces, ETS, and radon are high priority source categories for mitigation. Architectural coatings, consumer products, household and office equipment, and pesticides are also of concern, but are a lower priority than the other categories identified. Special priority should be paid to measures that reduce children's exposures.

It should be noted that indoor air controls cannot be substituted for the state and national ambient air quality programs. As discussed above, indoor and outdoor pollution operate in tandem, increasing the health risk to all Californians. That means that any new initiatives to mitigate indoor air pollution must be accomplished alongside California's decades-long efforts to improve our outdoor environment.

1. INTRODUCTION AND BACKGROUND

The Air Resources Board (ARB) prepared this report to the Legislature on indoor air quality in response to requirements of Assembly Bill 1173 (Keeley, 2002; Health and Safety Code Section 39930; see Appendix I). As required by the legislation, this report summarizes the best scientific information available on indoor air pollution, including:

- Common indoor pollutants and their sources.
- The potential health impacts of indoor pollutants and associated costs.
- Existing regulations and current industry practices.
- Options for mitigation in schools, non-industrial workplaces, homes, and other indoor locations.
- State and federal efforts related to control of biological and radiological substances.

In preparing the report, the ARB consulted with the Department of Health Services (DHS), the Office of Environmental Health Hazard Assessment (OEHHA), the California Energy Commission (CEC), the Department of Industrial Relations (DIR, Cal/OSHA), relevant industries and other stakeholders, and interested members of the public. On April 4, 2003 a public workshop was conducted to inform stakeholders about the report and the anticipated schedule, and to solicit comments from them. On June 30, 2004, another workshop was held to receive comments on the draft report. A website and an email list serve were established to keep stakeholders informed of progress on the report preparation. The website is available at http://www.arb.ca.gov/research/indoor/ab1173/ab1173.htm, which also includes information on how to sign up for the email list serve. ARB released a revised draft report in November 2004 for review by interested stakeholders and a University of California scientific peer review panel. A subsequently revised draft report is now considered by ARB Board members before being forwarded to the Legislature.

This report reflects key points from the large body of knowledge that has been generated about indoor air quality since it became a concern in the 1970s. Researchers worldwide have made great progress in identifying indoor pollutants and understanding their relationship to human exposure and resultant health effects. Several international conferences are now held annually, and one journal, *Indoor Air*, is devoted exclusively to the field of indoor air quality. Many additional journals carry articles on indoor air quality. This report summarizes key findings of the most recent literature, with emphasis on the major trends identified by multiple investigators.

1.1 INDOOR POLLUTION POSES A SIGNIFICANT HEALTH RISK

State and federal comparative risk projects have repeatedly ranked indoor pollutants and sources in the high-risk categories of their analyses relative to other environmental health problems. In the 1994 *California Comparative Risk Project* (CCRP, 1994), the Human Health Committee ranked risks by two different methods: by sources and media, and by pollutant. In the sources and media ranking, the "residential and consumer product releases to air (indoor air)" category was ranked in the high risk group, along with some outdoor air pollution categories. In the pollutant rankings, ETS, radon, PM, and VOCs were all ranked in the high-risk category, and carbon monoxide and lead were ranked in the medium risk category. The results of the CCRP had a somewhat more substantial scientific basis than most other comparative risk projects, because it was based to a greater degree on actual measurement data, and used distributions of exposure and risk, rather than just population averages. Additionally, it was reviewed by an external scientific review committee.

The U.S. EPA's 1987 national comparative risk project also ranked indoor air pollution high relative to other environmental areas: indoor pollution other than radon was ranked fourth among the top thirteen national environmental problem areas assessed for cancer risk (U.S. EPA, 1987a). Radon was ranked first, but the risk from radon has been subsequently re-examined and reduced. A number of other states and regions have conducted comparative risk projects as well, with results similar to those of the U.S. EPA and California.

The high ranking of indoor pollution relative to other environmental problems is not surprising, because there are numerous sources of pollutants indoors, indoor air concentrations of some pollutants are often high enough to pose a health risk, and people spend most of their time indoors. The total quantity of air pollutants emitted indoors is much less than that emitted by outdoor sources. However, once emitted, indoor pollutants are diluted much more slowly than pollutants from outdoor sources. When this factor is combined with the fact that Californians, like others from industrialized nations, spend most of their time indoors, there is a much higher likelihood that people will be exposed to pollutants emitted indoors than those emitted outdoors.

> *The Rule of 1000:*
>
> ***A typical pollutant release indoors is 1000 times as effective in causing human exposure as the same release to urban air.***
>
> KR Smith, in Air Pollution: Assessing Total Exposure in the United States, *Environment,* 30 (8): 10, 1988.

Investigators have developed concepts and terms to quantify the portion of pollutant emissions actually inhaled (Bennet *et al.*, 2002; Lai *et al.*, 2000). The inhalation transfer factor (ITF) is defined as the pollutant mass inhaled by an exposed population per unit mass emitted from an air pollution source (Lai *et al.*, 2000). Although more people are exposed to a pollutant released outdoors, the concentration is usually reduced due to wide dispersion, relative to a pollutant emitted indoors. Calculated inhalation transfer factors were several orders of magnitude greater for pollutants emitted indoors and in vehicles than those emitted outdoors, thus indicating a significantly larger fraction of pollutant is inhaled when it is released indoors as opposed to outdoors.

Investigators calculate that pollutants emitted indoors have a 1000-fold greater chance of being inhaled than do those emitted outdoors (Smith, 1988; Bennet *et al.*, 2002; Lai *et al.*, 2000). Thus, reducing indoor emissions by a given amount might be anticipated to have a greater impact on reducing exposure than would reducing outdoor emissions by that same amount. Regulation of outdoor sources such as motor vehicles and industrial plants has notably reduced many outdoor pollutant levels in California. Now, there are significant gains to be achieved in public health protection from reductions in indoor source emissions and other measures that might be taken to reduce indoor concentrations and exposures.

The health effects of indoor pollutants range from irritant effects to respiratory disease, cancer, and even sudden death. Indoor sources of pollutants are numerous, such as building materials, consumer products of all types, combustion appliances, and even some so-called "air fresheners". Common indoor activities such as cooking, cigarette smoking, burning candles, and vacuuming also generate pollutants. The health effects of indoor pollutants, and indoor pollutant sources and concentrations in California, are discussed in Chapter 2 of this report.

1.2 PEOPLE SPEND MOST OF THEIR TIME INDOORS

A key reason indoor pollution is so critical to health is that Californians, like others from industrialized nations, spend most of their time indoors – about 87%, on average. So, if pollutants are present indoors, there is a high likelihood that people will be exposed to them. As shown in Table 1.1, California adults spend an average of about 62% of their time in their home. Children spend even more time in their home; infants up to 2 years of age spend 85% of their time inside the home, on average. Thus, the home is a critical exposure microenvironment for all, and especially for children.

Table 1.1: Average Percent of Time Californians Spend in Major Locations

AGE	AVERAGE PERCENT OF TIME			
	Inside the Home	Other Indoors	Outdoors	Inside a Vehicle
Children[1] 0 - 2	85	4	7	4
3 - 5	76	9	10	5
6 - 11	71	12	13	4
All Children (0 - 11)[1]	76	10	10	4
Adults and Teens[2]	62	25	6	7

1. From: *Study of Children's Activity Patterns* (Wiley *et al.*, 1991a, ARB Contract No. A733-149; Phillips *et al.*, 1991).
2. From: *Activity Patterns of California Residents* (Wiley *et al.*, 1991b, ARB Contract No. A6-177-33; Jenkins *et al.*, 1992a).

1.3 CHILDREN'S HEALTH – VULNERABILITY IMPLIES INCREASED INDOOR AIR QUALITY IMPACT

In recent years there has been an increasing awareness that children may be more susceptible than adults to the harmful effects of air pollution. Additionally, children are more highly exposed to some indoor contaminants than are adults. Thus, children are likely at greater risk from indoor pollution than adults.

- **Children's physiology and developing lungs and bodies make them more susceptible to chemicals that affect development and lung function.** Children's immune systems are not fully developed and their growing organs and structures are more easily harmed. For example, lead is more readily absorbed from the digestive tract of children, and the developing central nervous system is more susceptible to damage than that of an adult. Pollutants that cause irritation or inflammation in the airways are more likely to obstruct a child's airways because they are narrower than airways of an adult. Results from the ARB-funded Children's Health Study indicate the lungs of children in high-pollution communities develop more slowly and move air less efficiently than lungs of children in low-pollution communities (Gauderman *et al.*, 2000). When exposed to ETS, children are at greater risk than adults for developing lower respiratory tract infections, bronchitis, pneumonia, fluid in

the middle ear, and asthma symptoms (NCI, 1999). Additionally, young children appear to be more susceptible to the effects of environmental tobacco smoke (ETS) than older children (IOM, 2000).

- **Infants and children inhale more air relative to their size** than do adults at a given level of activity (ARB/OEHHA, 2000; Adams, 1993). Additionally, children often breathe through their mouths, bypassing the filtering effect of the nose and allowing more pollutants to be inhaled. Thus, for the same amount of time spent in a given location/activity as an adult, a child will inhale a greater quantity and thus receive a greater dose of the chemicals in the air. Children also have a larger lung surface area per unit of body weight, relative to adults. This contributes to a higher breathing rate/unit surface area and elevated exposure in children (Thurlbeck, 1988; Plopper and Thurlbeck, 1994).

- **Children's activities bring them into close proximity to indoor sources.** First, infants and young children spend more time indoors at home (see Table 1.1) than do adults. Additionally, younger children spend more time near indoor sources such as operating gas stoves, e.g., near the parent while cooking (Phillips *et al.*, 1991), leading to higher exposures to nitrogen dioxide and other cooking emissions.

- **Also, children spend more time on floors and more frequently put fingers and objects into their mouth** (Zartarian *et al.*, 1998; Zartarian and Leckie, 1998). This can lead to additional dermal and ingestion exposure to airborne TACs deposited and adsorbed onto floor dust, such as lead and other toxic metals, PAHs, and pesticides. Because the breathing zone for an infant or small child is several inches to a foot or so above the floor, while that for adults is several feet above the floor, particles stirred up by activity may become available to be inhaled by the child but are not elevated in the breathing zone of adults (Bearer, 1995).

Concerns about children's health prompted the California Legislature to pass the Children's Environmental Health Protection Act (SB 25, Escutia) in 1999. This Act requires Cal/EPA to specifically consider children when setting Ambient Air Quality Standards (AAQS) and reviewing pollutants for identification or regulation as Toxic Air Contaminants (TACs). As required by this legislation, ARB and OEHHA have re-evaluated the AAQS for particulate matter and are currently evaluating the AAQS for ozone and nitrogen dioxide. OEHHA has identified dioxin, lead, polycyclic organic matter, diesel exhaust particles, and acrolein as the top 5 priority TACs that may impact infants and children the most (OEHHA, 2001). The ARB is beginning to assess the adequacy of existing control measures for these compounds relative to the health of children.

1.4 ENVIRONMENTAL JUSTICE CONSIDERATIONS RELATIVE TO INDOOR AIR QUALITY

Senate Bill 115 (1999) directs the California Environmental Protection Agency to design an environmental justice mission statement for boards, departments, and offices within the agency. State law (California Government Code § 65040.12c) defines environmental justice as the fair treatment of people of all races, cultures, and incomes with respect to the development, adoption, implementation, and enforcement of environmental laws, regulations, and policies. In response to SB 115, the ARB approved *Policies and Actions for Environmental Justice* in December, 2001. This document establishes a framework for incorporating environmental justice into all ARB programs, policies, and regulations (ARB, 2001a).

The ARB has taken several steps to directly and indirectly address environmental justice concerns related to indoor air quality. Special air-monitoring studies have been conducted in classrooms, homes, and at schools in communities located near industrial sources of pollution and/or heavy vehicular traffic. Preliminary results indicate pollutant levels in the selected communities are similar to levels in other communities. To assure that information is available to all stakeholders, documents designed for public education, such as fact sheets and a school advisory, have been published in both English and Spanish.

Census statistics indicate that the poor are more than three times as likely (22% versus 7%) to have substandard-quality housing (Evans and Kantrowitz, 2002) and that blacks and low-income persons are more likely than the general population to be in housing with severe physical problems (Krieger and Higgins, 2002). Children in low-income families may bear additional burdens because they are more likely to be in school buildings that have environmental problems; poor plumbing, inadequate heating, and poor indoor air quality (Evans and Kantrowitz, 2002).

Research is needed to determine the complex relationship between socioeconomic status (SES), environmental factors, and health status, particularly as they relate to indoor air pollution. To date, only factors related to the prevalence of asthma have received substantial study. The prevalence of asthma appears to be more strongly correlated with lower socioeconomic status than with race and ethnicity (IOM, 1993). Yet, California data show that African Americans, American Indians, and Alaska Natives experience a higher prevalence of lifetime asthma than other groups in the population (Meng *et al.*, 2003). Identifying the contribution of exposure to biological agents to poor health is difficult. The increase in asthma prevalence is greatest in children from lower income homes, probably due to an increase in prevalence of allergic conditions in their homes (Auinger *et al.,* 2003). Dust mites and cockroaches are important triggers for asthmatics that are more likely to be present in urban settings (IOM, 1999). Reviewing data accumulated in the second National Health and Nutrition Examination Survey (NHANES II) and the Harvard Six Cities Study, Eggleston (2000) concluded that ethnicity, poverty, and residence combined to influence asthma prevalence in inner-city children in ways that could not be easily disentangled (IOM, 2004).

"Economic factors [also] may encourage poor building practices. Combinations of pressure to build quickly and cheaply can result in poorly constructed buildings that are more likely to have water leaks.... Poverty combined with the lack of affordable housing may also create incentives to forgo or limit investment in maintenance that might help to prevent moisture problems and subsequent adverse impacts on the health of the occupants" (IOM, 2004). Those who live in substandard housing are exposed to more pests, as well as pesticides to get rid of them (Flynn *et al.*, 2000). Pesticides adsorb onto particles and accumulate in the carpet, where children and others can be exposed to them. In an effort to improve their environment, a higher percent of low-income individuals use room fresheners – products that may introduce additional toxic chemicals to the indoor environment (Wiley *et al.*, 1991b).

Children living in urban areas are disproportionately exposed to lead, primarily from lead-containing paint that has been used on older houses. Exposure to lead in both house dust and air takes a toll on children. Research indicates blood lead levels are higher for poor and minority children in central cities (IOM, 1999).

2. HEALTH EFFECTS, SOURCES AND CONCENTRATIONS OF INDOOR AIR POLLUTANTS

Emissions from indoor sources contribute to exposure and risk in two ways. Some pollutants are emitted in substantial quantities over extended periods from large surfaces, such as formaldehyde from composite wood products made with urea-formaldehyde resin. These sources contribute to elevated indoor pollutant levels in many buildings where a large portion of the population spends their time. Large numbers of individuals can be affected due to the large quantity of indoor emissions. Other products, such as aerosol sprays or solvents, emit much smaller quantities of pollutants, and are used by a subset of the population. However, those who use such products use them in such a manner that the chemicals emitted are released near the user's breathing zone (area near the nose and mouth). A high concentration of the chemical consequently may be inhaled during product use before the chemical has a chance to dilute in the air.

It is important to note that health effects are determined not only by the specific toxicology of the air pollutant, but also by the exposure and absorbed dose. The higher the exposure and dose, the higher the risk of adverse health effects. In addition, more severe effects generally occur with higher doses. It is not possible in this document to describe the dose-response relationship for all indoor air pollutants. Further information on dose-response relationships can be found on the OEHHA website (www.oehha.ca.gov).

Hundreds of substances representing a range of chemical, physical, and biological species have been identified as indoor air pollutants. Indoor air pollutants include volatile organic compounds (VOCs), inorganic gases, particulate matter (PM), and complex mixtures such as environmental tobacco smoke (ETS). Indoor concentrations of many pollutants are often higher than outdoor concentrations due to the use of indoor pollutant sources in a confined space. Some pollutants such as formaldehyde, radon, asbestos, cigarette smoke, and mold have received a substantial amount of study. Other pollutants have received minimal study, and undoubtedly some pollutants have not yet been identified.

Indoor air pollution can cause a variety of adverse impacts on human health, from irritant effects to respiratory disease, cancer, and death. The major health effects that can occur from exposure to common indoor pollutants are indicated in Table 2.1. These effects have a major impact on Californians' health each year, and most can be readily avoided. Asthma, cancer, irritant effects, and sick building syndrome are discussed below. Later in this chapter the more common indoor pollutants, their sources, and indoor concentrations are discussed.

2.1 KEY HEALTH IMPACTS

2.1.1 Asthma

Asthma is a chronic inflammatory lung disease that results in partially reversible constriction of the airways. It is characterized by episodes of wheezing, shortness of breath, or coughing that may occur at any time. Asthma is a critical health issue because of its negative impact on the quality of life, increased morbidity and mortality, and substantial economic impact.

Table 2.1. Sources and Potential Health Effects of Major Indoor Air Pollutants

POLLUTANT	MAJOR INDOOR SOURCES	POTENTIAL HEALTH EFFECTS ASSOCIATED WITH ONE OR MORE OF THE POLLUTANTS LISTED*
Asbestos	Building materials in older homes released during renovation, naturally occurring in some soils	Lung cancer, asbestosis, mesothelioma
Biological Agents (bacteria, fungi, viruses, house dust mites, animal dander, cockroaches, microbial VOCs)	House and floor dust, pets, bedding, poorly maintained air- conditioners, humidifiers, dehumidifiers, moist structures or furnishings, insect infestation, building occupants	Allergic reactions; asthma, eye, nose, and throat irritation, humidifier fever, influenza, and other infectious diseases
Carbon Monoxide	Unvented or malfunctioning gas and propane appliances, wood stoves, fireplaces, tobacco smoke, motor vehicles in attached garages	Headache, nausea, angina, impaired vision and mental functioning, fatal at high concentrations
Endocrine Disruptors (phthalates; DDT, chlordane, heptachlor, *o*-phenylphenol, PBDEs)	Plastics, pesticides, flame retardants	Mimic or block natural effects of hormones (estrogen and others); developmental abnormalities
Environmental Tobacco Smoke (ETS)	Cigarettes, cigars, and pipes	Respiratory irritation, bronchitis and pneumonia in children, asthma in preschool children, lung cancer; heart disease, aggravated asthma, decreased lung function
Formaldehyde, Other Aldehydes	Composite wood products such as plywood and particleboard, furnishings, wallpaper, durable press fabrics, paints, combustion appliances, tobacco smoke	Cancer, eye, nose, and throat irritation, headache, allergic reactions, aggravated asthma, decreased lung function
Lead	Lead paint chips, contaminated soil	Learning impairment
Nitrogen Dioxide	Unvented or malfunctioning gas appliances, other combustion appliances	Aggravated asthma, decreased lung function, eye, nose, and throat irritation, increased respiratory disease in children
Organic Chemicals (benzene, chloroform, para-dichlorobenzene, methylene chloride, perchloroethylene, phthalates, styrene, others)	Solvents; glues, cleaning agents, pesticides, building materials, paints, treated water, moth repellents; dry-cleaned clothing, air fresheners	Cancer; eye, nose, throat irritation, aggravated asthma, decreased lung function; headaches, at high levels: loss of coordination, damage to liver, kidney and brain
Ozone	Infiltration of outdoor air, ozone generating air "purifiers", office machines	Lung inflammation, aggravated asthma, cough, wheeze, chest pain
Particulate Matter	Cigarettes, wood stoves, fireplaces, cooking, candles, aerosol sprays, house dust	Increased mortality and hospital admissions, lung cancer, eye, nose, throat irritation, increased susceptibility to sinus and respiratory infections, bronchitis; aggravated asthma, decreased lung function
Polycyclic Aromatic Hydrocarbons (PAH)	Cigarette smoke, cooking, woodburning	Cancer, gene mutation
Radon	Uranium-bearing soil under buildings, ground-water, construction materials	Lung cancer (especially in smokers)

* Please note that when multiple pollutants are listed in a group, each pollutant may not cause all of the health effects listed in the third column.

A number of indoor pollutants can cause or exacerbate asthma and chronic bronchitis. Indoor biological agents are associated with these diseases (IOM, 2000); however, it is clear that biological agents alone cannot explain the increase in asthma over the last few decades. The recent rise in asthma prevalence has been too rapid to be attributed to genetic factors and biological allergens alone: indoor and outdoor air pollution have been identified as potentially important contributors to the increase of asthma (McConnell *et al.*, 2002; Platts-Mills and Carter, 1997; Duhme *et al.*, 1998; Karol, 2002).

Over the past three decades, asthma prevalence has been on the rise in industrial nations, and the death rate due to this disease has doubled (Karol, 2002). The Centers for Disease Control and Prevention (CDC) reported that in the year 2000, asthma was responsible for 4,487 deaths in the U.S., as well as approximately 465,000 hospitalizations, 1.8 million emergency department visits, and 10.4 million visits to physicians across all age groups (CDC, 2003). Currently, about 7.2% of adults in the U.S. have asthma (CDC, 2003). According to 2001 data, 11.9% of Californians, or 3.9 million people, have asthma (CHIS, 2003). California also has higher rates of asthma mortality than the nation as a whole. The reasons for these elevated rates in California are unknown at this time. Children have been particularly hard-hit; from 1980 to 1994, there was a 160% increase in asthma prevalence in those up to 4 years of age in the U.S. (Mannino *et al.*, 1998). In California, asthma prevalence is highest among children 12-17 years of age. Asthma is one of the leading causes of school absenteeism in children, and results in missed workdays and lost productivity in adults. Asthma affects all races and ethnic groups, and both genders. Every year about 40,000 Californians are hospitalized and about 500 Californians die because of asthma. Asthma hospitalization and death rates are higher among African-Americans compared to other racial groups. Further, hospitalization rates among children are much higher than other age groups. Although the causes for the observed increase in asthma prevalence, hospitalizations, and death are not fully identified, indoor air pollution has been identified as a contributing factor.

In a recent report by the National Academy Institute of Medicine, entitled *Clearing the Air: Asthma and Indoor Air Exposures* (IOM, 2000), the Committee on the Assessment of Asthma and Indoor Air examined the scientific literature relating indoor air pollutants and other factors to asthma. Their key findings are shown in Tables 2.2 and 2.3. Recent reviews of the medical literature have also established that building dampness approximately doubles the risk for respiratory symptoms in building occupants and can exacerbate asthma. The agent(s) responsible for dampness-related increased risk for asthma exacerbation have not been conclusively identified but may involve some or all of the following: house dust mites, microbiological agents such as fungi or bacteria, or organic chemicals released during degradation of building materials or furnishings (Bornehag, 2001; 2004).

The IOM committee found that, in addition to the known biological asthma triggers such as house dust mites, cockroaches, and animal dander, chemicals in ETS can exacerbate asthma in preschool children. Evidence for an association between formaldehyde exposure and wheezing is limited, often due to confounding factors such as exposure to multiple pollutants. Airway responses such as wheezing, waking with shortness of breath, and asthma attacks have been associated with gas stove use (Jarvis *et al.*, 1996). Sufficient evidence exists to conclude that ETS can exacerbate asthma in preschool-aged children and provides an association between ETS exposure and the development of asthma (IOM, 2000). However, scientists found only limited or suggestive evidence of ETS as an asthma trigger in older children and adults, and insufficient evidence to consider it a causal factor in these groups (IOM, 2000). The IOM committee noted that studies have linked outdoor PM with respiratory problems, and that outdoor fine particles, PM2.5, readily enter the indoor environment. Studies that have addressed

asthma specifically have found some evidence for asthma exacerbation due to outdoor PM exposure (IOM, 2000).

Although the Committee on the Assessment of Asthma and Indoor Air found inadequate evidence to link VOCs with exacerbation of asthma, more recent studies have found an association between residential VOCs to asthma and its symptoms. Delfino (2002) published a review of the epidemiological evidence for links between air toxics and asthma. Delfino cites Swedish studies that showed that self-reported asthma prevalence in school children increased with increasing VOC levels, and asthmatic adult symptoms occurred in association with toluene, C_8-aromatics, terpenes, formaldehyde, and limonene. Adult asthma prevalence, wheeze, and blood eosinophil concentrations were higher in newly painted homes, consistent with higher VOC levels (particularly 2,2,4-trimethyl 1,3-pentanediol diisobutyrate and formaldehyde) measured in such homes. In another European study cited by Delfino, elevated levels of benzene and styrene were associated with respiratory infections in newborns with compromised health. The newborns had either low birth weight or an abnormal immune response (indicated by high levels of IgE in cord blood). In the same study, wheezing was related to house painting and carpet installation during the first year of life. Delfino cautions that in these studies, the effects seen may be subject to confounding by other causal agents.

Table 2.2. Indoor Exposures and Exacerbation of Asthma

Sufficient Evidence of a Causal Relationship • Cat • Cockroach, House dust mite • ETS (preschoolers)
Sufficient Evidence of an Association • Dog • Fungi or molds, Rhinovirus • NO_2, NO_X (high-level exposures)
Limited or Suggestive Evidence of an Association • Domestic birds • *Chlamydia pneumonia, Mycoplasma pneumoniae*, Respiratory Syncytial Virus (RSV) • ETS (school-aged children and adults) • Formaldehyde, Fragrances
Inadequate or Insufficient Evidence to Determine Whether or Not an Association Exists • Cow, Horse, Rodents • *Chlamydia trachomatis*, Endotoxins • Houseplants, Pollen • Pesticides, Plasticizers, VOCs • Insects other than cockroaches

Source: IOM, 2000

Delfino's (2002) review identified several links between asthma symptoms and indoor air pollutants, especially formaldehyde:

- A relationship exists between formaldehyde exposure and occupational asthma.
- Children in homes with formaldehyde concentrations greater than 41 ppb are more often diagnosed with asthma and chronic bronchitis.
- Adults show more wheeze, chronic cough, and lower peak expiratory flow in homes with higher formaldehyde concentrations.
- Non-asthmatics in homes with formaldehyde levels of 50 ppb or higher have elevated levels of expired nitric oxide, a marker for lower airway inflammation.

Table 2.3. Indoor Exposures and Development of Asthma

Sufficient Evidence of a Causal Relationship • House dust mite
Sufficient Evidence of an Association • ETS (preschoolers)
Limited or Suggestive Evidence of an Association • Cockroach (preschoolers) • Respiratory Syncytial Virus (RSV)
Inadequate or Insufficient Evidence to Determine Whether or Not an Association Exists • Cat, Cow, Horse, Dog, Domestic birds, Rodents • Cockroaches (except for preschoolers) • Fungi or mold, *Chlamydia pneumoniae, Chlamydia trachomatis, Mycoplasma pneumoniae*, Endotoxins • Houseplants, Pollen • NO_2, NO_X • Pesticides, Plasticizers, VOCs, Formaldehyde, Fragrances • ETS (school-age and older)

Source: IOM, 2000

The association between VOCs and asthma is complex. Other reviews support the association between VOCs and symptoms of asthma (Duhme *et al.*, 1998; Leikauf, 2002). Delfino *et al.* (2003b) studied Hispanic children with mild asthma in a Los Angeles community with high VOC levels near major freeways. Bothersome or more severe asthma symptoms were associated with breath concentrations of benzene, but not other breath VOCs. On breath sample days, asthma symptoms were also associated with 1-hour ambient NO_2 and sulfur dioxide (SO_2.).

Studies conducted in the workplace also demonstrate an association between asthma symptoms and chemicals used indoors. Between 1993 and 1997, 12% of confirmed cases (236 of 1,915) of work-related asthma in California, Massachusetts, Michigan, and New Jersey were related to exposure to cleaning products. Chlorine bleach was identified as the cleaning agent associated with the greatest number of cases. Exposures were greatest in medical settings, schools, and hotels (all non-industrial workplaces) with janitors, cleaners, and housekeepers experiencing the highest incidence of the disease (both new-onset and work-aggravated cases

of asthma: 80% were new onset; Rosenman *et al.*, 2003). From 1993 through mid-2003, 3,188 cases of work-related asthma were identified from doctor's first report of occupational injury or illness (DFR) in California (DHS, 2004). These cases are not specific to cleaning products.

2.1.2 Cancer

Many indoor air pollutants are known or suspected carcinogens. Formaldehyde, benzo(a)pyrene and other polycyclic aromatic hydrocarbons (PAHs), environmental tobacco smoke, benzene, chlorinated solvents such as tetrachloroethylene, and radon are a few of the identified carcinogens commonly found in indoor air, some at levels much higher than outdoor levels. Several technical documents provide summary data regarding the carcinogenic potential of these pollutants. Cancer unit risks and potency factors for 121 of the 201 carcinogenic substances for which emissions must be quantified in the California Air Toxics Hot Spots program are provided in the *Technical Support Document for Describing Available Cancer Potency Factors* (OEHHA, 2002; http://www.oehha.ca.gov/air.html). The U.S. EPA's Integrated Risk Information System (IRIS) provides a similar list of cancer potency values (http://www.epa.gov/iris/).

Risk assessments are conducted to estimate the increased risk of health problems in people who are exposed to different amounts of toxic substances. Risk is dependent on the amount of a pollutant people actually inhale, which depends on the air concentration of the pollutant in a given environment, the length of time a person is in that environment, and the person's breathing rate during that time. Since people spend the majority of their time indoors, moderate and high concentrations of indoor pollutants generally translate to elevated risk.

Several field studies have measured indoor concentrations of carcinogenic chemicals in California (Wallace *et al.*, 1988; Wallace *et al.*, 1991a; Sheldon *et al.*, 1992a; Avol *et al.*, 1996; and others). Results of these studies indicate that carcinogens are routinely found in most homes, often at levels higher than outdoor levels, due to the presence of indoor sources. Table 2.4 lists key pollutants identified in California studies, and indicates their cancer classification by the International Agency for Research on Cancer (IARC), a part of the World Health Organization. For the most recent classifications, visit the IARC website. Table 2.4 also lists the status of each pollutant as a California toxic air contaminant (TAC).

ARB staff estimate that about 230 excess cancer cases occur per year in California from indoor sources of toxic air contaminants, not including the excess cancer from exposure to radon, environmental tobacco smoke (ETS), and some other indoor carcinogens. This estimate of 230 cancer cases per year is based on risk estimates from the 1994 California Comparative Risk Project (CCRP, 1994), updated to reflect reduced exposure and risk from indoor formaldehyde. Formaldehyde levels are estimated to have decreased by about one-half since the 1980s studies on which the 1994 estimate was based (see Appendices II and III). The chemicals with the highest estimated risk in the CCRP were formaldehyde (found in many building materials and consumer products) and para-dichlorobenzene (used in mothballs and air fresheners). Other chemicals included were perchloroethylene (used in dry-cleaning), chloroform (a by-product of water chlorination and use of chlorine in spas and washing machines), trichloroethylene, benzene, 1,3-butadiene, styrene, benzo(a)pyrene, and di-2-ethylhexylphthalate (DEHP–a plasticizer). The latter organic chemicals are found in many different consumer products and building materials, and some also are produced by combustion processes (such as when cooking food or burning wood). (DEHP is listed as a California TAC. However, the International Agency for Research on Cancer (IARC) recently determined that there is inadequate evidence for the carcinogenicity of DEHP in humans, yet sufficient evidence

in experimental animals for carcinogenicity (http://www-cie.iarc.fr/htdocs/monographs/vol77/77-01.html).

The 1994 CCRP estimates, like other cancer risk estimates, were derived using 95% upper-bound cancer potency factors, combined with measured indoor exposure distributions. Cancer risk methodology based on upper-bound cancer potency estimates provides a common, protective basis for comparing risks across topic areas. However, the risk estimates should not be interpreted as predictions of actual disease (CCRP, 1994). The risk can be much lower, depending on the actual dose of the pollutant inhaled and absorbed, and other factors.

Table 2.4. Carcinogenic Status of Selected Indoor Air Pollutants

Compound	Cancer Status[1] Classification of International Agency for Research on Cancer (IARC)[2]	Status as a California Toxic Air Contaminant
Acetaldehyde	Group 2B, possible human carcinogen	Yes
Asbestos	Group 1, known human carcinogen	Yes
Benzene	Group 1, known human carcinogen	Yes
Benzo(a)pyrene (PAHs)	Group 2A, probable human carcinogen	Yes
Chloroform	Group 2B, possible human carcinogen	Yes
p-Dichlorobenzene	Group 2B, possible human carcinogen	Yes
Di-2-ethylhexylphthalate	Group 3, not classified	Yes
Environmental Tobacco Smoke	Group 1, known human carcinogen	Under consideration
Formaldehyde	Group 1, known human carcinogen	Yes
Methylene chloride	Group 2B, possible human carcinogen	Yes
Perchloroethylene	Group 2A, probable human carcinogen	Yes
Radon	Group 1, known human carcinogen	Yes
Styrene	Group 2B, possible human carcinogen	Yes
Trichloroethylene	Group 2A, probable human carcinogen	Yes

[1]Source: OEHHA, 2002; IARC website

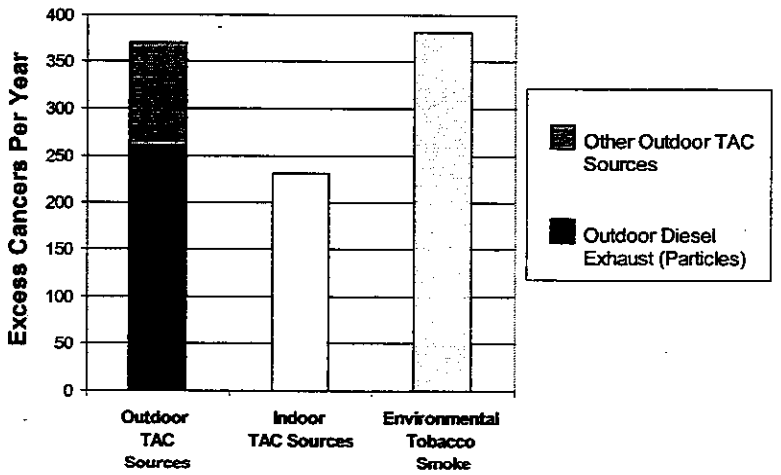
[2]Group 2A compounds have limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals. Group 2B compounds have limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals.

Since the time the studies used for the CCRP were conducted, some levels of indoor pollutants are estimated to have decreased while others likely have increased. However, there are insufficient new data available to refine the 1994 estimates for those chemicals. Because changes likely have occurred in both directions and are likely to be relatively small, and because the 1994 CCRP estimates did not include all known indoor carcinogenic pollutants (methylene chloride, other polycyclic aromatic hydrocarbons, and other phthalates were not included, for example), the 1994 estimates remain the best available estimates for the overall cancer risk posed by indoor chemical pollution in California, excluding that from radon gas, environmental

tobacco smoke, and asbestos (discussed later in this document). The details of ARB's assessment using the CCRP results are provided in Appendix II.

This estimate of 230 excess cancer cases approaches the cancer burden from diesel exhaust particles, which is estimated to result in 260 excess cases per year in California, and exceeds the cancer risk for other outdoor pollutants, estimated at about 110 cancer cases per year (Figure 2.1). Indoor air cancer risk is also nearly two-thirds of the total cancer risk estimated for outdoor pollutants. It is estimated that cancer risk due to diesel exhaust particles will decrease 75% by 2010 (ARB, 2000a), leaving indoor air as a predominant source of air pollution cancer risk.

Figure 2.1:
Estimated Potential Cancer Burden from Air Toxics in California by Source



Exposure to environmental tobacco smoke (ETS) makes a significant contribution to the cancer burden from air pollution as well. Although smoking prevalence and exposure of non-smokers has decreased in California, preliminary updated exposure and risk estimates for ETS developed by OEHHA are similar to those estimated in 1997 (OEHHA, 1997), due to the increase in the California population (and thus the number of individuals exposed). Updated estimates, which are currently undergoing peer review, show 380 excess lung cancer cases per year (OEHHA, 2004). This ETS risk level is similar to the total outdoor air pollutant cancer burden; however, because workplace exposure has decreased to nearly zero since the mid-1990s, and the prevalence of smoking has decreased substantially as well, the current cancer burden from ETS may be somewhat lower than shown in this graphic. Nonetheless, the contribution of ETS will remain significant for some time, because some individuals (including children) are still exposed to substantial levels of ETS.

Payne-Sturges *et al.* (2004) recently calculated cancer risk associated with indoor and personal exposure levels of VOCs and found risk levels similar to those estimated in this report. They measured the personal, indoor, and outdoor concentrations of 11 VOCs for 33 non-smoking adults in South Baltimore, Maryland. VOC concentrations were similar to earlier reported measurements in the California VOC Total Exposure Assessment Methodology (TEAM) studies,

with some variations noted both higher and lower. For personal monitoring, the highest median cancer risks were attributed to chloroform, benzene, and carbon tetrachloride. The authors assumed the cancer risks for the 11 VOCs were additive and calculated 165 lifetime cumulative cancer risks per 1 million based on average indoor concentrations of 11 measured VOCs, with formaldehyde not included. Calculations based on personal exposures and outdoor concentrations were 183 and 43 cumulative lifetime cancer risks per 1 million, respectively. A background risk (or ambient risk) was not subtracted from the indoor calculation. Applying this methodology to California, the estimate is comparable to the 230 excess cancer cases estimated in this report, based on a population of 35 million and the inclusion of formaldehyde, some other VOCs, and semi-volatiles such as benzo(a)pyrene, in the California estimate.

Other recently developed risk estimates also demonstrate the carcinogenic risk posed by indoor pollutants present at average concentrations. In the absence of indoor standards, Hoddinott and Lee (2000) applied U.S. EPA Superfund risk assessment methodology to selected VOCs to determine if indoor concentrations produce significant risks. Indoor VOC concentrations from two studies completed in the 1980s (Wallace, 1987; Cohen *et al.*, 1989) were used to determine the level of risk associated with VOCs measured inside residences. U.S. EPA considers acceptable levels of cancer risk to be one increase in lifetime cancer incidence per 10,000 to 1,000,000 persons (U.S. EPA, 1989). Hoddinott and Lee (2000) calculated that the risk for adults and children, based on average levels found in homes, exceeded the acceptable risk level of one in a million for a number of pollutants. Those pollutants are found in dry-cleaned clothing, ETS, cleaning agents, glued carpet, gasoline, and degreasers. The authors concluded that "Chemical concentrations resulting from 'off-gassing' from normal household activities and materials can result in a health risk estimate that exceeds the benchmark used at hazardous waste sites". The authors also note that "the data used in this evaluation predate changes in the manufacturing of indoor products. These efforts may have reduced some of the emissions in the average home."

2.1.3 Irritant Effects

Many indoor pollutants cause eye, nose, and throat irritation (Wolkoff and Nielsen, 2001). Tables of irritant thresholds have been developed to provide guidance for non-irritating levels of pollutants at workplaces and in the home (Molhave, 1991a; Devos *et al.*, 1990). The OEHHA Chronic Reference Exposure Level for formaldehyde is set at 2.4 ppb to protect against irritation of the eyes and upper and lower respiratory tract. Ozone, the primary ingredient in smog and a strong oxidant, also irritates the respiratory system, causing coughing, throat irritation, or a burning sensation in the airways. Ozone irritation can lead to a feeling of chest tightness, wheezing, and shortness of breath (ARB, 2000b). Other chemicals such as isoprene and terpenes, have been shown to react with oxidants, producing irritating products such as formaldehyde, terpene oxides, and fine particles (Long *et al.*, 2000; Wilkins *et al.*, 2001). Some biological contaminants such as some types of mold are also known to cause irritant effects.

2.1.3.1 Reaction Products

Indoor organic chemicals react with oxidants such as ozone, hydroxyl radicals, and nitrate radicals to produce secondary pollutants. Weschler (2004) provides an excellent review of studies investigating indoor chemical reactions published since 2000. The review focuses on the importance of hydroxyl radicals in indoor reactions, reactions occurring on indoor surfaces, and the impact secondary reaction products have on building occupants. Traditional analytical methods often do not detect some of the short-lived, highly reactive compounds that are produced. Sensory measurements have been used to detect changes in indoor air quality

associated with the reactions. It is thought that the ozone/terpene reactions dominate indoor chemistry based on the frequent presence of ozone and ubiquitous presence of terpenes in indoor environments (Weschler, 2004).

Terpenes are reactive chemicals (e.g., α-pinene, *d*-limonene, myrcene) that are frequently used in cleaning products and other products for their favorable odor characteristics and solvent properties. In order to use less toxic ingredients in consumer products, manufacturers have replaced petroleum-based hydrocarbons with plant-derived compounds, such as *d*-limonene. These compounds are Generally Recognized as Safe (GRAS) by the Food and Drug Administration (FDA). They can be airway irritants at concentrations greater than normally encountered in indoor air (Wolkoff *et al.*, 2000). However, terpenes also have been associated with irritation at lower levels: the irritant chemicals are hypothesized to be a product of the reaction of terpenes with oxidants, rather than the terpene itself (Wilkins *et al.*, 2001; Weschler and Shields, 1997; Weschler and Shields, 1996; Atkinson and Arey, 2003).

Investigators are exploring potential reactions between unsaturated hydrocarbons and oxidants in an effort to more positively identify the cause of sick building health effects. Several reactions, such as between ozone and unsaturated hydrocarbons, ozone and nitrogen oxides, and free radical reactions, may lead to the formation of more irritating indoor compounds. These reactions can lead to the production of submicron particulate matter, aldehydes (formaldehyde) and ketones with lower odor thresholds and greater irritancy than precursors, carboxylic acids such as formic acid and acetic acid, and free radicals (Weschler and Shields, 1997; Sarwar *et al.*, 2004). Pollutants with reactive double bonds such as terpenes and alkenes react with ozone and nitrogen oxides to produce products that result in airway irritation similar to that of formaldehyde. Fan *et al.* (2003) confirmed the reaction of ozone with d-limonene and ozone with α-pinene under indoor conditions to generate submicron particles and other potentially irritating species, such as aldehydes and organic acids. To minimize these reactions, Fan *et al.* (2003) suggest "limiting use of products that emit high-reactivity alkenes during episodes when outdoor ozone levels are elevated", reducing outdoor ozone levels, and minimizing the penetration of ozone from outdoors.

In this growing area of research, investigators have identified an increase in fine particles associated with mopping floors with a pine-scented cleaning product. It is hypothesized that the generation of particles was the result of ambient ozone (up to 48 ppb) reacting with the terpenes in the cleaning product (Long *et al.*, 2000). Sarwar *et al.* (2004) clearly demonstrated the indoor reaction between ozone and terpenes from various consumer products, leading to increases in fine particle mass concentrations. This area of research warrants increased effort in order to understand the association between indoor air pollutants and related health effects.

2.1.3.2 Sick Building Syndrome

Sick Building Syndrome (SBS) is a term used to describe a collection of irritant and neurological effects that occur while occupants are in a building, that generally disappear when affected people are out of the building. Specific causes of SBS have not yet been firmly identified. The most common symptoms include eye irritation, congested nose, headache, fatigue, difficulty concentrating, and dry skin (Tenbrinke *et al.*, 1998). SBS differs from building-related illness (BRI; see Biological Contaminants section) in which an identifiable factor causes a specific illness such as bacteria causing Legionnaires' disease or humidifier fever.

In an attempt to identify factors related to SBS, Seppanen and Fisk (2002) reviewed the literature to summarize factors associated with SBS. They found that relative to natural

ventilation, air conditioning was consistently associated with a statistically significant increase in the prevalence of one or more SBS symptoms, by approximately 30-200%. This finding reinforces the use of ventilation as a mitigation measure for reducing indoor pollution, but still may not identify the primary cause. The review identifies several confounding factors that are not affected by the HVAC type: quantity of carpet or textile surfaces; sealed windows; building age; depth of the building bays; and dusty surfaces. A European review also found an association between ventilation and comfort and health (Wargocki *et al.,* 2002).

Investigators who study indoor reactive chemistry suggest that the degradation products of VOCs may be responsible for the reported SBS symptoms (Carslaw, 2003; Wolkoff *et al.*, 2000; Wolkoff and Nielsen, 2001; Weschler, 2004). These reactions include ozone/terpene reactions with propagation of hydroxyl radicals and reactions on indoor surfaces such as ozone interacting with carpet. The impact that the products of indoor chemistry can have on building occupants has also been studied on a physiological level (Weschler, 2004).

Mendell (1993) conducted a review of the epidemiological literature related to SBS. In reviewing 32 studies, he found consistent findings for an association of SBS symptoms with air-conditioning, carpets, more workers in a space, video display use, and ventilation rates at or below 10 liters/second/person. With specific causes unidentified, Mendell stressed the importance of using prudent design, operation, and maintenance practices to prevent sick building symptoms.

Tenbrinke *et al.* (1998) reported a new approach for using VOC exposure metrics as predictors of SBS. These authors were able to confirm a link between exposure to low level VOCs and SBS symptoms. Apte and Daisey (1999) used the methodology developed by Tenbrinke to identify an association between mucous membrane symptoms and photocopiers. Apte and Daisey also identified a relationship between sore throat symptoms and fresh paint.

Many biological agents can provoke an immunological response, which most frequently takes the form of allergic reactions to the agent. Common symptoms and signs are watery eyes, runny nose, sneezing, nasal congestion, itching, coughing, wheezing, difficulty breathing, headache, and fatigue. Allergic rhinitis (hay fever) and allergic asthma are examples of hypersensitive responses to biological contaminants. Fungal spores, microbial byproducts, dust mites, cat allergen, and pollens are frequently associated with allergic responses.

2.2 Traditional (Criteria) Pollutants

Several pollutants for which outdoor air quality standards have been established occur at elevated levels indoors as well, and can pose a serious health risk. Ambient PM has been associated with premature death and serious respiratory and cardiovascular effects in numerous studies. CO can cause near-term death with high exposures of relatively short duration. All of these pollutants can impose serious, non-fatal health impacts: NO_2 from indoor combustion appliances can harm the lungs and other mucous membranes and cause respiratory disease, and ozone can have similar effects at elevated levels. The health effects, indoor sources, and indoor air concentrations of these traditional (criteria) pollutants are discussed below.

2.2.1 Particulate Matter

Particulate matter (PM) is broadly defined as any non-gaseous material suspended in the air. PM can include solid material (i.e., dust), liquid material (i.e., a sprayed aerosol), or a

combination of solid/liquid materials (i.e., a hydrated vehicle exhaust particle). PM is generally classified by its size. PM2.5 refers to all suspended matter having aerodynamic diameters less than 2.5 microns (µm: one µm = one millionth of a meter) and is commonly referred to as 'fine' PM. PM10 refers to all suspended matter having aerodynamic diameters less than 10 µm. PM between 2.5 µm and 10 µm is commonly referred to as 'coarse' PM. Both federal and state ambient (outdoor) air quality standards incorporate these size distinctions. Recent studies suggest that PM2.5 mass may be a better indicator than PM10 mass for predicting potential health effects resulting from ambient (outdoor) PM exposure (Williams *et al.*, 2000abc; Schwartz and Neas, 2000), although some recent studies have specifically linked health impacts to the coarse fraction (Lippmann *et al.*, 2000; Mar *et al.*, 2000; Ostro *et al.*, 2000).

A substantial portion of indoor particles originate outdoors from outdoor sources (Ozkaynak *et al.*, 1996ab; Abt *et al.*, 2000, Long *et al.*, 2000; Liu and Nazaroff, 2001). Outdoor sources of PM that may infiltrate indoors include PM from transportation sources (i.e., gasoline and diesel powered highway vehicles), agricultural activities (i.e., biomass combustion emissions, fugitive dust emissions, pesticide sprays), biogenic emissions (i.e., forest fire smoke), and many others. Further reduction of these concentrations in outdoor air would likewise lower their concentrations in indoor environments. However, there are numerous indoor sources of PM as well. These include combustion devices and activities such as stoves, fireplaces, cigarette smoking, cooking, and candle burning, all of which can produce indoor PM with harmful components similar to those from outdoor air (Lofroth *et al.*, 1991). Indoor particles also include fibrous materials, pollen, mold spores and fragments, and tracked-in soil particles (Wallace, 1996a). These particles become trapped in/on building surfaces, particularly carpets, and have been shown to persist for a very long time, due to the lack of applying effective cleaning and maintenance procedures, and they may be re-suspended into the air. Some can trigger asthma attacks and allergy symptoms, as discussed previously. Others can have a mix of toxic components such as PAHs, lead, and pesticides adsorbed onto them; these components may contribute to serious health effects such as cancer (PAHs) and developmental effects (lead).

Major epidemiologic studies have shown a strong association between ambient (outdoor) PM concentrations and increased death and disease (e.g., Dockery *et al.*, 1993; Pope *et al.*, 1995) and an increase in the rate of death from cardiovascular and respiratory disease (Samet *et al.*, 2000). Indoor PM, particularly from indoor combustion sources, may be similar in composition to outdoor PM, and might be expected to cause the same impacts as outdoor PM. However, research has only recently been undertaken to examine the differences in indoor and outdoor PM composition, and the relative contribution of indoor PM and outdoor PM to the total PM effects of death and disease have not been studied, and are high priorities for further research. Although considerable progress has been made in elucidating the toxicological mechanisms of outdoor PM toxicity, it is difficult to draw inferences between indoor and outdoor PM without focused studies: indoor PM could be more or less toxic than outdoor PM. However, because ambient PM epidemiological studies are based on particle size and include a mix of particles from combustion sources, soil, and other sources, the epidemiological relationships from ambient PM studies and their magnitude should be examined when considering the potential risk from indoor PM. The effects of ambient PM are summarized below, followed by a brief discussion of the potential impacts of indoor PM.

2.2.1.1 Death

Both acute and chronic ambient PM exposure have been associated with an increased risk of premature death, primarily in older adults with preexisting heart and/or lung disease. Studies conducted in California, the U.S., and in diverse cities worldwide suggest that risk of death

increases about 0.25 to 3.5 percent with each 10 μg/m^3 increase in daily mean ambient PM2.5 concentration (e.g., Burnett and Goldberg, 2003; Dominici *et al.*, 2003; Fairley, 2003; Goldberg and Burnett, 2003; Moolgavkar, 2003; Schwartz, 2003; Ponka *et al.*, 1998). Long-term cohort studies suggest that the increase in risk of death is about 4% with each 10 μg/m^3 increase in annual mean ambient PM2.5 concentration (Dockery *et al.*, 1993; Krewski *et al.*, 2000; Pope *et al.*, 1995). Meta-analyses of earlier studies suggest that the effects on death are fairly consistent (Ostro, 1993; Dockery and Pope, 1994; Schwartz, 1994), regardless of where the study was performed. About 6500 deaths occur each year in California due to outdoor particulate pollution levels above the State ambient air quality standards (ARB/OEHHA, 2002).

In a recent assessment of global and regional health risks, Cohen *et al.* (2004) used PM2.5 (measured or estimated) as an index for urban air pollution. They reviewed literature from all parts of the globe, and estimated the portion of death from specified diseases attributable to urban pollution (PM2.5). They estimated that pollution in urban areas worldwide causes about 3% of death attributable to cardiopulmonary disease in adults; about 5% of death attributable to cancers of the trachea, bronchus, and lung; and about 1% of death attributable to acute respiratory infections in children. Based on a year 2000 population, this totals about 800,000 excess deaths and 6.4 million disability-adjusted life years. The greatest burden was estimated to occur in the more polluted and rapidly growing cities of developing countries. The authors noted the universality of PM effects found worldwide, despite some differences in ambient PM sources and composition. This lends support to the likelihood of similarities of impacts from ambient and indoor PM. The authors noted that the estimates derived in their analysis are likely an underestimate, and that they cannot be extrapolated to smaller regions or for other purposes.

2.2.1.2 Non-lethal Health Impacts

Several hundred studies have been published examining the association between various measures of ambient PM and a variety of adverse health effects other than premature death. The health outcomes associated with ambient PM concentrations include hospitalization and emergency room visits for respiratory or cardiovascular disease; respiratory symptoms, including asthma symptoms; restrictions in activity and school absenteeism; and reduced lung function and other effects in children. Although these effects are not as serious as immediate death, they are serious (some can lead to death) and affect a greater proportion of the population, and thus have a major impact on public health. The key results from some of the published studies include the following:

- **Hospitalization and serious respiratory disease**

  - ✓ Studies consistently report associations between both ambient PM2.5 and PM10 and hospital admissions for respiratory and cardiovascular illness (e.g., Atkinson *et al.*, 2003; Sheppard *et al.*, 1999; Sheppard, 2003; Ito, 2003; Zanobetti and Schwartz, 2003; Moolgavkar, 2003; Le Terte *et al.*, 2003). These effects have been reported mainly for people over age 65 who already have some form of cardiopulmonary disease. Respiratory causes of admission include pneumonia, chronic obstructive pulmonary disease (COPD) and asthma, while cardiovascular causes have included general cardiovascular disease, congestive heart failure, stroke, and ischemic heart disease. Overall, ambient PM10 has been associated with an estimated increase in risk of hospitalization of 1.25% - 5% per 10 μg/m^3 increase in the daily mean ambient PM10 concentration for respiratory endpoints, and 0.3% to 2.6% for cardiovascular endpoints.

- ✓ Associations have also been reported between ambient PM10 and PM2.5 and emergency department visits, primarily for asthma exacerbation, which may or may not result in hospital admissions (e.g., Lipsett *et al.*, 1997; Delfino *et al.*, 1997).

- **Respiratory symptoms**

  - ✓ Studies have associated ambient PM10 and PM2.5 exposure with asthma and respiratory symptoms, for example cough, phlegm, chest pain, or wheeze (e.g., Delfino *et al.*, 2003; Mortimer *et al.*, 2002; Schwartz and Neas, 2000), asthma exacerbation (e.g., Whittemore and Korn, 1980), and use of asthma medications (e.g., Delfino *et al.*, 1996; Pope *et al.*, 1991). People with asthma retain a greater number of ultrafine PM particles than do healthy subjects, thus making them more susceptible to the health effects of air pollution Chalupa *et al.* (2004).

  - ✓ Cellular level effects have also been identified. Exposure to respiratory irritants can result in local airway inflammation, altered epithelial cell permeability, increased mucus secretion, and bronchoconstriction. Disease states such as asthma and chronic bronchitis can adversely affect particle clearance or removal (e.g., Foster, 1999). Also, the viability and functional integrity of cells in the lungs can be adversely affected by ambient PM exposures (e.g., Soukup and Becker, 2001).

- **Work loss, absenteeism, reduced productivity:**

  - ✓ Ostro (1987) and Ostro and Rothschild (1989) reported 10 to 15% reduction in activity due to respiratory-related causes per 10 $\mu g/m^3$ of ambient PM10.

  - ✓ Ransom and Pope (1992) reported about a 4% increase in absenteeism per 10 $\mu g/m^3$ of ambient PM10 at an elementary school in Utah.

  - ✓ Gilliland et al. (2001) reported an increase of 5.7% in total illness-related absences among 4th grade school children (ages 9-10) in 12 southern California communities.

- **Effects on children**

  - ✓ Investigators with the ARB-sponsored Children's Health Study found that, among children with asthma, respiratory symptoms increased with increasing ambient particle levels (McConnell *et al.*, 1999). Results also suggest that children who live in communities with high concentrations of ambient PM may have decreased lung function growth compared to children living in communities with lower concentrations of ambient PM (Gauderman *et al.*, 2000; Peters *et al.*, 1999ab). However, both of these results were also true for NO_2 and acid vapor, and the independent effects of the different pollutants cannot be assessed because of high inter-pollutant correlations. Similar results have also been reported by Horak *et al.* (2002) in Austrian children. A recent study by Delfino *et al.* (2004) found clinically relevant decreases in lung function associated with personal PM exposure in schoolchildren with asthma.

  - ✓ Several recent studies have suggested that the unborn may also be at risk of adverse effects from ambient PM pollution, based on statistically significant relationships between outdoor PM concentration and low birth weight (Ritz *et al.*, 2000; Bobak, 2000), premature birth (Bobak, 2000), neonatal death (Penna and

Duchiade, 1991; Woodruff *et al.*, 1997; Bobak and Leon, 1998), and fetal growth retardation (Dejmek *et al.*, 1999). However, except for Ritz *et al.*,. (2000) and Ritz *et al.*, (2002), these studies have been conducted outside the U.S., in areas with higher ambient PM concentrations than those typically observed in the U.S.

The following incidences of illnesses are estimated to occur annually in California due to outdoor PM10 levels above the State ambient air quality standard.

- 7,900 cases of chronic bronchitis among people age 27 or older,
- 6,000 hospital admissions for cardiopulmonary causes among the elderly (ages 65 or more),
- 1,000 asthma-related hospital admissions among people age 64 or less,
- 2,300 asthma-related emergency room visits among people age 64 or less,
- 340,000 asthma attacks among all ages.

The California ambient air quality standard for PM2.5 is 12 $\mu g/m^3$ (annual mean), and the PM10 standards are 50 $\mu g/m^3$ (24-hour) and 20 $\mu g/m^3$ (annual mean). There are no federal or California standards for indoor PM levels. The national ambient air quality standards (NAAQS) for PM2.5 are 65 $\mu g/m^3$ (24-hour average) and 15 $\mu g/m^3$ (annual mean), and for PM10 are 150 $\mu g/m^3$ (24-hour on average) and 50 $\mu g/m^3$ (annual mean). These levels are often exceeded in California's indoor environments, particularly when indoor sources are used or when particle-generating activities occur.

2.2.1.3 Potential Health Impacts of Indoor PM

The serious adverse health impacts of certain indoor PM components are well documented. As discussed in other sections of this document, tobacco smoke particles, radon daughters, metals such as lead, semi-volatiles such as PAHs, and biological components such as pollens and mold all exert serious, sometimes fatal, impacts on human health. However, based on the serious impacts documented for outdoor PM, it is likely that indoor PM is much more than the sum of its parts–there are likely serious effects not yet measured, quantified, or properly accounted for.

As indicated in the preceding section, large numbers of cases of illnesses are expected to occur each year in California due to outdoor particulate matter pollution levels above the State ambient air quality standards (ARB/OEHHA 2002). Indoor PM is comprised of varying proportions of PM of indoor and outdoor origin (discussed below). Because the additional PM burden from indoor sources is generally not well represented in epidemiology studies, indoor PM emissions may be significant contributors to the adverse impacts seen in the epidemiology studies. Additionally, indoor PM may also contribute to premature mortality, hospital admissions, chronic bronchitis, and other effects beyond the levels quantified in the epidemiology studies.

Two groups have recently examined the literature to determine whether there is sufficient information available to permit a rough estimate of the impacts from indoor PM. The first group was a panel of indoor air quality and PM experts convened by ARB in February 2004 to review and assess what is known regarding the impacts of indoor PM on health. The panel found that only one study provides suggestive evidence of the health effects of indoor combustion emissions. In that study of rat alveolar macrophages, investigators found that indoor-generated particles triggered greater production of tumor necrosis factor than did a comparable amount of outdoor PM, suggesting that indoor-generated PM may be more bioactive than ambient particles (Long *et al.*, 2001). This may be due to PM emissions from indoor combustion sources being relatively "fresh", and small in size. Alternatively, outdoor PM may actually be much more

toxic than indoor PM, due to emissions from complex sources such as diesel trucks and industrial plants. There is considerable variability in the chemical composition, acidity, and size distribution of outdoor PM depending on geological conditions, traffic mix, meteorological conditions, proximity to major roadways, and significant stationary sources. However, the Cohen *et al.* (2004) study found that PM impacts were essentially global, with few differences across regions, lending support to the likelihood of similar impacts from indoor PM. The panel determined that available studies document the known effects of ETS particles and infectious and allergenic indoor biological contaminants, but that few studies have been designed to specifically identify effects of other types of indoor-generated PM, such as that from candles or woodsmoke. They concluded that research is sorely needed in this area, to determine the actual toxicity of indoor-generated PM, particularly from indoor combustion sources, and the relative toxicities of indoor and ambient PM.

The second group that examined this issue was a European interdisciplinary group of researchers who reviewed the relevant literature to determine whether particle mass, surface area, or number concentration could be used as risk indicators for health effects in non-industrial buildings (Schneider *et al.*, 2003). The group concluded that the study design of most of the reviewed studies was not focused on finding associations between airborne PM and health outcomes, and consequentially, while airborne particles are likely to cause health effects in non-industrial environments, the scientific evidence was inadequate to permit the use of indoor PM mass, surface area, or number concentration as risk indicators for health effects in buildings.

2.2.2 Indoor PM Sources and Emissions

Indoor PM concentrations are typically equal to or higher than concurrently measured outdoor levels (see next section), depending on the sources and activities that are present indoors. Outdoor air infiltration and indoor combustion sources such as smoking and cooking are typically the greatest sources of indoor PM (Wallace, 1996a; Ozkaynak *et al.*, 1996ab; Brauer *et al.*, 2000; Abt *et al.*, 2000; Fortmann *et al.*, 2001). Prominent indoor sources include cigarettes, woodstoves, and candles; cooking and cleaning activities (Ozkaynak *et al.*, 1996ab; Abt *et al.*, 2000, 2001; Long *et al.*, 2000); the presence and activities of occupants (Abt *et al.*, 2000; Rodes *et al.*, 2001); the use of personal care products (Conner *et al.*, 2001); and indoor chemical reactions (Weschler and Shields, 1997; Nazaroff and Weschler, 2004). Fibrous materials, pollen, mold spores and fragments, and tracked-in and blown-in soil particles are also components of indoor PM (Wallace, 1996a).

The contribution of outdoor PM to indoor PM concentrations can be substantial but highly variable. For residential buildings, the main entry routes of outdoor air are open windows and doors, cracks in the building shell, and mechanical ventilation systems such as swamp coolers and whole house fans. Investigators of a large, population-based study in California, the Particle Total Exposure Assessment Methodology Study (PTEAM Study) estimated that residential indoor PM10, on average, is roughly comprised of about 66% outdoor PM10; 75% for PM2.5 (Ozkaynak *et al.*, 1996a,b). In a study of four Boston homes with air exchange rates below 1.0 air exchange per hour (ACH), Abt *et al.*, (2000) estimated that only 20-43 percent of indoor PM2 to PM10 were from outdoors, while 63-92 percent of indoor PM 0.02-0.3 µm were from the outdoors. Abt *et al.*, (2000) and Long *et al.*, (2000) also found that the relative contribution of outdoor PM to indoor levels varied by particle size, with outdoor air generally contributing a majority of the smaller particles measured indoors, while indoor sources contributed more to the coarse (2-10 micrometers) fraction. Because these studies examined primarily older individuals who are less active in their homes than younger families may be, and were conducted on the

east coast, the results of these studies may not reflect typical California proportions; however, they show that reductions in outdoor PM levels could have a major effect on indoor concentrations

Indoor combustion source emissions, such as those from smoking and cooking, are often intermittent and highly variable, but emissions can be very high, resulting in exposures that can have significant impacts on people's total daily exposure to PM (Long *et al.*, 2000). In the PTEAM Study homes with smokers, it was estimated that 30% of the PM2.5 mass and 24% of the indoor PM10 mass came from smoking. For homes in which cooking occurred during the monitoring period, 25% of the indoor PM2.5 and PM10 was estimated to come from the cooking activity (Ozkaynak, 1996b). These results are consistent with those of previous indoor studies that examined the impact of cigarette smoking on indoor PM levels, and they are consistent with subsequent studies of indoor cooking emissions that confirmed the high impact of cooking on indoor and personal PM levels (Abt *et al.*, 2000; Wallace, 2000b; Brauer *et al.,* 2000; Fortmann *et al.,* 2001).

For example, in an ARB-sponsored study, Fortmann *et al.* (2001) measured indoor and outdoor PM during 32 types of cooking activities with both gas and electric ovens and stovetops. Although concurrent outdoor levels reached only 20 $\mu g/m^3$, indoor PM levels during and after cooking often exceeded 50 $\mu g/m^3$, ARB's indoor air quality guideline level and ambient air quality standard for ambient PM10 for 24 hours. Kitchen PM10 levels exceeded more than 1400 $\mu g/m^3$ during frying, broiling, and baking activities with the gas stove (Fortmann *et al.,* 2001). Indoor PM levels during cooking with the electric stove were generally lower; however, cooking with the electric stove produced much higher indoor PM levels in two cases - frying tortillas and stovetop stir-frying. The highest concentrations of indoor PM were produced when using the self-cleaning cycle of the oven for several hours. Indoor PM10 was over 3,600 $\mu g/m^3$ (over 2,000 $\mu g/m^3$ PM2.5) for the gas stove, and nearly 400 $\mu g/m^3$ PM10 for the electric stove. Measurement of particle counts during cooking activities and oven cleaning indicated that particles were primarily smaller than 0.1 µm.

The burning of wood, candles, and incense can also be important combustion sources of residential indoor PM, especially in the 2.5 µm size range and below (Wasson *et al.*, 2002; Jetter *et al.*, 2002; Brauer *et al.*, 2000; Guo *et al.*, 2000; Lofroth *et al.*, 1991). Individual candles, for example, released 200-3600 µg/hr of PM10, and 100-1700 µg/hr of lead from the lead wick (Wasson *et al.*, 2002). Another investigator found a mean lead emission rate from candles of 770 µg/hr of lead (Alphen, 1999). Guo *et al.* (2000) modeled indoor PM2.5 concentrations ranging from 4.3-1173 $\mu g/m^3$ based on PM emission measurements from candle burning. These indoor combustion sources produce PM with potentially harmful components similar to those from some outdoor PM combustion sources (Lofroth *et al.*, 1991).

Physical generation or re-suspension of particles also can contribute to airborne indoor PM levels. Soft or porous interior surfaces such as carpets and draperies have the potential to attract and re-emit particles (Thatcher and Layton, 1995; Kamens *et al.*, 1991). Particle concentrations can be high even in homes where good cleaning practices are used. The particles can become re-entrained in the indoor air when people walk or play (Wallace, 2000a; Roberts and Dickey, 1995; Abt *et al.*, 2000; Vette *et al.*, 2001).

Particles in house dust, such as metals, and semi-volatile chemicals such as pesticides and some PAHs that have their own toxic properties pose a risk to children (Rothenberg *et al.*, 1989; Roberts and Dickey, 1995; Lewis *et al.*, 1999; U.S. EPA, 1999a). Particles of house dust with these contaminants can be re-emitted to the air and subsequently inhaled, and may be ingested

by children through hand-to-mouth behavior, often the primary route of exposure, or dermally absorbed when they spend time on the floor (Lewis *et al.*, 1994; Zartarian *et al.*, 1998; Zartarian and Leckie, 1998). For toxics such as lead, floor dust levels can be a major determinant of exposure.

Biological contaminants such as fungi, bacteria, house dust mites and pollen also contribute to indoor particle concentrations, especially in buildings with moisture problems from flooding or roof leaks that have not been properly repaired. Bioallergens, such as pollen, in outdoor air can also penetrate indoor spaces. Re-entrained road dust may be a particularly important source of bioallergens in both indoor and outdoor air (Miguel *et al.*, 1998).

2.2.3 Indoor and Personal PM Concentrations

Indoor PM concentrations sometimes exceed outdoor air concentrations, due to the presence of indoor sources of PM. For example, in one of the first comprehensive residential PM field studies, Spengler *et al.* (1981) found an increase in indoor concentrations of respirable PM of approximately 1 $\mu g/m^3$ per cigarette smoked per day, and about a 20 $\mu g/m^3$ increase per pack. Other examples are discussed in the Indoor PM Sources and Emissions Section above.

Additionally, people's personal exposures to PM sometimes exceed both indoor and outdoor concentrations, primarily because people tend to spend time very near pollutant sources, such as when cooking or cleaning. This has been called the "proximity effect", and reflects the fact that pollutant levels are highest near the source than farther away where emissions have become diluted in the air (McBride *et al.*, 1994). A small portion of the elevated personal exposure levels seen across PM studies also is attributed to the existence of a "personal cloud" of PM surrounding a person, due to re-suspension from clothing, the use of personal care products (Conner *et al.*, 2001), and skin flakes. Williams *et al.* (2000b) estimated a mean personal cloud of 3.1 $\mu g/m^3$, and Rodes *et al.* (2001) estimated personal clouds of 3 $\mu g/m^3$ in two separate studies. However, Wallace (2000a) examined a breadth of studies and estimated an average personal PM10 cloud of 30 $\mu g/m^3$, ranging from 3-67 $\mu g/m^3$. Personal PM2.5 clouds were estimated to range from 6-27 $\mu g/m^3$.

Consequently, because proximity to PM-emitting sources and activities can significantly increase people's actual exposures, most studies of indoor residential PM concentrations have included, or even focused on, measurement of occupants' personal exposures to PM. These have been obtained using personal samplers worn by study participants for one or more days as they go about their daily routines. These measurements provide a more accurate measure of people's exposure to PM, because PM levels are often higher very near people than at the location of indoor air sampling equipment in a room. Table 2.5 summarizes the major indoor and personal exposure PM studies conducted in the U.S. in recent years. Unlike earlier studies, there was little or no cigarette smoking in most of the studies listed in Table 2.5.

The first major study to measure indoor, outdoor, and personal PM concentrations in California was the PTEAM Study. Investigators measured PM10 and PM2.5 for 12-hour daytime and nighttime periods in 178 homes during the fall in Riverside, California. They found 12-hour daytime personal PM10 concentrations to be about 50% higher than simultaneously measured daytime residential indoor or outdoor concentrations. Daytime personal concentrations averaged 150 $\mu g/m^3$, while indoor and outdoor concentrations both averaged about 95 $\mu g/m^3$ (Clayton *et al.*, 1993, Ozkaynak *et al.*, 1996ab). Most importantly, 12-hour daytime personal PM10 concentrations exceeded the California 24-hour ambient air quality standard level of 50 $\mu g/m^3$ for about 90% of the monitoring days, and exceeded the federal PM10 standard level of

150 μg/m^3 for 25% of the monitoring days. During nighttime, personal PM10 concentrations decreased and were similar to concurrent indoor and outdoor concentrations (roughly 80 μg/m^3), reflecting the influence of the proximity of people to PM sources during normal daytime activities in determining personal exposure concentrations.

Studies conducted since the PTEAM study in other locations and seasons have measured average personal PM10 concentrations from 11-68 μg/m^3, and average personal PM2.5 concentrations ranging from 9-34 μg/m^3. Average indoor concentrations of PM10 have ranged from 13-52 μg/m^3, with indoor PM2.5 concentrations from 7-34 μg/m^3. However, peak indoor levels have been high. For example, in a seven city study, Wallace *et al.* (2003) found that in all cities, at least 2% of all 1-hour measurements exceeded 1000 μg/m^3. Long *et al.* (2000) measured indoor PM concentrations as high as 473 μg/m^3 in study homes. These elevated levels signify the presence of significant indoor source emissions for short periods of time relative to the average measurements.

Several studies have been conducted to examine the exposure of sensitive populations to PM. These have reported PM10 and PM2.5 exposures for subsets of individuals with chronic obstructive pulmonary disease (COPD), coronary heart disease, and asthma (Liu *et al.*, 2003; Williams *et al.*, 2000ac; Rojas-Bracho *et al.*, 2000; Linn *et al.*, 1999). Liu *et al.* (2003) found that mean personal PM2.5 concentrations were higher than indoor and outdoor concentrations for each of these three sensitive groups (as well as for healthy subjects) in Seattle, Washington, and that PM10 indoor concentrations were higher than outdoor concentrations for asthmatics. Williams *et al.* (2000ac) also found personal PM2.5 concentrations higher than indoor and outdoor concentrations in a Baltimore retirement facility in the winter of 1997. Rojas-Bracho *et al.* (2000) found that mean personal PM10 and PM2.5 concentrations were above indoor and outdoor PM10 and PM2.5 concentrations for 18 COPD patients in Boston.

Linn *et al.* (1999) monitored 15 COPD patients for PM2.5 exposures and 15 patients for PM10 exposures in Los Angeles during the fall and winter. Unlike other studies, this study found personal concentrations similar to both those indoors or outdoors. The authors suggest that the lack of increased personal PM in these subjects having severe COPD may be due to reduced personal activity, less time spent outside of the home, and other reasons. The pooled correlation of personal PM concentrations to ambient concentrations at a monitoring station were quite low.

A recently completed study by Suh (2003) has increased our understanding of Californians' exposures to PM in a sensitive subpopulation. The investigators examined the relationships among outdoor and indoor concentrations and personal exposures across different seasons. They examined daily PM exposures of a group of 15 individuals with COPD in Los Angeles over seven sequential days in the summer-fall and/or winter. Personal, indoor, and outdoor PM2.5, NO_3^- and elemental carbon (EC) concentrations varied by season, with the exception of outdoor NO_3^-. For winter and summer-fall PM levels, respectively, the personal PM2.5 means (19.6, 25.1 μg/m^3) and maxima (63.5, 137.8 μg/m^3) were higher than the indoor means (16.9, 18.1 μg/m^3) and maxima (49.5, 94.8 μg/m^3) and outdoor means (13.5, 19.3) and maxima (56.5, 53.5 μg/m^3). For NO_3^- and EC, higher outdoor (2.8-3.1 μg/m^3), as compared to indoor (1.1-1.7 μg/m^3) and personal (1.2-1.6 μg/m^3) levels, were found in both seasons, reflecting the fact that motor

Table 2.5. Summary of Recent Indoor PM Exposure Studies (means)

				PM 2.5 means - µg/m³						
LOCATION	GROUP	YEAR	SEASON	PERSONAL	(n)	INDOOR	(n)	OUTDOOR	(n)	REFERENCE
Los Angeles, CA	COPD Subjects	2001-2002	multiple	17.7 ± 11.9	91	17.6 ± 11.4	106	28.8 ± 20.4	103	Suh 2004
Raleigh, NC	African Americans	2000-2001	multiple	23.0 ± 16.1	712	19.3 ± 8.4	761	19.3 ± 8.4	761	Williams 2003b
Seattle, WA	COPD Subjects	2000-2001	multiple	10.5 ± 7.2	307	8.5 ± 5.1	443	9.2 ± 5.1	437	Liu 2003
	Healthy Subjects			9.3 ± 8.4	183	7.4 ± 4.8	193	9.0 ± 4.6	194	
	Asthmatics			13.3 ± 8.2	263	9.2 ± 6.0	276	11.3 ± 6.4	272	
	Coronary Heart Dis			10.8 ± 8.4	325	9.5 ± 6.8	329	12.6 ± 7.9	323	
Boston, MA	Asthmatic Children	2000	multiple			12.3[1]	NR	9.0[1]	NR	Brugge 2003
						33.8[2]	NR	13.1[2]	NR	
Los Angeles, CA	COPD Subjects	2000	winter	19.6 ± 14.5	87	16.9 ± 11.7	92	13.5 ± 8.5	92	Suh 2003
		1999	summer-fall	25.1 ± 20.8	92	18.1 ± 11.1	97	19.3 ± 9.0	96	
Detroit, MI	Asthmatic Children	1999-2000	multiple			34.4 ± 21.7	362	15.6 ± 8.2	NR	Keeler 2002
Fresno, CA	Retirement Facility	1999	winter	13.3	24	9.7	24	20.5	28	Evans 2000
			spring	11.1	12	8.0	24	10.1	28	
Baltimore, MD	Elderly	1999	winter	19				5.6		Samat 2000
		1997	summer	27				25		
Baltimore, MD	Retirement Facility	1998	summer	13.0 ± 4.2	23	10.0 ± 4.7	16	22.0 ± 12.0	28	Williams 2000c
		1997	winter	34.4 [$PM_{1.5}$]	NR	17.4	NR	17.0 [$PM_{1.5}$]	NR	Williams 2000a
Boston, MA	Residential Homes	1998	multiple			11.9 ± 9.6	211	11.1 ± 6.8	210	Long 2000
		1996	multiple			13.9 ± 15.2	63	11.7 ± 6.5	64	Abt 2000
Birmingham, AL	Residential Homes	1997-1998	summer	18.6 ± 6.4	30	16.1 ± 9.6	30	26.5 ± 9.5	30	Lachenmeyer 2000
			winter	10.0 ± 3.3	30	11.2 ± 5.4	30	12.2 ± 5.1	30	
Boston, MA	Residential Homes (people with COPD)	1996-1997	winter	21.6 ± 15.2	93	17.2 ± 13.0	93	10.9 ± 9.2	94	Rojas-Bracho 2000
			summer	21.5 ± 11.9	131	17.7 ± 14.9	138	16.4 ± 13.0	138	
Los Angeles	Elderly w/ COPD	1996-1997	winter	24		25		25		Linn 1999
7 U.S. Cities	Asthmatic Children	NR	NR			27.7 ± 35.9	4480	13.6 ± 7.5	4031	Wallace 2003a
United States	Office Buildings	1994-1998	summer-winter			7.2	453	14.7	453	Burton 2000

				PM 10 means - µg/m³						
LOCATION	GROUP	YEAR	SEASON	PERSONAL	(n)	INDOOR	(n)	OUTDOOR	(n)	REFERENCE
Raleigh, NC	African Americans	2000-2001	multiple			27.7 ± 19.6	761	30.4 ± 14.1	761	Williams 2003b
Seattle, WA	COPD Subjects	2000-2001	multiple			14.1 ± 6.6	437	14.3 ± 6.8	435	Liu 2003
	Healthy Subjects					12.6 ± 7.8	206	14.5 ± 7.0	200	
	Asthmatics					19.4 ± 11.1	274	16.4 ± 7.4	269	
	Coronary Heart Dis					16.2 ± 11.3	324	18.0 ± 9.0	324	
Los Angeles, CA	COPD Subjects	2000	winter	35.0 ± 22.0	89	30.6 ± 21.2	95	36.1 ± 13.2	94	Suh 2003
		1999	summer-fall	29.1 ± 12.8	19	29.0 ± 14.7	21	15.2 ± 8.6	21	
Detroit, MI	Asthmatic Children	1999-2000	multiple	68.4 ± 39.2	252	52.2 ± 30.6	363	25.8 ± 11.8	NR	Keeler 2002
Fresno, CA	Retirement Facility	1999	winter			15.1	24	28.2	28	Evans 2000
			spring	37.3	12	16.7	24	28.7	28	
Baltimore, MD	Elderly	1999	winter	28				7.5		Samat 2000
		1997	summer	34				34		
Baltimore, MD	Retirement Facility	1998	summer			13.5 ± 5.9	15	30.0 ± 13.7	28	Williams 2000c
Boston, MA	Residential Homes	1998	multiple			19.4 ± 12.7	212	12.7 ± 7.5	107	Long 2000
		1996	multiple			19.6 ± 16.1	64	17.1 ± 9.1	64	Abt 2000
Boston, MA	Residential Homes (people with COPD)	1996-1997	winter	40.7 ± 26.8	93	37.3 ± 23.2	93	18.5 ± 15.9	95	Rojas-Bracho 2000
			summer	34.7 ± 17.5	132	28.3 ± 25.4	138	24.8 ± 21.9	137	
Los Angeles	Elderly w/ COPD	1996-1997	winter	35		33		40		Linn 1999
Riverside, CA	Residential Homes	1996	fall	150	NR	95	NR	95	NR	Ozkaynak 1996
United States	Office Buildings	1994-1998	summer-winter			11.4	588	23.1	588	Burton 2000

vehicles are their major source. In addition, the loss of NO_3^- may occur indoors due to its dissociation of particulate nitrate back to gaseous form under conditions of increasing temperature and decreasing humidity. NO_3^- and EC comprised a small fraction of personal, indoor, and outdoor PM2.5 (max. 28.5% and 17%, respectively). The indoor PM levels were low compared to those measured in studies of the general population, consistent with the subjects' limited personal activity and very little time spent near smoking, cooking, vehicles, or other major PM sources. Personal PM was highly correlated with indoor PM, and indoor and outdoor PM correlations were significant as well. Statistical modeling results indicated that significant predictors of higher personal PM were time near ETS, location near a major road, higher population density, cooking activity, and location in an inland area (vs. coastal).

A companion study to the above study by Suh and Koutrakis (2004) used a similar design for 16 healthy persons in Los Angeles during the summer and/or winter. The study also involved more detailed characterizations of the PM levels and ventilation characteristics of the subject's homes than the COPD study. Both personal and indoor PM2.5 averaged approximately 18 $\mu g/m^3$ (range 2 - 68 $\mu g/m^3$). Outdoor PM2.5 levels were generally much higher (mean 29 $\mu g/m^3$, range 5 - 103 $\mu g/m^3$) than corresponding indoor and outdoor concentrations in both seasons. Outdoor nitrate levels averaged 11 $\mu g/m^3$ (range 0.3 - 55 $\mu g/m^3$), and were also higher than indoor and personal levels. In contrast, outdoor EC levels were similar to corresponding indoor and personal levels, averaging 2 $\mu g/m^3$ (range 0 - 7 $\mu g/m^3$). Personal exposures to these three particulate measures were more strongly associated with indoor concentrations as compared to outdoor concentrations, which may be attributed to the fact that the subjects spent a majority of their time indoors at home. The investigators found that indoor PM2.5 was the largest contributor to personal PM2.5 levels, accounting for 65-100% of daily personal PM2.5, on average. Significant individual and diurnal variations in all PM measurements, air exchange rates, and PM-generating activities such as cooking and cleaning were also observed.

Far fewer studies have been conducted in public buildings than in residences. Indoor PM concentrations in public and commercial buildings appear to often be lower than ambient concentrations. Lower indoor PM concentrations in public and commercial buildings are due to the use of particle filters in mechanical ventilation systems, un-openable windows, and the lack of many indoor sources typically present in residences. As part of the U.S. EPA's Building Assessment Survey and Evaluation (BASE) study, Burton *et al.* (2000) reported PM concentrations from 100 randomly selected office buildings throughout the United States. Mean indoor PM2.5 and PM10 concentrations were 7.2 $\mu g/m^3$ and 11.4 $\mu g/m^3$, respectively, while outdoor levels were higher, at 14.7 $\mu g/m^3$ and 23.1 $\mu g/m^3$, respectively. However, as with residences, the presence of indoor sources in public and commercial buildings can produce indoor concentrations that exceed concurrent ambient concentrations, especially if smoking is allowed in the building. For example, in a study of 38 commercial buildings in the Pacific Northwest, Turk *et al.* (1987) found that buildings where smoking was permitted had average indoor PM levels 3.5 times higher than concurrent outdoor levels and indoor levels in buildings where smoking was prohibited. Sheldon *et al.* (1988) measured indoor PM in six buildings in the eastern U.S., and found indoor PM concentrations generally lower than outdoors where there was no smoking, but much higher indoor concentrations where smoking was allowed.

Lillquist *et al.* (1998) reported indoor and outdoor PM10 measurements in three Utah hospitals over one winter season. Significant variability in indoor PM levels was found both among room types and among hospitals, and the relationship between indoor PM10 levels and outdoor levels was highly variable. The ICUs had significantly lower PM10 levels than other types of rooms, after adjusting for hospital differences. Thus, the most critically ill individuals may experience

some protection in hospitals from ambient PM; however, in general, hospitals do not offer protection from ambient PM.

2.2.4 Carbon Monoxide

Carbon monoxide (CO) is a colorless, odorless, tasteless gas. It is a product of incomplete combustion, emitted from sources such as vehicles (in exhaust), gas and propane appliances, woodstoves, kerosene heaters, and cigarettes. CO can trigger acute health effects, even death, at very high levels, or flu-like symptoms and other effects at lower levels over longer periods of time.

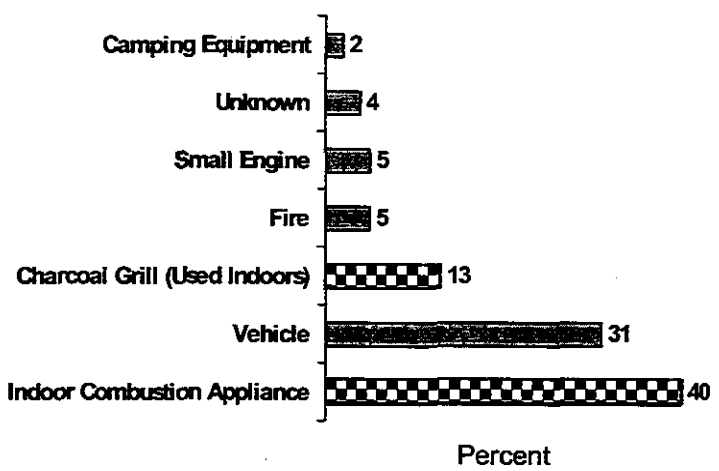
CO is regulated in the ambient environment, but not indoors. However, transient elevated concentrations in outdoor places such as tunnels and parking garages are not widely regulated. The state of California has an 8-hour average ambient air quality standard of 9.0 ppm (10 mg/m^3) and a one-hour standard of 20 ppm (23 mg/m^3). These are also ARB's recommended indoor guideline levels for CO. In one large California study, about 5% of homes were found to have CO levels that exceeded the State 8-hour ambient air quality standard for CO (Wilson *et al.*, 1993).

2.2.4.1 Death

The acute health effects of CO exposure have been well documented in multiple studies since the 1970s. Acute CO poisoning results from a lack of oxygen in the bloodstream due to formation of a CO-hemoglobin complex (carboxyhemoglobin) that prevents oxygen from binding to hemoglobin. Symptoms of acute poisoning include headache, nausea, lethargy and inability to concentrate, unconsciousness, and death at very high concentrations. An estimated 600 deaths per year in the United States throughout the 1990s were attributable to unintentional CO poisoning (cited in Raub *et al.*, 2000).

A California study of ten years of death certificates showed that about 30 - 40 deaths occur in California each year, on average, due to unintentional carbon monoxide (CO) poisoning (Girman *et al.*, 1998; Liu *et al.*, 1993a, 2000). About two-thirds of those deaths were attributable to indoor sources. As shown in Figure 2.2, the indoor sources most implicated in the CO poisonings were combustion appliances (usually malfunctioning or poorly tuned) and charcoal grills and hibachis used indoors. Together these accounted for over 50% of the deaths. The types of cooking and heating appliances included in the indoor combustion appliance category were wall heaters (37%), free-standing heaters (19%), stoves (16%), water heaters (9%), furnaces (9%), and floor heaters (7%).

Figure 2.2: Causes of CO Deaths in California



Natural gas was associated with 75% of the deaths, propane with 24%, and kerosene with less than 1%. Motor vehicles, such as those left running in an attached garage, also took a substantial toll.

2.2.4.2 Non-lethal Health Effects

In an examination of 1991-1994 California CO deaths and hospitalization discharge data, Waldman (1996) found that about 3 to 7 times as many hospitalizations for non-fatal CO poisoning occurred as did deaths from CO poisoning. This totals about 100-300 documented hospitalizations for an average year. However, uncertainties in the patient discharge database and the omission from the database of emergency room discharges that did not result in hospitalization led the investigator to conclude that the actual number of hospitalizations attributable to CO poisoning was at least several times higher. From the pertinent literature, ARB staff and Waldman estimate that five to twenty times (175-700) as many individuals as die from accidental CO poisoning are treated in emergency rooms or hospitalized each year due to serious, non-fatal CO poisonings, and that hundreds to thousands more suffer from undiagnosed heart problems, headache, flu-like symptoms, and other illnesses attributable to CO exposure (Cook *et al.*, 1995; Mah, 2000; Kirkpatrick, 1987; MMWR, 1982; Waldman, 1996). Figure 2.3 illustrates the likely magnitude of these non-lethal effects on the population.

Recent studies have further documented chronic health effects following acute exposures and those due to prolonged exposures to CO. These chronic effects include lethargy, headaches, concentration problems, amnesia, dementia, psychosis, Parkinsonism, memory impairment,

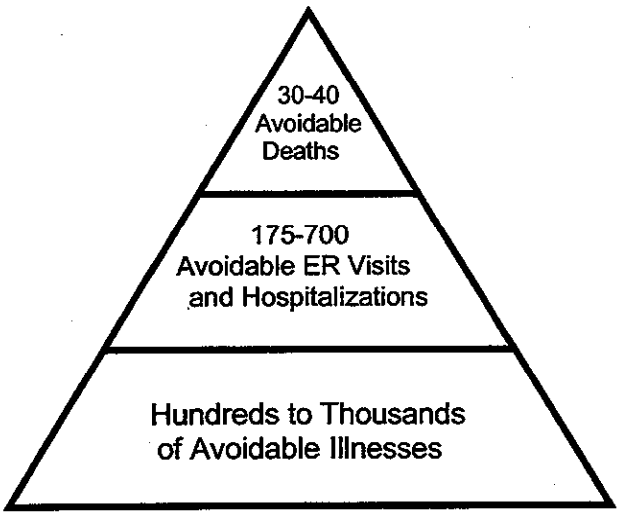


Figure 2.3: Annual California CO Cases

personality alterations, signs of parietal dysfunction, and other minor symptoms (Townsend and Maynard, 2002; Mathieu-Nolf, 2002). In a study of cardiovascular hospital admission data in Los Angeles between 1992 and 1995, Linn *et al.*, (2000) found that CO showed the most consistently significant relationship among the pollutants considered, and concluded that a wintertime increase in CO of 1.1 to 2.2 ppm predicted an increase of 4% in cardiovascular

admissions (~20 extra admissions per day). The elderly are especially susceptible to chronic effects of CO.

Children are especially susceptible to harm from CO. Because they inhale more air per unit of body weight than do adults at similar activity levels, children will inhale a higher dose proportionately than adults, and will experience symptoms more quickly than adults. The unborn appear to be susceptible to adverse effects after exposure to CO as well. In a study of the relationship between ambient air pollution and low birth weight in the northeastern U.S., Maisonet *et al.* (2001) observed increased odds of low birth weight for every 1 ppm increase in CO during the third trimester for the entire population, and a similar increased risk in the African-American population across all three trimesters. In an unrelated study, Ritz *et al.* (2000) estimated that the risk of pre-term birth increases by 12% per 3 ppm increase in CO averaged over six weeks before birth, and by 4% averaged over the first month of pregnancy. Ritz *et al.* (2000) also found an increased risk of heart defects with increased ambient CO exposure during the second trimester of pregnancy. However, attributing the effects strictly to CO is difficult due to the presence of other correlated pollutants. Because these effects were seen with small increases in CO, the presence of indoor sources of CO in homes with pregnant women is a serious concern.

2.2.4.3 Carbon Monoxide Emissions and Concentrations

Most homes have relatively low CO levels, except for short intermittent elevations during use of an indoor source. Indoor CO concentrations can increase rapidly when a highly emitting source is present in an enclosed environment. For example, Jetter *et al.* (2002) measured CO emission rates from incense burning from 159-531 mg/hr, resulting in an estimated peak CO concentration of 9.6 mg/m^3 in a modeled typical room in a home. This is just at the California 8-hour standard level of 9 ppm (10 mg/m^3); in combination with outdoor CO and any additional indoor sources, this home would have exceeded the standard. Pelham *et al.* (2002) reviewed CO levels in indoor ice arenas, stressing that U.S. and global CO exposure problems remain in these locations (only three U.S. states regulate CO levels in ice arenas).

In a study of 277 Californian homes in 1992, Wilson *et al.* (1993) measured indoor and outdoor CO levels. Thirteen homes had indoor 8-hour CO concentrations above the California 8-hour outdoor standard and indoor air quality guideline of 9 ppm. Several homes had indoor CO 1-hour values greater than the state standard of 20 ppm; in one case this was attributed to the (dangerous) use of gas burners for residential heating. In a second California study (focused on PAHs) of 280 homes in northern California, only two homes exceeded California CO standard levels, one due to use of a fireplace and the other from gas heat (Sheldon *et al.*, 1993). However, in many homes, short-term excursions up to 42 ppm were observed, such as when a gas space heater was turned on.

CO levels in well-maintained indoor environments where appliances are operated properly tend to be relatively low. In surveys of 136 'non-problem' buildings in New York City between 1997 and 1999, Springston *et al.* (2002) measured indoor CO concentrations ranging from 0.2 to 10.3 ppm; only 0.04% of all readings exceeded 10 ppm.

A limited number of studies have shown that infiltration of vehicle exhaust emissions into residences can increase the indoor concentration of CO within those residences. Wilson *et al.* (1993) found that the presence of an attached garage was a significant factor related to elevated indoor concentrations of CO versus outdoor concentrations. Gamage *et al.* (1994) found that the CO concentration in the bedroom located above a garage increased from less

than 1 ppm to 17 ppm after a garaged vehicle's engine was operated for a period of 3 minutes. In a detailed Canadian study, Graham *et al.* (2004) measured the concentration of CO inside 16 residences over two seasons while a vehicle in the attached garage was operated under brief cold-start and hot-soak conditions. Significant net changes in indoor CO concentrations were observed to be associated with the vehicle cold start, but not the hot soak running condition.

2.2.5 Nitrogen Dioxide and Associated Acids

Nitrogen dioxide (NO_2) is a red to dark brown gas with a pungent acrid odor. It is emitted from combustion sources such as natural gas and propane-fueled appliances, wood burning stoves and fireplaces, kerosene heaters, charcoal grills and motor vehicles. Adverse health effects attributable to NO_2 include exacerbation of asthma (especially in children), respiratory symptoms and infection, lung damage, and lung disease after long periods of exposure. Several nitrogen compounds related to NO_2 are found in indoor environments; these include nitrous acid (HONO) and nitric oxide (NO). NO_2 is the only nitrogen oxide regulated as a pollutant in outdoor air. California has a one-hour ambient air quality standard for NO_2 of 0.25 ppm, not to be exceeded; this also serves as an indoor air quality guideline. The national ambient air quality standard is an annual mean of 100 $\mu g/m^3$ (0.053 ppm). Based on reports that 20-30% of the population use their gas stove (despite clear warnings against this) for space heating (Phillips *et al.*, 1990), indoor NO_2 is estimated to exceed the State ambient air quality standard at times in 10-30% of California homes.

2.2.5.1 Health Effects of Nitrogen Dioxide

A number of studies have shown that nitrogen dioxide (NO_2) exposure can result in detrimental effects in the lung. Investigators using human clinical studies have recently reported on NO_2 and its effects on airway antioxidant status, inflammatory cell and mediator responses, and lung function (Becker and Soukup, 1999; Barck *et al.*, 2002; Blomberg *et al.*, 1999; Delvin *et al.*, 1999). Data from Delvin *et al.* (1999), for example, demonstrate that 2 ppm NO_2 can induce a mild inflammatory response in the airways of healthy adults, and that NO_2 may cause a mild impairment of lung antibacterial capacity. This study suggests that possible increases in viral clinical symptoms associated with NO_2 may result from effects of the NO_2 on host defenses that normally prevent the spread of virus. Data from European studies indicate that NO_2 is a pro-inflammatory air pollutant under conditions of repeated exposure at a relatively high concentration of 2 ppm, 4 hours per day, for 4 days(Blomberg *et al.*, 1999). However, brief exposures (less than 1 hour) to ambient concentrations of NO_2 can enhance allergic inflammatory reaction in the airways of asthmatics(Barck *et al.*, 2002).

As discussed earlier, the NAS Institute of Medicine's Committee on the Assessment of Asthma and Indoor Air determined that there is sufficient scientific evidence to conclude that high levels of indoor NO_2 can exacerbate asthma (IOM, 2000). A recent epidemiology study conducted in Australia (Pilotto *et al.*, 2003) supports this finding and concludes that asthma symptoms were reduced in primary school children after intervening to remove a high-NO_2-production source at school

Evidence suggests an association between exposure to NO_2 and increased respiratory symptoms in children. Neas *et al.* (1991) studied the effect of indoor NO_2 on respiratory symptoms in 1,567 children aged 7-11 in six U.S. cities from 1983 to 1988. Analysis of symptoms obtained through a questionnaire indicate that a 15 ppb increase in annual (average) indoor NO_2 was associated with an increased cumulative incidence of lower respiratory

symptoms. Girls showed a stronger association (OR = 1.7) than did boys (OR = 1.2). There was not an association between pulmonary function and NO_2 levels.

Chauhan *et al.* (2003) examined the relationships between NO_2 exposure and asthma severity in 8-11 year old children during a respiratory viral infection. Investigators concluded that exposure to 7-day average NO_2 levels of about 11 ppb before the start of a respiratory viral infection is associated with an increase in the severity of virus-induced asthma exacerbations.

California investigators also have reported health effects of NO_2 exposure on children. Peters *et al.* (1999a) studied school children to assess respiratory effects due to long-term exposure to four pollutants: ozone, particulate matter, acids, and NO_2. Peters found NO_2 significantly associated with reduced lung function in female children within a cohort of 3,292 school children in twelve Southern California communities. Increases in bronchitis symptoms of children with pre-existing asthma also were associated with increases in ambient NO_2 levels (McConnell *et al.*, 2003). It is important to note in the McConnel *et al.*, study the annual average concentration of NO_2 was about 19 ppb, well below the national annual average standard of 53 ppb. Within a cohort of 846 asthmatic children residing in eight urban areas of the U.S., Mortimer *et al.* (2002) found a 48% increase in the likelihood of asthma symptoms associated with an increase in the 6-day average NO_2 levels. Within a panel of 138 children in central Los Angeles, Ostro *et al.* (2001) found increased odds for shortness of breath and wheezing associated with a 50 ppb increase in the 1-hour maximum NO_2 concentrations.

Aggregate results from numerous individual studies dealing with exposure to NO_2 and respiratory illness in children have been inconclusive. However, Hasselblad *et al.* (1992) conducted a meta-analysis on studies with inconsistent results to conclude that children exposed to a long-term increase of 30 $\mu g/m^3$ NO_2 (approximately 16 ppb) have about a 20% increase in the odds for developing respiratory illness.

Investigators have also identified a relationship between women (age 20-44) in England who use gas appliances and develop asthma-like symptoms (Jarvis *et al.*, 1996). Women who primarily used gas cooking appliances (known to emit NO_2) had an increased risk for asthma attacks, wheeze, and waking with shortness of breath. The women who used a gas stove also had reduced lung function and increased airway obstruction compared to women who did not use gas stoves. These associations were not observed in men, possibly because they did not experience the high concentrations of pollutants near the cooking source (according to the authors).

2.2.5.2 Nitrogen Dioxide Sources, Emissions, and Concentrations

Nitrogen dioxide is emitted during combustion; sources include motor vehicles, tobacco smoke, and combustion appliances such as gas kitchen stoves, gas, propane, and kerosene-fueled heaters, wood burning stoves, fireplaces, and charcoal grills. In the absence of indoor sources, indoor NO_2 levels are influenced by outdoor levels due to the infiltration of outdoor air (Spengler *et al.*, 1994b; Weschler and Shields, 1994; Levy *et al.*, 1998). When indoor combustion sources such as wall furnaces, floor furnaces, gas stoves, and unvented gas logs (not permitted in California) are present, they have a large influence on indoor NO_2 concentrations (Spengler *et al.*, 1994b; Pitts *et al.*, 1989; Wilson *et al.*, 1986; Wilson *et al.*, 1993).

Several oxidized nitrogen compounds in addition to NO_2 are emitted during combustion by gas appliances and/or are formed through chemical reactions. The most notable additional species, are nitric oxide (NO), nitrous acid (HONO), and nitric acid (HNO_3) (Spicer *et al.*, 1993). Pitts *et*

al., (1985) reported direct observation of the formation of gaseous HONO from the reaction of NO_2 (at ppm levels) with water vapor in indoor environments. Based on removal reactions, indoor NO_2 has a lifetime of about one hour, while the lifetime for NO and HONO is several hours (Spicer *et al.*, 1993). HONO can be retained on indoor surfaces for extended periods, then be released causing elevated concentrations after a combustion appliance is no longer in use (Spicer *et al.*, 1993; Febo and Perrino, 1991). HONO concentrations in homes with gas appliances are typically greater indoors than outdoors and range from 10-20 ppb (Febo and Perrino, 1991; Spengler *et al.*, 1993). HONO is present in indoor air as an acidic aerosol and is likely to be a respiratory irritant, though its respiratory toxicity has not been thoroughly investigated.

Nitrogen dioxide is the most prevalent of the nitrogen oxides and has been the focus of numerous emission and indoor concentration studies. In an ARB-funded cooking study, Fortmann *et al.* (2001) measured indoor NO_2 during various cooking protocols. Measurement periods varied from approximately 1 to 5 hours, representing food preparation, cooking, and clean-up times. Nitrogen dioxide levels increased when a gas stove was used for cooking. For example, while making a fried chicken dinner, average indoor NO_2 levels reached 400 ppb. Other cooking tasks such as broiling fish, baking lasagna, frying tortillas, and stir-frying produced average indoor NO_2 levels ranging from 30 to 170 ppb. During a cycle of automatic oven cleaning with a gas stove, average indoor NO_2 levels exceeded 400 ppb. NO_2 concentrations remained below 45 ppb during the cooking protocols performed with an electric stove and range.

Indoor NO_2 levels can reach unhealthy indoor levels in some situations. In the California Residential Indoor Air Quality Study, indoor NO_2 levels were measured up to 177 ppb as 48-hour averages (Wilson *et al.*, 1993). At this level, it is very likely that the 250 ppb California one-hour standard was exceeded for at least some portion of the time. Dennekamp *et al.* (2001) measured 5-minute peaks up to 1000 ppb NO_2 when cooking with a 4-burner gas stove (measured at face level in front of the cook). In a Boston study, Brugge *et al.* (2003) found indoor NO_2 levels were either close to or exceeded the NAAQS annual level of 53 ppb.

When investigators collect samples over several days, reported concentrations are lower than peak concentrations measured during a distinct exposure event. For example, Lee *et al.* (2002) measured indoor NO_2 and HONO levels in 119 residences in southern California over a 6-day sampling period. The average indoor and outdoor concentrations of NO_2 were 28 and 20.1 ppb, respectively. Zipprich *et al.* (2002) collected 48-hour passive NO_2 samples in Richmond, Virginia. Mean concentrations in the bedrooms, living rooms, and outdoors were 18, 19, and 15 ppb, respectively. 98% of the homes had gas stoves.

Spengler et al. (1994b) measured personal exposures, as well as indoor and outdoor levels of NO_2, for about 700 individuals in the Los Angeles basin. Passive samples were collected over 24- or 48-hour periods. The median personal and outdoor levels were 35 ppb while the median indoor level was 24 ppb. However, the contribution of gas appliances and gas pilot lights to total exposure was evident. "Personal exposures for those in homes with gas ranges with pilot lights average 10 ppb greater than those with electric ranges, and 4 ppb greater than those with gas ranges without pilot lights" (Spengler et al., 1994b).

Levy et al. (1998) identified the use of a gas stove in a home as the most significant contributor to personal NO_2 exposure. Their study, conducted in 15 countries found that mean personal (2-day average) NO_2 exposure was 34.8 ppb in homes with a gas stove that was used during the sampling period, compared to 20.5 ppb in homes without gas stove use.

2.2.6 Ozone

Ozone (O_3), the primary component of smog, is an invisible, yet highly corrosive, odorous, and chemically reactive gas. Ozone is chemically unstable, and so it breaks down or reacts with many surfaces, liquids, and chemicals. Ozone is typically higher outdoors than indoors. It becomes elevated indoors most commonly from the infiltration of outdoor ozone through doors, windows, and swamp coolers, and sometimes from direct emissions indoors by devices such as certain types of copy machines, laser printers, and "air purifiers".

2.2.6.1 Health Effects of Ozone

Ozone is an oxidizing pollutant and strong irritant that attacks the respiratory system, leading to the damage of lung tissue. Exposure to ozone damages the alveoli, the individual air sacs in the lung where the exchange of oxygen and carbon dioxide between the air and blood takes place (ARB/OEHHA, 2004). Increased occurrence of respiratory symptoms, such as cough, pain on deep breath, and difficulty taking a deep breath are associated with exposure to ozone (Schelegle and Adams, 1986; McDonnell *et al.*, 1999; Kulle *et al.*, 1985; Folinsbee *et al.*, 1977; Seal *et al.*, 1993). Ozone also induces cellular and biochemical changes indicative of lung inflammation (Devlin *et al.*, 1991, 1996; Balmes *et al.*, 1996; Aris *et al.*, 1993). A major result from the Children's Health Study indicates that children living in high ozone communities who actively participate in several sports are three times more likely to develop asthma than children in these communities not participating in sports or those that live in low ozone communities (Gauderman *et al.*, 2000).

Epidemiological studies have found statistically significant associations between outdoor O_3 concentrations and various adverse health impacts, including increased asthma symptoms (Wittemore and Korn, 1980; Thurston *et al.*, 1997; Delfino *et al.*, 1996; Mortimer *et al.*, 2002), increased respiratory symptoms (e.g., Brunekreef *et al.*, 1994), and reduced lung function (Brunekreef *et al.*, 1994; Brauer *et al.*, 1996). Some studies also report statistically significant associations between O_3 and hospital admissions or emergency room visits, primarily for asthma or other respiratory causes including COPD and bronchitis (Sheppard *et al.*, 1999; Schwartz, 1995; Delfino *et al.*, 1997, 1998; Burnett *et al.*, 1997; Anderson *et al.*, 1997). Ozone has also been linked to increased school absenteeism for respiratory illnesses (Gilliland *et al.*, 2001), and reduced lung function growth in children (Frischer *et al.*, 1999).

California has an ambient air quality standard for O_3 of 0.09 ppm for one hour, while the federal standard is 0.08 ppm averaged over eight hours. Both standards are currently under review, and California has proposed an 8-hour standard of 0.070 ppm (ARB/OEHHA 2005).

2.2.6.2 Indoor Sources and Concentrations of Ozone

Outdoor air is the most common source of indoor ozone (Weschler *et al.*, 1989). Outdoor ozone (a component of smog; formed by the photochemical reaction of volatile organic compounds and nitrogen oxides emitted primarily by motor vehicles and industries) enters homes through doors, windows, and numerous air leaks in buildings and their ventilation systems. Studies have shown that indoor ozone levels generally follow the diurnal and seasonal patterns of outdoor ozone, with higher levels in the daytime and summer months (Liu *et al.*, 1993b; Weschler *et al.*, 1994; Liu *et al.*, 1995; Avol *et al.*, 1998; Geyh *et al.*, 2000; Lee *et al.*, 2002). Like outdoor concentrations, indoor ozone levels can also remain elevated for long periods of time (eight hours or more), and display peak variations throughout the day (Weschler *et al.*, 1989). Indoor ozone levels typically range from 20 to 80% of outdoor ozone levels (Weschler *et al.*, 1989).

Using a swamp cooler or whole-house fan on a high-ozone day can increase air exchange rates enough to produce indoor ozone levels very close to outdoor levels for hours at a time (Avol *et al.*, 1996).

The most common indoor sources of O_3 are poorly maintained laser printers and photocopiers, and O_3 generating-devices that are marketed as various types of room deodorizers and air cleaners. In particular, ozone generators that are marketed as "air purifiers" have been found to produce hazardous levels of indoor ozone. Several studies have shown that ozone at levels produced by ozone generators does not effectively control indoor air pollution, odors, or mold growth on surfaces (Boeniger, 1995; Kissel, 1993; Foarde *et al.*, 1997). Ozone generators can destroy microorganisms and gases, but only at concentrations unsafe for occupied spaces. In addition, ozone from ozone generators can react with indoor surfaces, such as latex paint, or airborne chemicals, including the fragrance compounds from commercial air fresheners, to produce toxic and irritating byproducts such as formaldehyde (Kleno *et al.*, 2001; Wainman *et al.*, 2000; Weschler, 2000; Weschler and Shields, 1999; Moriske *et al.*, 1998; Reiss *et al.*, 1995ab; Weschler *et al.*, 1992). Most importantly, these devices can result in levels of ozone well above health-based standards and guideline levels. For example, a "personal air purifier" tested for its emissions when used according to manufacturer's directions resulted in ozone levels in the users breathing zone that exceeded various standard levels, including the California ambient air quality standard (Phillips *et al.*, 1999).

Other sources of indoor ozone include other types of electronic air cleaners and office equipment that uses electrostatic processes. Negative ion generators and electrostatic precipitators (ESP) and dry-process copiers, laser printers, and fax machines can generate significant levels of indoor ozone as a by-product (U.S. EPA, 1995; Kissel, 1993; Selway *et al.*, 1980; Allen *et al.*, 1978).

2.3 TOXIC AIR CONTAMINANTS AND OTHER INDOOR AIR POLLUTANTS

There are a number of other important indoor pollutants that are somewhat unique in their exposure parameters or health effects. Toxic air contaminants (TACs) are air pollutants "which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health" (HSC Section 39655). Once a compound is identified as a toxic air contaminant, ARB determines the need and appropriate degree of regulation for the compound. Regulations have been implemented to control the release of numerous TACs into outdoor air; however, regulations do not presently exist to control their release into indoor air. Foremost among these pollutants are formaldehyde, chloroform, *p*-dichlorobenzene, benzene, radon, polycyclic aromatic hydrocarbons (PAHs), and environmental tobacco smoke (currently in the identification process). TACs were also identified by the federal government as Hazardous Air Pollutants (HAPs); many are carcinogenic. Semi-volatile pollutants such as pesticides, phthalates, and polybrominated diphenyl ethers can be released indoors, or tracked indoors from outdoors. The health effects, indoor sources, and indoor air concentrations of these pollutants are discussed in this section.

2.3.1 Formaldehyde

Formaldehyde is a pungent smelling gas emitted from numerous indoor sources. These include many building materials (especially pressed wood products), some new carpet assemblies, composite wood furnishings, consumer products, permanent pressed clothing, and combustion sources. Formaldehyde is listed as a TAC and a Proposition 65 substance, based on its carcinogenicity. It is also an upper respiratory tract irritant that produces eye, nose, and throat

irritation. Indoor formaldehyde concentrations nearly always exceed outdoor levels due to the many indoor sources. Indoor and urban ambient levels typically exceed the OEHHA Chronic Reference Exposure Level (REL) of 2.4 parts per billion (ppb), which is based on irritant effects on the mucous membranes of the upper airways and eyes, and levels sometimes exceed OEHHA's 8-hour REL of 27 ppb designed to protect against the same effects. Nearly all indoor environments also exceed the one-in-a-million cancer risk level.

2.3.1.1 Health Effects of Formaldehyde

A number of adverse health effects in humans have been associated with formaldehyde exposure. Short-term effects include eye, nose, throat and skin irritation; nausea; headache; and there is limited evidence for exacerbation of asthma. Dermal allergic sensitization may occur following relatively high occupational exposure. People vary substantially in their sensitivity to formaldehyde. For most individuals, effects typically occur at exposure levels between 0.037 and 3 ppm (ARB, 1992; U.S. EPA, 1987b). Sensitive individuals may experience acute symptoms related to irritation at lower concentrations. OEHHA has set the acute REL, based on a one hour exposure, at 94 $\mu g/m^3$ (75 ppb) with eye irritation as the toxicological endpoint (OEHHA, 2000a). The OEHHA interim REL, based on an 8-hour exposure, is 27 ppb designed to protect against the same effects.

The ARB identified formaldehyde as a TAC in 1992, based on its carcinogenic potential. In 2004, the International Agency for Research on Cancer (IARC) classified formaldehyde as a human carcinogen due to sufficient evidence for development of nasopharyngeal cancer in humans. IARC also found limited evidence that formaldehyde may cause other respiratory tract cancers, and a possible link with leukemia. The California Proposition 65 No Significant Risk Level for formaldehyde is 40 µg/day (equivalent to 1.6 ppb, based on inhaling 20 m^3/day). This level represents the daily intake level calculated to result in a cancer risk of one excess case of cancer in 100,000 individuals exposed over a 70-year lifetime (OEHHA, 2004).

2.3.1.2 Sources of Formaldehyde

Many materials and products emit formaldehyde. However, emissions studies have shown that building materials, particularly composite wood products, are likely the greatest contributors to formaldehyde in indoor air. Kelly *et al.* (1999) reported the highest emission rates for numerous composite wood products manufactured with urea-formaldehyde resin. These products, commonly used in home construction, cabinetry, and furniture, displayed formaldehyde emission rates ranging from 8.6 to 1,580 $\mu g/m^2/hr$. Over half of the urea-formaldehyde products tested had emission rates between 100 and 200 $\mu g/m^2/hr$. A covering over the wood such as a paper laminate, melamine laminate, or vinyl coating substantially reduced the emission rates to levels at or below 55 $\mu g/m^2/hr$ for all products tested. Composite wood products designed for outdoor use are made with phenol-formaldehyde resin. When tested, these products emitted 4.1 to 9.2 $\mu g/m^2/hr$ formaldehyde, a substantial reduction from the urea-formaldehyde resin products. Coated products and phenol-formaldehyde resin products are preferred alternatives to urea-resin products.

After numerous lawsuits in the 1970s and 1980s, the composite wood industry developed voluntary emission standards for medium density fiberboard (MDF) and particleboard. A comparison of emission rates from Pickrell *et al.* (1983) and Kelly *et al.* (1999) showed that the emission rates from current composite wood products averaged 49% lower than the emissions in the early 1980s. Industry data provided to ARB by the Composite Panel Association indicate that emissions of particleboard have decreased by 80% in this time frame. In response to an

ARB survey, members of the composite wood industry responding to the survey (53%) indicated 100% of their particleboard meets the HUD large chamber test concentration of 0.3 ppm (this chamber concentration is not equivalent to the concentration that would be expected in a home). Of the products Kelly *et al.* (1999) tested, all of the bare MDF products and most of the particleboard samples were below the industry limits.

Formaldehyde emissions are greatest when building materials are new, and it takes years to complete the off-gassing. Sexton *et al.* (1986) carefully designed a study to measure indoor formaldehyde levels relative to the age of manufactured homes. Investigators found statistically significant higher formaldehyde concentrations in newer homes than older homes. For example, data collected during the summer indicated the mean concentration for houses less than four years old were 80 ppb, compared to houses older than four years with a mean concentration of 61 ppb. Data from winter sampling reveal mean concentrations of 90 ppb for newer homes and 64 ppb for older homes. Data from the study was further analyzed by the age of the mobile home (Sexton *et al.*, 1989). For homes manufactured from 1980 to pre-1966, the data follow the expected pattern, with indoor values gradually decreasing with increasing age. The highest average concentration was in homes manufactured in 1980. Although homes manufactured in 1981 and 1982 had maximum values greater than those from 1980, the mean concentrations were less. The mean for homes manufactured in 1983 continued this downward trend. Investigators comment that the particle board industry began introducing products with lower formaldehyde emission rates at this time. It is likely that these changes are reflected in the lower formaldehyde levels observed in mobile homes built after 1980.

Composite wood products still release high levels of formaldehyde to the indoor environment for long periods of time–from months to years. Brown (1999), an investigator in Australia, measured formaldehyde emission rates from particleboard, medium density fiberboard, and office furniture over several months. Emission rates measured in the study declined from a value of 300-400 $\mu g/m^2/hr$ for relatively new products to 80-140 $\mu g/m^2/hr$ for products 5 to 10 months old.

In a recent study funded by the California Integrated Waste Management Board (CIWMB) and conducted by DHS (Alevantis, 2003), investigators identified nine products in six categories that emitted formaldehyde at levels high enough to exceed the California Section 01350 guideline level (see Section 4.3.3.2 of this report). When modeling (for use in a state office) was conducted independently on emissions from acoustical ceiling panels, a carpet, medium density fiberboard, gypsum board, resilient flooring (non-rubber based), and thermal insulation, room concentrations were estimated to exceed 16.5 $\mu g/m^3$ (13.5 ppb), the upper bound allowed for formaldehyde contribution from a single product under Section 01350 guidelines. For thermal insulation, products exceeding the upper limit included a standard product and a product marketed as formaldehyde-free. Only one of the ten standard resilient flooring samples exceeded the limit, while six of the samples had undetectable levels of formaldehyde. The nine products with elevated emissions accounted for 11% of the samples tested in this study. Formaldehyde was detected in 34% of the total samples tested.

Since the creation of the Section 01350 emission guideline for building materials, many manufacturers have met the requirement for a variety of products. A list of these compliant materials is available at http://www.chps.net/manual/lem_table.htm.

Recent studies in other states provide additional information on source contributions of indoor formaldehyde. In a manufactured house produced in Florida, Hodgson *et al.* (2002) determined that the greatest contributors to indoor formaldehyde levels were a particleboard cabinet case and passage doors, each contributing about 33% of the total house formaldehyde

concentration. The house, which was a furnished sales model, had an indoor formaldehyde concentration of 77 ppb. The combined emission rate for all wood products in the house was approximately 10 mg/hr. The other 33% of formaldehyde was contributed by cabinet stiles, countertops, subflooring, and other sources. The authors recommended several techniques for reducing indoor residential formaldehyde levels.

- Use vinyl coated or alternative passage doors.
- Use fully coated particleboard in cabinet cases.
- Use frameless cabinets.
- Apply a laminate backing to the underside of particleboard countertops.

Floor finishing materials such as new carpet assembly components and vinyl flooring may also emit formaldehyde. In a study funded by the ARB, Hodgson (1999) measured the formaldehyde emissions of several flooring products. Results for formaldehyde emissions at 24 hours showed the following.

- Formaldehyde chamber concentrations were generally below the limit of detection of 1 $\mu g/m^3$ for carpet.
- One carpet cushion had an emission rate of 8 $\mu g/m^2/hr$.
- Seam tape applied to carpet had an emission rate of 5 $\mu g/m^2/hr$.
- Emissions from five different sheet vinyl flooring samples were less than or equal to 4 $\mu g/m^2/hr$.
- Adhesives applied to sheet flooring and cove base had emission rates ranging from 72 to 258 $\mu g/m^2/hr$.
- When the vinyl flooring or coving was placed on top of the adhesive, the surface product served as a relatively effective barrier, causing a drop in emissions.

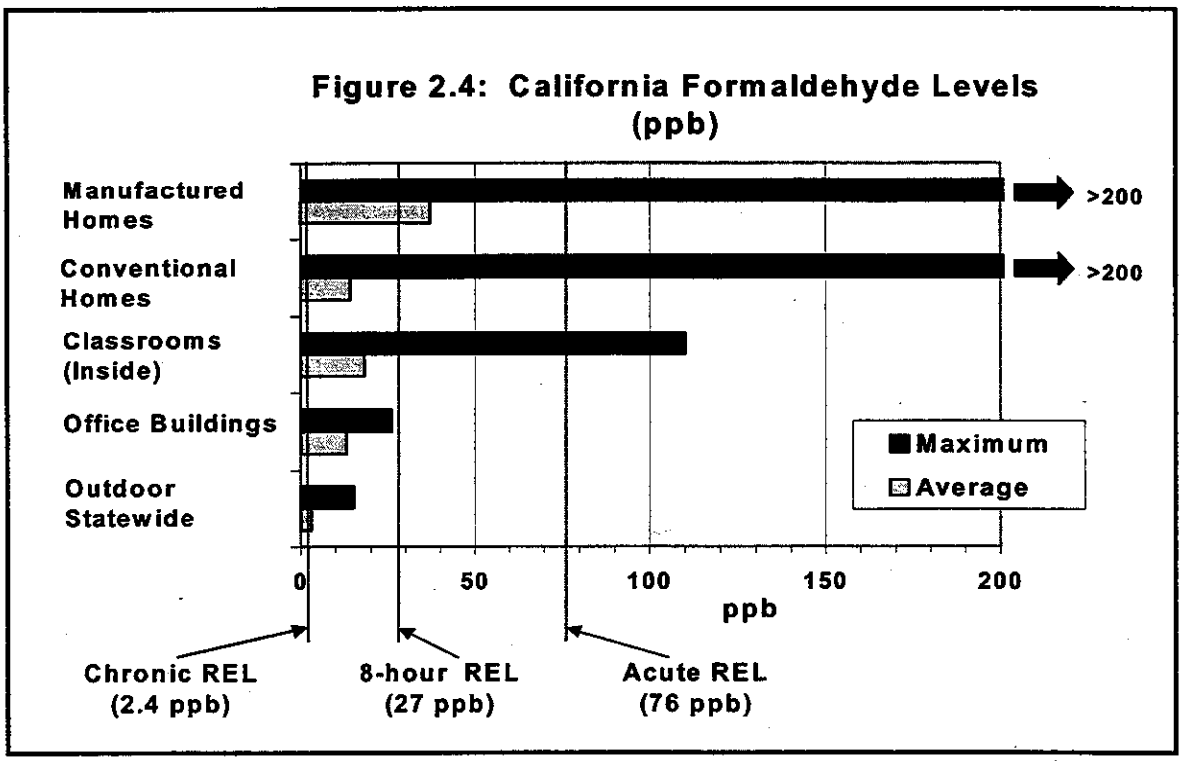
Paint is another building material known to emit formaldehyde. Hodgson (1999) measured formaldehyde emissions from 10 different paints widely used in California. As with most wet products, concentrations peaked initially, then declined over several hours. At 96 hours, five paints had emission rates greater than 10 $\mu g/m^2/hr$. To address the desire for lower-emitting products, many paint manufacturers are formulating low-VOC paints. These paints have substantially lower total VOC emissions than traditional paint; however, they may emit formaldehyde. Two of the paints in the study were identified as "non-VOC", however their formaldehyde emissions rates at 48 hours were 43 and 12 $\mu g/m^2/hr$. Chang *et al.* (1999) also evaluated emissions from low-VOC paints. In small chamber tests, two of four low-VOC paints emitted formaldehyde with peak concentrations at 3.15 mg/m^3 and 5.53 mg/m^3.

2.3.1.3 Indoor Formaldehyde Concentrations

Indoor levels of formaldehyde can reach high levels in some indoor locations despite changes in the manufacture of pressed wood products and changes in the construction of manufactured housing. Current estimated average and maximum indoor formaldehyde concentrations are shown in Figure 2.4 for different California environments. Figure 2.4 illustrates that concentrations in some homes and schools exceed OEHHA's interim 8-hour REL of 27 ppb, established to identify levels above which sensitive individuals could experience acute eye, nose, and lung irritation. The figure also shows the relative levels of formaldehyde in different environments, indicating that manufactured homes are of greatest concern, while conventional homes and classrooms are also of concern.

Figure 2.4 also illustrates that levels in virtually all indoor environments exceed OEHHA's chronic REL (2.4 ppb) for irritant effects and OEHHA's one-in-a-million excess lifetime cancer risk level (0.13 ppb) for formaldehyde. It is generally not feasible to achieve levels below these guideline levels because outdoor levels average about 3-5 ppb.

Data for Figure 2.4 have been compiled from several sources. Due to the lack of more recent field studies that measured indoor formaldehyde levels, adjustments have been made to the results from older studies. Manufactured home levels are from a large, older study of manufactured homes (Sexton *et al.*, 1985), with levels adjusted downward based on the



average reduction in emissions from manufactured wood products since 1983 (Kelly *et al.*, 1999). The conventional home data were obtained by combining results from a 1996 study of southern Californian homes (Avol *et al.*, 1996) and the National Human Exposure Assessment Survey (NHEXAS) exposure study in Arizona (Gordon *et al.*, 1999). The maximum values estimated for current California manufactured and conventional homes are similar, between 220-240 ppb, although measured values have been obtained well above these levels in some studies. The classroom data are taken from the California Portable Classrooms Study (Whitmore *et al.*, 2003), using both Phase I and Phase II data. Concentrations in office buildings are from the U.S. BASE study (U.S. EPA, 2003a; Girman *et al.*, 1999) of about 100 medium and large office buildings throughout the U.S., including buildings from California. The sources and derivations of the averages and maxima shown in Figure 2.4 are discussed in Appendix III.

New homes built with standard construction products are likely to have unhealthy levels of formaldehyde. Hodgson *et al.* (2000) measured formaldehyde concentrations inside homes two months old, then continued measurements for a seven and one-half month period. The geometric mean concentration of formaldehyde in four manufactured homes was 34 ppb, and in seven site-built homes was 36 ppb. Both were substantially greater than the outdoor formaldehyde level of 6 ppb, and above health-based guidelines. The formaldehyde emission

rates were fairly consistent over the seven to eight month sampling time, indicating that the off gassing of formaldehyde emissions from building materials in new homes extends for a long period of time. The author commented that these levels are approximately 50% lower than formaldehyde concentrations measured during the 1980s due to use of less plywood paneling in manufactured homes and reduced emission rates from composite wood products relative to 20 years ago. Emission rates were calculated for these homes and found to be 45 $\mu g/m^2/hr$ for the manufactured homes and 31 $\mu g/m^2/hr$ for the site-built homes.

Older housing stock has greatly reduced formaldehyde off-gassing rates relative to new housing stock. As mentioned in Section 2.3.1.2, longitudinal formaldehyde concentration studies extending over years do not exist and emission rates are generally not derived for older houses. However, the body of data on home formaldehyde concentrations indicates newer homes have higher levels than older homes (Hodgson *et al.* 2000; Gordon *et al.*, 1999). Therefore, new homes, or homes with recent remodeling are of most concern relative to formaldehyde exposure.

In the most recent large-scale study of formaldehyde levels in homes (NHEXAS), conducted in Arizona, 25% of the homes sampled had formaldehyde levels above the 8-hour REL of 27 ppb. This is of concern because people are typically in their homes for longer than 8 hours a day and the study was designed to be representative of the general population. The formaldehyde limit of detection was fairly high at 10 ppb, so that formaldehyde was detectable in only 69% of the indoor air samples (131/189) with a median concentration of 21 $\mu g/m^3$ (17 ppb). The 75th and 90th percentiles in residences were 34 $\mu g/m^3$ (28 ppb) and 46 $\mu g/m^3$ (37 ppb), respectively, with a maximum value at 408 $\mu g/m^3$ (332 ppb). These investigators also commented that mean values are lower than those reported in the 1980s (Gordon *et al.*, 1999).

Although not evident in Figure 2.4, the results from Phase II of the California Portable Classroom Study (PCS) indicated that formaldehyde levels in at least 4% of California classrooms exceed OEHHA's interim 8-hour REL of 27 ppb (Whitmore *et al.*, 2003), the level at which an 8-hour exposure might result in irritant effects. This is equivalent to about 10,720 classrooms, or at least 214,400 children (assuming 20 children per classroom...usually there are more) exposed to formaldehyde levels that could potentially result in irritant effects in sensitive individuals. Average and peak levels in the classrooms were somewhat higher than those measured in the U.S. EPA BASE study of public and commercial buildings, indicating that, during the day, school children may experience greater exposures than most adults. Data analyses from the PCS indicate that several factors were associated with indoor formaldehyde levels in classrooms including the presence of plywood and particleboard; vinyl tackboard; bookcases and cabinets made of pressed wood; increased temperature and humidity; and classroom age (higher levels in newer classrooms) (Whitmore *et al.*, 2003).

2.3.2 Volatile Organic Chemicals

Volatile organic chemicals (VOCs) is a generic term for thousands of compounds with widely varying physical, chemical, and toxicological properties. VOCs include a variety of chemicals such as alkanes, alcohols, esters, ethers, and aromatic compounds. A number of specific VOCs are widely found in indoor environments and are known to have adverse toxicological properties. Potential indoor sources of VOCs include building and furnishing materials such as carpet, paint, and vinyl flooring; consumer products such as air fresheners, adhesives, and cleaning agents; water treated with chlorine; dry-cleaned clothing; environmental tobacco smoke; plastic products, computers, and others.

Many of the 'traditional' VOCs reported in indoor air have been studied simultaneously because, due to their chemical properties, they are collected via the same medium and analyzed through the same processes. Scientific study has only touched the 'tip of the iceberg' in understanding all VOCs in indoor air. Complex compounds such as fragrances are widely used, minimally regulated, and an area of emerging concern for environmental impacts (Bridges, 2002). Many semi-volatile compounds that can be present in the gaseous phase, or adsorbed to particles have not been extensively studied (Rudel *et al.*, 2003). Because the link between indoor air pollutants and health impacts is so complex, researchers are investigating the reaction of VOCs with oxidants present in indoor air and are discovering new areas for future study (Carslaw, 2003; Weschler, 2004; Wolkoff and Nielsen, 2001; Wolkoff *et al.*, 2000).

Some VOCs have been identified as California TACs or federal HAPs due to their cancer-causing potential. Some VOCs also cause eye, nose, and throat irritation, and neurological effects such as headache. Indoor and personal exposure levels of these VOCs are typically higher than concurrent outdoor levels, and may exceed acceptable cancer risk levels and other health-based guideline levels. Ambient VOC levels are generally considered 'background' levels for indoor pollutants due to infiltration of outdoor air, thus any additional pollutant released indoors results in a concentration greater than outdoors.

2.3.2.1 Health Effects of VOCs

The ability of organic chemicals to cause health effects varies greatly from those that are highly toxic, to those with no known health effect. Some of the VOCs found in indoor air in California have been identified as TACs based on their carcinogenic potential. Accordingly, there is no level of exposure to these chemicals that is known to be absolutely safe. However, health effects are determined not only by the specific toxicology of the air pollutant but also by the extent of exposure and the absorbed dose. The higher the exposure and dose, the higher the risk of adverse health effects. More information on dose-response can be found on the OEHHA website (www.oehha.ca.gov). Some of the more common carcinogenic indoor VOCs were listed earlier in Table 2.4. The cancer risk posed by some of the most prevalent VOCs was discussed above in Section 2.1.2, and a detailed discussion of those risks is provided in Appendix II.

Many indoor VOCs also can irritate the eyes, nose, and throat (OEHHA 2000a; OEHHA 2003a). They are emitted from a wide range of sources and have a variety of impacts on occupant health and comfort (Hodgson *et al.*, 1994; Wolkoff, 1995; Molhave, 1991b). Hodgson and Levin (2003a) compared indoor VOC concentrations to odor thresholds, sensory irritation levels, and noncancer chronic health guidelines. "The methodology demonstrated that only a small number of the more than 100 reported indoor VOCs exceeded levels likely to be of concern with respect to the endpoints considered. The results indicated carboxylic acids and less volatile aldehydes and aromatic hydrocarbons are most likely to be perceived by olfaction and that the probability of detection is higher in residences than in offices. Although more consideration of the underlying toxicological data is needed, the results suggested only a few commonly measured VOCs, considered singly, are likely to produce serious irreversible health effects not associated with cancer". The authors conclude, "For a few compounds, such as acrolein and formaldehyde, the evidence based on sensory irritation and chronic toxicity is sufficient to warrant efforts to reduce and otherwise control the sources of these compounds in buildings."

At higher concentrations (usually not encountered in homes or offices, but sometimes found in occupational settings), some VOCs can impact the nervous system, causing acute effects such as nausea, tremors, drowsiness, dizziness, and headache (OEHHA 2000a; OEHHA 2003a). Such VOCs include, most notably, aromatic hydrocarbons (such as benzene), chlorinated

chemicals (such as perchloroethylene), and some pesticides. While concentrations that can trigger neurotoxic effects are not normally experienced in homes and offices, special circumstances can lead to elevated exposures in some specialized non-industrial workplaces. The nature of activities and products used in medical offices, hospitals, beauty salons, high production copy shops, and other non-industrial workplaces can lead to unusually high concentrations of some pollutants. Business parks that merge industrial and non-industrial businesses may also create unique situations leading to exposure to elevated levels of these pollutants.

Exposure of pregnant women to organic solvents may affect the neurodevelopmental outcome of their children. After controlling for potential confounding related to maternal IQ and maternal education, Laslo-Baker *et al.* (2004) found that children exposed in utero to organic solvents obtained lower scores on subtests of intellect, language, motor, and neurobehavioral functioning. Many of the occupations were non-industrial and included painter, hair stylist, salon receptionist, and science teacher. Exposure levels were not reported.

2.3.2.2 Sources and Emissions of VOCs

Studies have shown that many indoor sources emit VOCs, that some indoor sources emit substantial amounts of VOCs, and that groups of related VOCs often have common sources. Exposure to VOCs is influenced by people's activities and their proximity to sources of pollutants. Exposure studies indicate the greatest exposures generally result from close contact with specific sources, such as cleaning products. Manufacturers strive to produce products that meet consumers' needs and can be used safely; however, VOCs are often required in the manufacture of products to impart desired properties for a given application. This results in trade-offs: proper use of some cleaning products, for example, can remove biological contaminants and some allergens and asthma triggers in the indoor environment, yet occupants sensitive to the odor and irritant effects of the VOC components may be affected.

<u>Chlorinated Solvents</u>
Chlorinated hydrocarbons, a large group of VOCs with solvent properties, contain one or more chlorine atoms. Chemicals in this group have diverse sources. Levels of perchloroethylene, identified as a California TAC due to its carcinogenic potential, can increase when dry-cleaned clothes are brought into a house. Levels in a home containing recently dry-cleaned clothes can be 100 to 150 times greater than outdoor levels of perchloroethylene (Wallace, 2001). Levels of methylene chloride, another chlorinated hydrocarbon and TAC, have been greatly reduced in consumer products; however, it is still common in paint strippers with a label warning to use adequate ventilation. Short-term exposures can be significant for individuals who use paint strippers (Wallace, 1991).

Para-dichlorobenzene (*p*-DCB), another chlorinated solvent, has been used as an "air freshener," although it is listed as a TAC due to its potential carcinogenicity. Data from the U.S. EPA's TEAM studies indicated that, at the time the study was conducted, about one-third of homes used products containing *p*-DCB (Wallace, 1991). In another study, the indoor air concentration of *p*-DCB increased from 1 $\mu g/m^3$ to more than 500 $\mu g/m^3$ the day after a toilet bowl cleaner was introduced into a home (Wallace, 2001). In the NHEXAS study, the two highest *p*-DCB levels measured in Arizona homes were 3949 and 4400 $\mu g/m^3$, presumably from the introduction of household consumer products (Gordon *et al.*, 1999). These examples illustrate the extremely high concentrations of chlorinated chemicals that can occur in residences.

Chlorine is intentionally added to domestic water for public health purposes. However, chlorine and organic matter in the water react to produce chloroform, another compound listed as a California TAC. Exposure to chloroform and other trihalomethanes occurs from drinking water, taking showers and baths, and operating washing machines (Wallace, 2001). In one study, investigators measured chloroform concentrations while individuals actually took showers in residential shower stalls. Average concentrations during and immediately after the shower ranged from 67 $\mu g/m^3$ to 265 $\mu g/m^3$. The chronic REL for chloroform is 300 $\mu g/m^3$ (OEHHA, 2003a). Air concentrations of trihalomethanes were about three times higher on average for a typical shower compared to a bath. Higher concentrations were observed with hotter water (Kerger *et al.*, 2000). Chloroform can also be produced during the use of bleach and other cleaning agents that contain chlorine. Investigators predicted that 5.3 mg to 9.8 mg of chloroform can be released to indoor air during a ten-minute wash cycle when a laundry bleach containing sodium hypochlorite is used in a residential washing machine (Shepherd *et al.*, 1996). If this amount of chloroform is released into a 10 foot by 10 foot room (22.7 m^3), the chloroform concentration would be 230 to 430 $\mu g/m^3$, the high end of which would exceed the chronic REL.

Benzene and Other Aromatic Hydrocarbons

Benzene, styrene, toluene, and xylene are aromatic hydrocarbons containing a hexagonal ring structure. In general, the major sources of these chemicals in the indoor environment are environmental tobacco smoke, motor vehicle exhaust, and evaporative emissions from vehicles in attached garages. To a lesser extent, consumer products such as solvents, adhesives, glues, and paint emit some of these VOCs, especially toluene (Wallace *et al.*, 1988; Akland and Whitaker, 2000; Guo *et al.*, 1999). The overwhelming source of benzene exposure for smokers is mainstream cigarette smoke (Wallace, 1996b); nonsmokers living with smokers may experience a substantial benzene exposure due to ETS as well (Wallace, 2001). For nonsmokers with infrequent exposure to ETS, most benzene exposure comes from auto exhaust, especially while traveling on busy roadways (Rodes *et al.*, 1998), gasoline vapor emissions during fueling at gas stations, and in houses with an attached garage (Wallace, 1996b).

Some of these VOCs are also emitted from office copy machines and printers (Lee *et al.*, 2001). Several investigators have studied the emission of aromatic hydrocarbons from office machines. Numerous VOCs, including benzene, ethylbenzene, *o,m,p*-xylene, and styrene were emitted from a dry-process photocopier studied by Brown (1999). The author noted a 40% increase in VOC emission rates for double-sided operation versus single-sided operation. Leovic *et al.* (1996) measured emissions from 4 dry-process photocopy machines. The compounds with the highest emission rates overall were ethylbenzene (highest emission at 28,000 µg/hour), *o,m,p*-xylene, 2-ethyl-1-hexanol, and styrene (lowest emitter of these compounds at 12,000 µg/hour). The relative amounts of individual compounds varied with the machine. Other investigators (Wolkoff *et al.*, 1993) measured emissions from toners and processed paper from office copiers and printers. They also report substantial variation in emissions between machines. Aromatic compounds such as toluene, xylenes, ethyl and propyl benzene, and styrene dominated the emissions. The authors concluded "a realistic estimate (assuming first order decay) of handling 200 freshly processed copies in a 17 m^3 office room, 0.25/hour air exchange rate, and an emission of 6 $\mu g/m^2hr$ could reach a styrene concentration, assuming complete mixing, of 12 $\mu g/m^3$ from the processed paper." This concentration is well below the chronic REL of 900 $\mu g/m^3$, however results indicate total emissions including ozone and particles can have a significant impact on indoor air quality (Wolkoff *et al.*, 1993; Leovic *et al.*, 1996).

In an unpublished study of over 100 products sold in California stores, Akland and Whitaker (2000) detected toluene most frequently in auto care products, glues, and personal care products, such as in fingernail polishes. Estimated maximum air concentrations and daily doses were high, up to 4000 µg/day. In the Arizona NHEXAS study, investigators measured toluene levels at non-smokers' homes and found an average level of 24 µg/m^3 in homes with an attached garage (n=40), but only 5 µg/m^3 in homes without an attached garage (n=9) (Gordon *et al.*, 1999). These concentrations are below the chronic inhalation REL for toluene of 300 µg/m^3 (OEHHA, 2003a).

VOCs from Building Materials

The pollutants discussed above are associated with one or a few predominant sources. But, many sources, such as building materials emit numerous VOCs. A number of studies have been conducted to examine VOC emissions from a variety of building materials. Most recently, a building material emissions testing study funded by CIWMB reported that building materials emit a number of VOCs that were identified as chemicals of concern (Alevantis, 2003). Several products in each of the categories identified below exceeded the Section 01350 guideline levels as follows:

- Carpet exceeded the emission rate limits for naphthalene and acetaldehyde.
- Fiberboard and particleboard exceeded the limits for acetaldehyde.
- Resilient flooring products exceeded the limit for acetaldehyde, naphthalene, and phenol.

As was mentioned in the section on formaldehyde, products meeting Section 01350 emission guidelines are listed at http://www.chps.net/manual/lem_table.htm.

In a study funded by ARB, Hodgson (1999) identified 17 toxic air contaminants in the emissions from new carpet assembly, vinyl flooring, and latex paint. These TACs are routinely emitted to the indoor environment, particularly in new or recently remodeled homes and offices. Table 2.6 contains a list of TACs identified by Hodgson in building material emissions. In addition to TACs, all of the bonded urethane carpet cushions emitted butylated hydroxytoluene (BHT), an irritant, and all carpet samples emitted 4-phenylcyclohexene (4-PC), the compound largely responsible for new carpet odor. The investigator tested a limited number of products on the market at the time. The study conducted by Alevantis (2003) contains more recent emissions data, reflective of current manufacturing processes. It also compares emissions to health-based guidelines.

Table 2.6. Toxic Air Contaminants Emitted From Building and Finishing Materials

Carpet and Cushion	Paint	Vinyl flooring
Toluene	*m,p*-Xylene	Toluene
m,p-Xylene	Ethylene glycol	*m,p*-Xylene
o-Xylene	2-(2-Butoxyethoxy)ethanol	*o*-Xylene
Styrene	Formaldehyde	Styrene
Ethylene glycol	Acetaldehyde	1,2,4-Trimethylbenzene
Formaldehyde		Naphthalene
Acetaldehyde		Acetophenone
Acetophenone		Phenol
2-(2-Butoxyethoxy)ethanol		Formaldehyde
Ethylbenzene		Acetaldehyde
Tetrachloroethane		
Naphthalene		
Phenol		

Emissions from polymeric building materials can be attributed to several factors (Yu and Crump, 1998).

- Solvent residues from the manufacturing process.
- Unreacted monomers trapped in the product structure.
- Secondary products from reactions of monomers.
- Plasticizers used in production.

Measurement of VOCs in new homes is another method of determining the variety of compounds emitted from building materials. Hodgson *et al.* (2000) identified α-pinene, formaldehyde, hexanal, and acetic acid as the predominant compounds measured in 11 new homes. For manufactured houses and site-built houses, the geometric mean total volatile organic chemicals (TVOC) emission rate was 1.7 mg/m^2-h, and 2.1 mg/m^2-h, respectively. Individual compounds with the highest emission rates (greater than 50 $\mu g/m^2$-h) include α-pinene, ethylene glycol, hexanal, acetic acid, β-pinene, and 2,2,4-trimethyl-1,3-pentanediol monoisobutyrate (TMPD-MIB).

Paint not only emits numerous TACs, but emissions can continue for extended periods of time. Sparks *et al.* (1999) estimated that less than 50% of the VOCs in latex paint are emitted in the first year. Compounds studied include ethylene glycol, propylene glycol, Texanol®, and butoxyethoxyethanol. Alkyd paints contain substantially more VOCs than latex paints due to the use of mineral spirits as the solvent. Compounds emitted from alkyd paints include alkanes such as decane, nonane, octane, undecane, and aromatic compounds such as xylenes, toluene, and ethylbenzene (Guo *et al.*, 1999).

It is becoming increasingly apparent that VOCs emitted into indoor air can undergo chemical reactions. Morrison and Nazaroff (2002) studied the reaction of ozone with VOCs emitted by carpet to produce aldehydes. Of particular interest was the production of 2-nonenal, a compound with a low odor threshold. Investigators estimated that 2-nonenal could be generated from the carpet emissions at rates leading to odor detection for longer than three years.

VOC Emissions from Consumer Products

Consumer products such as cleaning products, personal care products, art supplies, and hobby supplies can release pollutants to the indoor environment and cause high personal exposures to pollutants. Wallace *et al.* (1989) measured the impact of activities and product use on personal exposure to VOCs, several of which are carcinogens. Breath levels of VOCs were often significantly correlated with previous personal exposures. Use of consumer products was associated with a variety of increased exposures as follows: use of deodorizers (*p*-dichlorobenzene), washing clothes and dishes (chloroform), visiting a dry cleaners (1,1,1-trichloroethane, tetrachloroethylene), smoking (benzene, styrene), cleaning a car engine (xylenes, ethylbenzene, tetrachloroethylene), and painting and using paint remover (n-decane, n-undecane).

During the late 1980s and early 1990s, the U.S. EPA conducted an indoor air quality research program to develop test protocols, determine emission factors of products, and develop emission models (Tichenor, 1989). During this time much was learned about emission profiles, sink effects, and the VOCs emitted by various consumer products. A list of organic compounds was identified in products such as paint, stain, adhesive, furniture polish, and caulk (Tichenor, 1989; Tichenor and Mason, 1988). These early studies were instrumental in understanding the role consumer products play in indoor air quality. However, changes in product formulations make newer studies more appropriate for understanding current indoor air quality impacts.

In an unpublished study, Akland and Whitaker (2000) found six products that they estimated would exceed the Proposition 65 no-significant-risk level for formaldehyde, including a nail finish, make-up, floor-cleaning spray, formica laminate, foaming tire care, and sheet flooring adhesive. Two products exceeded the no-significant-risk level for benzene, one product exceeded the level for acetaldehyde, and another product exceeded the level for tetrachloroethylene. The authors state "Comparison with the California levels would be reasonable only to the extent that the emission testing conditions represent typical indoor conditions, and that the product emissions actually resulted in exposure to the person for a 24-hour period". Products were applied to a glass substrate, so emissions may be different than would be expected in a typical situation.

Cleaning products have been studied to assess their impact on personal exposure in indoor environments. Zhu *et al.* (2001) tested 13 cleaning products to assess the potential for human exposure to three glycol ethers. 2-Butoxyethanol (BE) was present in the head space samples of seven products, of which five were household cleaning agents. BE concentration in the products ranged from 0.50 to 3.72%. 2-Methoxyethanol (ME), 2-ethoxyetyhanol (EE) were not detected; both of these are more toxic than BE. Investigators calculated average daily inhalation exposure levels for an individual cleaning with these products at home. Calculations were based on product use scenarios developed by U.S. EPA and a "standard room". For two all-purpose spray cleaners the average daily inhalation exposure for 2-butoxyethanol was 0.075 and 0.186 mg/kg body weight/day, and for two spray glass cleaners it was 0.004 and 0.006 mg/kg body weight/day. The high-end exposure of 0.186 mg/kg body wt/day for a 71 kg person would be 13.2 mg/day. This is below the U.S. EPA's inhalation reference concentration (RfC) for BE of 13 mg/m^3, the daily inhalation exposure that is likely to be associated with an appreciable risk of deleterious effects during a lifetime (U.S. EPA, 1999c). (To compare product emissions to the RfC, multiply the RfC by 20 m^3/day [daily breathing rate] to yield 260 mg/day, which is notably greater than the concentrations measured during the cleaning protocols.)

Other investigators also have studied emissions from consumer products and personal care products. Cooper *et al.* (1995) identified ethanol, α-pinene, camphene, β-pinene, diethylene glycol monoethyl ether, benzaldehyde and others as components of fragrance in two colognes, a perfume, a soap, and an air freshener. Toxicological data indicate the compounds may be irritants, mutagenic, teratogenic, or carcinogenic; however, risk of these health effects may be low given typical use of these products.

The impact of human occupancy and activities on VOC concentrations was documented in a study conducted by Shields *et al.* (1996). Investigators measured VOC concentrations indoors and outdoors at 70 offices having ranges in occupant density. The results showed that VOC concentrations were associated with the density of occupancy and ventilation rate. The authors were able to identify six compounds associated with occupant density: limonene used in cleaning products and air fresheners; tetrachloroethylene from dry-cleaned clothes; n-dodecane through n-hexadecane, probably from cosmetics, hand lotions, and shaving creams; and octa- and deca-methylcyclopentasiloxanes, which are associated with underarm deodorants and antiperspirants. It is important to note that the compounds associated with personal care products were present at low concentrations and they are not TACs or on the Proposition 65 list. The database generated by the study can be used to identify atypical compounds or concentrations in office buildings.

2.3.2.3 *Indoor Concentrations of VOCs*

Several major studies of concentrations of selected toxic pollutants in California residences have been conducted under the sponsorship of the U.S. EPA and/or ARB. Studies of VOCs were conducted over different seasons in three different years (1984, 1987, and 1990) in a total of nearly 500 California homes (Wallace, 1991; Wallace *et al.*, 1988; Sheldon *et al.*, 1992a). Analyses of the results across these and other studies indicate that indoor VOC concentrations are typically two to five times higher, and sometimes many times higher, than outdoor air (Wallace, 1991; Wallace *et al.*, 1988; Sheldon *et al.*, 1992a; Ott and Roberts, 1998). Results also indicate that personal (actual) exposure levels are generally higher than either indoor or outdoor residential levels. These results were consistent across different seasons and different geographic locations.

California Indoor VOC Studies

The average and 90th percentile personal, indoor, and outdoor concentrations of several VOCs measured in a northern California study are presented in Table 2.7 (Sheldon *et al.*, 1992a). Personal levels are usually higher than indoor and outdoor levels due to people's frequent use of, or proximity to, sources such as consumer products as they go about their daily activities. Table 2.7 includes a risk level for chemicals listed on the Prop 65 list. At the risk level presented, one excess cancer case per 100,000 individuals may be expected over a 70-year lifetime. The table indicates that a substantial portion of the population could be exposed to levels of *para*-dichlorobenzene, benzene, and perchloroethylene that pose an unacceptable excess cancer risk.

Table 2.7. Personal, Indoor, and Outdoor VOC Concentrations From a Northern California Study[1] (µg/m^3; means and 90th percentile)

Compound	Cancer Risk Level[2]	Personal Concentration		Indoor Concentration		Outdoor Concentration	
		mean	90	mean	90	mean	90
para-dichlorobenzene	1.0	21	88	18	36	0.30	0.94
Benzene	0.65	5	8.9	4.7	8.3	1.2	1.9
Perchloroethylene	0.7	1.6	3.0	1.1	2.3	0.53	0.59
Trichloroethylene	4.0	2.3	3.4	0.68	2.0	NQ[3]	NQ
Styrene	Not listed	2.4	3.3	2.8	3.9	0.24	0.70
1,1,1-Trichloroethane	Not listed	22	36	6.5	11	1.5	1.9
m,p-xylene	Not listed	9.3	18	6.3	13	1.8	2.9

1. From Sheldon *et al.*, 1992a.
2. Air concentrations that should not be exceeded to meet Proposition 65. For Prop 65, the "no significant risk" level is defined as 1 excess case of cancer per 100,000 individuals exposed over a 70-year lifetime.
3. NQ = not quantifiable: below the method quantifiable limit.

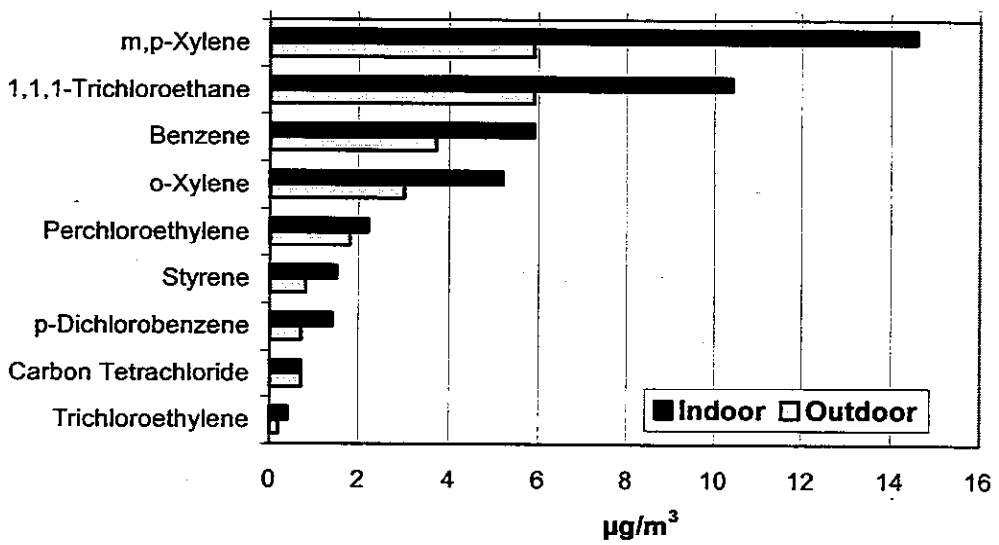
Another example of California VOC indoor and outdoor concentrations is presented in Figure 2.5. Figure 2.5 summarizes the median levels (levels at which half of the homes are higher and half lower) of indoor and outdoor concentrations of selected VOCs using combined data from one of the California VOC TEAM studies (February and July, 1987 in Los Angeles; Pellizzari *et al.*, 1989) and a study conducted in Woodland, California that was similar to the TEAM studies

(Sheldon *et al.*, 1992a). Figure 2.5 reflects the presence of indoor sources for all chemicals shown except for carbon tetrachloride, for which there are no longer indoor sources because of product restrictions imposed at the national level (CPSC, 1987b). Benzene concentrations measured in Texas homes during 1993 (Mukerjee *et al.*, 1997a,b) were comparable to levels measured in the California studies. It is important to note that the indoor concentrations presented in Figure 2.5 are less than the corresponding chronic REL for each given pollutant by a factor of six, or greater.

Trends

Some spatial and temporal trends in VOC levels (not illustrated in the figure) also have been noted: indoor concentrations in southern California appear to be somewhat higher than indoor levels in northern California, and indoor levels in winter tend to be higher than indoor levels in summer (Jenkins *et al.*, 1992b).

Figure 2.5:
Statewide Indoor and Outdoor Concentrations of VOCs
(medians, µg/m³)



Some additional trends found in indoor air VOC studies were identified by Brown *et al.* (1994) in their review of studies conducted between 1978 and 1990. The review encompasses measurements from residences, offices, schools and hospitals. Significant findings include:

- For all compounds, indoor concentrations were greater than outdoor concentrations by a factor of two to 73, indicating indoor sources were present. Mean VOC and TVOC concentrations were generally greater in established residences than established public buildings.
- New buildings had considerably higher TVOC concentrations than established buildings. For example, the weighted-average geometric mean TVOC concentration from 33 new residences was 4,500 µg/m³, whereas it was 1,130 µg/m³ from 1,081 established buildings.

- VOCs specific to new buildings included 2-ethoxyethylacetate, n-butanol, α-pinene, undecane, dodecane, tridecane, tetradecane, and 2-propanol.

Apparent Changes in Concentrations Over Time

Levels of some VOCs in California homes, such as benzene, have ~~most likely~~ decreased since these studies were conducted, due to changes in some building materials and consumer products, and the reduced smoking rates in homes. Some studies from other locations appear to support this, although others indicate continued levels of concern.

In a review of 12 studies conducted in North America. Hodgson and Levin (2003b) analyzed residential VOC concentration data collected from 1990 through 2003. They hypothesized that the 1990 Clean Air Act Amendments impacted indoor VOC concentrations through reduced infiltration of ambient pollutants and through industry changes in consumer products and other materials used indoors to use less toxic compounds. Hodgson and Levin found 1,1,1-trichloroethane concentrations to be more than three times lower than the TEAM study value. Benzene, 1,2-dichloroethane and tetrachloroethene were approximately one-half an order of magnitude lower in the current review. The authors noted that the data were limited in a number of aspects. They also highlight that "despite these trends, indoor exposures to most common VOCs undoubtedly still dominate human exposures to these compounds". In another study, VOC concentrations for five out of six VOCs measured recently in Baltimore, Maryland residences (Payne-Sturges *et al.*, 2004) were somewhat lower than levels shown in Figure 2.5. However, there were differences in sample collection methods (passive samplers in Baltimore, active samplers in California) and undoubtedly differences in housing characteristics based on region. The focus of the Baltimore study was to estimate cancer risk. Although concentrations are lower than measurements taken from studies conducted during the 1980s, the authors estimate a cumulative indoor cancer risk of 120 per million based on the median indoor concentrations.

Recent studies in other states continue to show higher levels indoors than outdoors, and indoor levels above health risk guidelines. For example, results from the recent Arizona National Human Exposure Assessment Survey (NHEXAS) indicate that indoor levels of pollutants continue to be greater than outdoor levels, and some are comparable to levels measured in the initial TEAM studies (Gordon *et al.*, 1999). In another recent study conducted in Minneapolis/St. Paul, investigators confirmed the continued risk due to elevated indoor concentrations of VOCs (Sexton *et al.*, 2004). Pollutant concentrations were compared to risk thresholds established in Minnesota. Median indoor concentrations for benzene and chloroform exceeded the level for a 70-year lifetime cancer risk of 1 in 100,000. The 90th percentile concentrations exceeded the cancer risk level for benzene, chloroform, and *p*-dichlorobenzene. The identified pattern of personal VOC concentrations exceeding indoor VOC concentrations, which in turn exceed outdoor VOC concentrations, also was reaffirmed by this study. This relationship held for 13 of the 15 VOCs measured by Sexton *et al.* in Minneapolis/St. Paul over three seasons in 1999.

Impacts of Attached Garages on Indoor VOC Concentrations

Several studies have investigated the impact that attached garages have on residential indoor levels of VOCs. These studies implicate vehicles as well as other activities (such as storage of chemicals and small engines) within attached garages as the sources of these VOCs. Adgate *et al.* (2004) measured outdoor, indoor, and personal VOC exposures to children in 284 households in Minnesota for ten VOCs. Households with attached garages had significantly higher concentrations of benzene, chloroform, styrene, and *m/p*- and *o*-xylene compared to households without an attached garage. Thomas *et al.* (1993) investigated the temporal variability of benzene exposures for residents in several New Jersey homes with attached

garages or tobacco smoke. In homes with attached garages or ETS, mean indoor and personal benzene concentrations were two to five times higher than outdoor levels at all but one home. Measured benzene levels in four garages ranged from 3-196 μg/m3; garage source strength estimates ranged from 310-52,000 μg/hr. The mass transfer rate for benzene from sources in the garage to living areas ranged from 730-26,000 μg/hr. Material and activities inside the garage were the sources of the benzene.

Several studies have explicitly studied VOC emissions from vehicles in attached garages. Gammage *et al.* (1984) determined that high levels of VOCs indoors in two houses were associated with the operation of a car in a basement garage. Tsai *et al.* (2000) also found elevated indoor concentrations of benzene, toluene, and methanol resulting from automobile emissions released in an attached garage. In a detailed Canadian study, Graham *et al.* (2004) measured the concentration of 175 VOCs (non-methane hydrocarbons and halogenated compounds) inside 16 residences over two seasons while a vehicle in the attached garage was operated under brief cold-start and hot-soak conditions. Positive net changes of indoor VOC concentrations were observed whose signatures were similar to those of the vehicle profile releasing the emissions. Chemical Mass Balance (CMB) modeling suggested that the infiltration of vehicle emissions into the house could account for between 9 and 85% of the measured hydrocarbon concentrations in the house during the test period.

In a study of indoor air samples collected from 137 Anchorage homes between December 1994 and February 1996, the Anchorage Air Pollution Control Agency determined that the presence of an attached garage was the factor most strongly associated with in-home benzene; indoor elevated benzene levels were strongly related to the use of a garage to park vehicles or store fuel or small engines. Other VOCs (toluene, ethylbenzene, *m/p*-xylene, *o*-xylene) were also higher in homes with attached garages; geometric mean concentrations were two to four times higher in homes without attached garages. A regression model for benzene in homes with attached garages revealed a significant impact of a car parked in the garage (vs. no car), number of trips originating from the attached garage, fuel having been opened within the last three days, presence of forced air furnaces (vs. hot water boilers), and living areas above the garage (vs. adjacent to the garage) (*Architectural, Behavioral and Environmental Factors Associated with VOCs in Anchorage Homes,* 1998).

The primary reason that attached garages contribute to elevated indoor levels of CO and VOCs is due to air leakage into the residence through the garage-to-residence interface. Air leakage rates between attached garages and homes have been measured in several different northern climate locales (i.e., Canada, Minnesota) throughout the last decade, with garage-to-residence leakage/infiltration rates ranging from 9% to 17% (*Air Infiltration from Attached Garages in Canadian Houses* 2001, *Evaluation of Homes Built to the New Minnesota Energy Code* 2002, *Garage Performance Testing* 2004). Garage-to-residence infiltration rates may differ in California from these reported values to climatic differences as well as variations in regional building codes.

Several mitigation options may be useful in reducing leakage (Built Green of Colorado, 2004), including aerodynamically decoupling the garage from the house, building ductwork 10 times tighter than the current standard, ventilating garages with either active or passive vents, equipping every home with a CO sensor, testing a subset of houses in a given area to ensure efforts are working, and informing homeowners (Built Green of Colorado, 2004).

VOCs in Public and Commercial Buildings

Girman *et al.* (1999) identified VOCs in public and private buildings, but did not associate the presence of VOCs with a particular source. Girman analyzed data collected at 56 office buildings (most with no known indoor air quality problems) across the U.S. Of the 48 VOCs identified in indoor air, eight were found in all of the samples, another 26 were found in 81-99% of the samples. Indoor concentrations of 27 VOCs were at least twice the corresponding outdoor concentrations. The 12 VOCs with the highest median indoor concentrations were acetone, toluene, *d*-limonene, *m*- and *p*-xylenes, 2-butoxyethanol, n-undecane; benzene, 1,1,1-trichloroethane, n-dodecane, hexanal, nonanal, and n-hexane. Only acetone (29 $\mu g/m^3$) was present at a concentration above 10 $\mu g/m^3$. The study was designed to provide baseline data on VOCs in U.S. office buildings relative to complaint buildings. The author did not compare indoor concentrations to guideline levels; however, levels were below available minimum risk levels.

2.3.3 Environmental Tobacco Smoke

Environmental tobacco smoke (ETS) is the complex mixture of thousands of gaseous and particulate compounds emitted from the burning end of cigarettes and cigars, and in smokers' exhaled breath. ETS is also called "second-hand smoke", "passive smoke", and "involuntary smoking". Many chemicals in ETS have been identified as toxic air contaminants, and not surprisingly, ETS is estimated to cause thousands of deaths due to cancer and cardiac disease, and many thousand asthma episodes and other illnesses (ARB/OEHHA, 2004/2005). Cigarettes are the most common source of ETS, but cigars and pipes are also sources.

Passage of a statewide smoke-free workplace law in California in 1995 (AB 13, Labor Code 6404.5) eliminated smoking in nearly all California indoor workplaces–including restaurants, bars and gaming clubs. In 1999, 93.4% of California's indoor workers reported working in a smoke-free environment, compared to only 45% in 1990 (Gilpin *et al.*, 2001). Additionally, the percentage of Californians with children under the age of 18 who do not allow smoking in the household has increased substantially. In 1994, 63% of Californians with children did not allow smoking in the house; by 2001, 78% did not allow it (Gilpin *et al.*, 2001). Because of such laws and trends, California has lower ETS exposures than most other states.

2.3.3.1 Health Effects of ETS

Exposure to ETS has been linked with a variety of adverse health effects, including heart disease, asthma episodes, other respiratory illness, and lung cancer (OEHHA, 1997). Primary tobacco smoke is an established human carcinogen for smokers (IARC, 2002; U.S. DHHS, 1989). ETS has been identified as a cause of lung cancer in nonsmokers (U.S. DHHS, 1986; U.S. EPA, 1992; IARC, 2002). More recently, there is evidence from analyses of epidemiological studies that ETS exposure has a causal association with breast cancer (ARB/OEHHA, 2004/2005).

Table 2.8, below, provides the current estimates of the health impacts of ETS on Californians and Americans every year, taken from the ARB/OEHHA draft report for the identification of ETS as a toxic air contaminant, which is currently in its last stage of scientific review (ARB/OEHHA, 2004/2005). In addition to the large cardiac and lung impacts on adults, ETS has a number of serious impacts on children's health. These include exacerbation of asthma, increased respiratory tract infections, increased middle ear infections, low birth weight, sudden infant death syndrome (SIDs), and developmental impacts (ARB/OEHHA, 2004/2005). Children under 18 months of age have greater tendency to suffer from bronchitis or pneumonia when their parents are smokers. A small reduction in birth weight has been repeatedly observed with

mothers who are active smokers. Studies have shown fairly consistently that maternal smoking during pregnancy is adversely associated with measures of cognition and behavior in children (NCI, 1999). There is epidemiological evidence of a causal relationship between maternal smoking in general and risk of SIDS.

Table 2.8 Current Estimated Attributable Risks Associated with ETS
(from ARB/OEHHA, 2005)

OUTCOME	EXCESS # IN CA PER YR	EXCESS # IN U.S. PER YR
Pregnancy: **Low Birth Weight** **Pre-term Delivery**	1,600 4,700	24,300[1] 71,900
Cardiac death (Ischemic heart disease death)	1,700-5,500[2]	22,700[3] - 69,600
Lung Cancer Death	400 [4]	3400
Asthma episodes (children)	> 31,000 [5]	202,300 [6]
Otitis media visits	51,700 [7]	789,700 [8]
SIDS	21[9]	431[10]
Breast cancer	All studies: OR 1.26 (95% CI 1.10-1.45)[11] Best studies: OR 1.90 (95% CI 1.53-2.37) Approximate 26-90% increased risk	

[1] Based on adult females reporting exposure to ETS in NHANES III for 1995 (Pirkle et al., 1996)
[2] Based on California Dept Health Services. www.dhs.cs.gov/hisp/chs/OHIR/vssdata/2000data/OOCh5pdf/5_9_Reorg.PDF. Table 5-9 for yr 2000
[3] Based on Anderson and Arias (2003). National Vital Statistics Report. Vol 51(9) Table 2 for yr 2000 Ischemic heart diseases including AMI.
[4] Assuming California exposure and death rates are similar to national rates and California population is 12% of national population.
[5] Based on number asthma attacks or episodes in previous 12 months for 0-17 year olds. Calculated from California Health Interview Survey for 2001
[6] Based on number asthma attacks or episodes in previous 12 months for 0-14 year olds. CDC-MMWR 2002 51(SS01)
[7] Calculated by applying national value (H6) and assuming 12% of US population lives in California
[8] Based on National Center for Health Statistics Series 13 No. 137. Ambulatory Health Care Visits by Children: Principal Diagnosis and Place of Visit for yrs 1993-1995.
[9] Based on California Dept Health Services. www.dhs.ca.gov/hisp/chs/ohir/vssdata/2000data/00ch4pdf/8reorg.pdf. Table 4-8 for yr 2000
[10] Based on National Center for Health Statistics. www.cdc.gov/nchs/fastats/infort.htm for yr 2000 LBW = low birth weight; N/A = data not available.
[11] OEHHA is unable at this time to calculate an attributable risk as it is not possible to account accurately for the portion attributable to other known risk factors.

ETS affects many tissues and organs of the body. ETS is causally associated with coronary heart disease in smokers and nonsmokers (NCI, 1999). Research suggests that chronic ETS exposure may increase the risk of stroke by about 82% (Bonita *et al.*, 1999). ETS is estimated to cause about 400 excess deaths due to lung cancer each year in California (ARB/OEHHA, 2004/2005). In reviewing the literature on asthma and indoor air, the Institute of Medicine (2000) found that:

> "There is sufficient evidence to conclude that there is an association between ETS exposure and the development of asthma in younger children. [However], there is inadequate or insufficient evidence to determine whether an association exists between ETS exposure and the development of asthma in school-aged or older children, or in adults. There is sufficient evidence to conclude that there is a causal relationship between ETS exposure and exacerbations of asthma in preschool-aged children. There is limited or suggestive evidence of an association between chronic ETS exposure and exacerbations of asthma in older children and adults. Limited or suggestive evidence of an association between acute ETS exposure and exacerbations also exists for asthmatics sensitive to this exposure".

Eye and nasal irritation are the most commonly reported symptoms among adult nonsmokers exposed to ETS (ARB/OEHHA, 2004/2005).

Nazaroff and Singer (2004) estimated mean daily exposure to and inhalation intake of specific hazardous air pollutants found in ETS for non-smokers who live with smokers. The approach used a model based on cigarette consumption patterns, emission factors, residence characteristics, and population statistics. Results indicate "the ratio of estimated average exposure concentrations to reference concentrations is close to or greater than one for acrolein, acetaldehyde, 1,3-butadiene, and formaldehyde, indicating potential for concern regarding noncancer health effects from chronic exposure. In addition, lifetime cancer risks from residential ETS exposure are estimated to be substantial (~2 to 500 per million) for each of five known or probable human carcinogens: acetaldehyde, formaldehyde, benzene, acrylonitrile, and 1,3-butadiene."

2.3.3.2 ETS Sources

The variability in emissions between brands of cigarettes is relatively low, though emissions from cigars and cigarettes vary in magnitude. Daisey *et al.* (1998) conducted a chamber study testing six of the most popular commercial brands in California and one reference cigarette for emissions of 21 different air toxics and other airborne compounds, including VOCs, nicotine, aldehydes, and airborne particulate matter (estimated to be PM2.5). Diluted sidestream smoke (produced by a smoking machine that smoked three cigarettes sequentially) was used to approximate ETS aging in a room-sized chamber, and a mass-balance model was used to generate estimates of indoor concentrations. Among the VOCs, acetaldehyde and formaldehyde displayed the highest emission factors (average emission factors 3,340 ng/mg tobacco and 2,040 ng/mg tobacco, respectively), and PM showed an emission factor of 12,400 ng/mg. The nicotine emission factor was 1,410 ng/mg tobacco. These results suggest that ETS has a substantial influence on indoor concentrations of these compounds.

Chuang *et al.*, (1999) investigated children's exposure to polycyclic aromatic hydrocarbons (PAHs) in low-income rural and inner-city areas in North Carolina. Indoor/outdoor air samplers and real-time PAH monitors were used to obtain measurements. The researchers determined that potentially carcinogenic PAH concentrations were significantly higher in smokers' homes than in non-smoking homes (geometric mean: 6.14 ng/m^3 vs. 1.38 ng/m^3, respectively). Additionally, the authors discovered that children in both rural and inner-city homes received higher potential doses of PAHs than adults, in part due to their lower body weights.

2.3.3.3 ETS Concentrations

Restrictions on smoking in California from the late 1980s to mid 1990s in workplaces and in public locations such as restaurants, bars, and gaming clubs have led to a reduction in smoking in indoor environments in California, with commensurate reductions in indoor concentrations of ETS and non-smokers' exposure levels. A number of studies published since 1996 have shown that ETS constituents are present at lower concentrations in public places following smoking bans than they were prior to the bans (Ott *et al.*, 1996; Hammond, 1999; Switzer *et al.*, 2001; Repace, 2003), and that levels can be considerably higher in smoking versus comparable nonsmoking areas (Glasgow *et al.*, 1998; Hammond, 1999; Graves *et al.*, 2000).

Despite California's workplace smoking ban, high indoor ETS concentrations still can be found in smokers' homes and in private vehicles (Ott *et al.*, 2003; Park *et al.*, 1998; Offermann *et al.*, 2002). Children's exposure to ETS is greatly impacted in these two environments when in the presence of a smoking parent or other adult. Children spend up to 85% of their time in the home (Wiley *et al.*, 1991a). Thus, the potential for exposure to ETS can be extremely high when smoking occurs in a child's home. As of 2001, 22% of smoking parents still allowed smoking inside the home (Gilpin *et al.*, 2001). Likewise, exposure in vehicles can be quite high due to the presence of a strong source in a relatively small volume of air. Recent residential respirable particulate matter (RSP, PM3.5) measurement is limited to a single study (Ott *et al.*, 2003). A level of 300 $\mu g/m^3$ was measured in the bedroom where one cigarette was smoked; 5,500 $\mu g/m^3$ was the maximum bedroom level when three cigarettes were smoked. RSP levels ranging from 92 $\mu g/m^3$ (with ventilation) to 1,195 $\mu g/m^3$ (without ventilation) have been measured inside a minivan (Offermann *et al.*, 2002).

Three comprehensive review documents summarize nicotine and RSP concentrations measured in smoking environments prior to 1996 (U.S. EPA, 1992; Guerin *et al.*, 1992; NCI, 1999). Comparison of mean nicotine concentrations from these earlier reviews with data published after 1995 reveals that the means have decreased in workplaces and restaurants, but less so in homes. In studies conducted before 1996, mean nicotine concentrations in offices and restaurants ranged from about 1 to 36 $\mu g/m^3$. In a more recent review, Hammond (1999) reported means of 2 to 8 $\mu g/m^3$ for these locations. Also, according to the Hammond review, nicotine levels were two- to six-times lower in indoor workplaces with smoking bans than in offices that allowed smoking (less than 1 $\mu g/m^3$ vs. 2-8 $\mu g/m^3$, respectively). It appears that nationally, as smoking has become a less accepted social behavior, individuals are not smoking in indoor public locations as much as they did a few years earlier (in California, smoking is prohibited in indoor workplaces).

Levels of RSP are generally comparable in both older and newer studies, but slightly lower in the newer studies (relative to 1996). Studies highlighted in the NCI review (1999) reported RSP concentrations consistent with other reviews. All measured levels tend to range from about 100 to 400 $\mu g/m^3$ in offices and restaurants that allow smoking. Switzer *et al.*, (2001) measured RSP levels at a church bingo site of 87 to 348 $\mu g/m^3$ above outdoor levels, and at less than 15 $\mu g/m^3$ when smoking was banned. Similarly, PM3.5 concentrations at a sports tavern in California were 56.8 $\mu g/m^3$ with smoking, and 5.9 – 12.9 $\mu g/m^3$ with smoking banned (Ott *et al.*, 1996). Similar to nicotine levels, these recent RSP data from smoking locations are somewhat lower than the pre-1996 data. RSP levels are much lower (<15 $\mu g/m^3$) in indoor locations where smoking is prohibited.

The majority of California homes never or rarely contain smokers, and would typically have a nicotine level lower than 0.5 $\mu g/m^3$. However, the recent body of data indicates that those who

choose to smoke in their home have remained consistent in their smoking patterns over the years. It is important to note that concentrations of lower volatility ETS components such as nicotine, 3-ethenylpridine, phenol, cresols, and naphthalene accumulate on surfaces to the extent that their re-emission (when a smoker is not present) is an important route of indirect exposure for non-smokers (Singer et al., 2003; Singer et al., 2002).

Table 2.9 summarizes current estimates of indoor levels of nicotine and RSP for California. A more detailed discussion of indoor ETS concentrations is provided in the Draft *Technical Support Document – Part A, Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant* (ARB/OEHHA, 2004/2005).

Table 2.9. Estimates of Current California Indoor Concentrations of Nicotine and Respirable Suspended Particulates (RSP)

Environment	Nicotine Concentration $\mu g/m^3$	RSP Concentration $\mu g/m^3$
Homes with smokers present	0.5 – low[1] 3.0 – medium 6.0 – high	300-5,500[3]
Offices/public buildings With smoking Smoking prohibited	 2 – 8[2] <1	 56.8 – 348[4] <15
Vehicles With ventilation Without ventilation	 NA NA	 ~100[5] ~1,200

1. Glasgow *et al.*, 1998
2. Hammond, 1999
3. Ott *et al.*, 2003
4. Ott *et al.*, 1996 and Switzer *et al.*, 2001
5. Offermann *et al.*, 2002

2.3.4 Biological Contaminants

Biological contaminants are substances of plant, animal, or microbial origin. They are naturally abundant in the outdoor and indoor environments, but are considered contaminants when found in undesired locations or at elevated concentrations. They include bacteria, viruses, and fungi; allergens from animal dander, pollen, fungi, and the fecal particles or body fragments from house dust mites and cockroaches; as well as chemicals emitted by mold and bacteria such as endotoxins and mycotoxins. Exposure to biological contaminants may cause a variety of health effects, including asthma, allergy, infection, irritation, and toxic responses. Building related illness, or BRI, is an illness for which the specific cause–usually a virus, bacteria, or fungi–can be identified within the building. Examples include Legionnaire's disease, caused by the *Legionella* bacterium, or humidifier fever.

2.3.4.1 Health Effects of Biological Contaminants

The health effects of biological contaminants can be grouped into three major categories:

- Communicable disease transmission: Many infectious diseases are transmitted from person to person in indoor air. For example, inhalation of viruses is associated with influenza, measles, and chicken pox. The primary mode of transmission for the common cold virus has

not been proven conclusively. Studies have shown transmission by both inhalation and direct or indirect contact between infected droplets from coughing or sneezing onto mucous membranes of the eyes, nose or mouth (Goldmann, 2002). Inhalation of bacteria is associated with tuberculosis and Legionnaire's disease. Epidemiological studies have often found significantly lower prevalence of respiratory illness or surrogates for respiratory illness (sick leave, total absence from school) in buildings with higher ventilation rates, reduced office sharing, and less crowding (Fisk, 2000; Myatt *et al.*, 2004; Shendell *et al.*, 2004). These and other studies indicate that a significant portion of common respiratory illness may be transmitted indoors by airborne particles; however, the proportion of total disease transmission that occurs through this route cannot be easily quantified.

- Hypersensitivity reactions: Many biological agents can provoke a hypersensitivity response in individuals who are genetically predisposed to developing allergic disorders. Allergic rhinitis (hay fever) and allergic asthma are the most common examples of hypersensitive responses to biological contaminants. Common symptoms and signs are watery eyes, runny nose, sneezing, nasal congestion, itching, coughing, wheezing, difficulty breathing, headache, and fatigue. Allergens from fungal spores, house dust mites, cockroaches, dog and cat dander, and pollen are frequently found indoors. When high concentrations of these allergens are present indoors they can trigger allergic responses or asthmatic exacerbation. Hypersensitivity pneumonitis is a rare immune-mediated lung disorder initially found in farmers and workers exposed to high doses of organic dusts in agricultural or industrial settings. A few cases of this disease have been attributed to indoor exposure to bacteria or mold growth. Only susceptible persons exposed to large amounts of these allergens are at risk of developing hypersensitivity pneumonitis (IOM, 2004).

- Toxic responses: Many individuals in persistently damp or moldy buildings report symptoms such as headache, memory difficulties, vomiting, diarrhea, and increased frequency of cold/flu illnesses that do not appear to be caused by allergic or infectious mechanisms. The causes of such symptoms have not been identified. Some researchers have postulated that exposure to biological toxins such as endotoxins (components of the outer membrane of Gram-negative bacteria) and mycotoxins (secondary metabolic products of some fungi) may induce such symptoms. However, the health impact of inhalation exposure to biological toxins in indoor environments is not well understood.

- More detailed information on the health effects of mold is discussed below in Section 2.3.4.3.

2.3.4.2 Sources of Biological Contaminants

Biological contaminants include allergens such as animal dander, house dust mites, cockroaches, and pollen; bacteria, viruses, and fungi; and chemicals emitted by mold and bacteria such as endotoxins and mycotoxins.

Animal Dander

Dogs and cats are kept as pets in 32 and 27% of U.S. households, respectively (AVMA, 1997). Allergy to cats is reported to be about twice as common as allergy to dogs. Dog and cat allergens are found on small particles that can remain airborne for prolonged periods. The particles also adhere readily to fabrics such as clothing, upholstered furniture, and carpet and can easily be carried from animal–owning homes into offices, schools and day care centers (Custovic *et al.*, 1998). In a recent nationally representative study, both dog and cat allergens were detected in all U.S. homes (Arbes, 2004). Asthmatics who are sensitive to cat allergen

may experience allergic symptoms (congestion, runny nose, itching), asthma exacerbation and compromised lung function (chest tightness or wheezing) at cat allergen levels typically found in schools and homes without cats (Bollinger *et al.*, 1996).

House Dust Mites

House dust mites are microscopic relatives of spiders that feed on human skin cells and other organic material. Primary mite allergens are concentrated in their fecal particles and body fragments. These allergens are very potent, but concentrations in indoor air typically are low because the allergens are attached to larger particles (at least 10 μm) that settle rapidly. Consequently dust mite allergens are found predominantly in carpets, pillows, bedding, and upholstered furniture. In locations where humidity is high for most of the year, dust mites produce larger quantities of allergen (Rosenstreich *et al.*, 1997). In contrast, mites cannot survive in desert or mountain (5,000 feet elevation or higher) regions where indoor humidity is routinely low. In a recent study of U.S. homes, mite allergens were found in all beds tested. 46 percent of these homes had mite allergen at levels previously associated with allergic sensitization, while 24% had levels associated with an increased risk for asthma attacks in asthmatics allergic to dust mites (Arbes *et al.*, 2003).

Cockroaches

While many insects have been implicated as sources of inhaled allergens in small indoor studies, cockroaches are the only insects that have routinely been identified as a common source of indoor allergens (IOM, 2000). The exact part of the cockroach responsible for its allergens is unknown but may involve fecal particles and body parts. Like house dust mites, cockroach allergen is associated with particles larger than 5 μm and these particles become airborne only when settled dust is disturbed. Cockroach activity frequently occurs in indoor areas where food or standing water is available, such as kitchens and bathrooms. Cockroach hypersensitivity is highest among the urban poor, but the complex interrelationship between race, poverty and residence has been difficult to resolve (IOM, 2000). Exposure to cockroach allergen has been associated with almost a two-fold increased risk of wheeze in infants less than one year old (Belanger *et al.*, 2003). In a recent study of southern California children, asthma diagnosis before five years of age was associated with exposures in the first year of life to cockroaches and other environmental agents (Salam *et al.*, 2003).

Endotoxins

Endotoxins are components of the outer membranes of Gram-negative bacterial cells. These bacteria occur naturally on plants and are abundant in soil and in the human intestinal tract. Endotoxins are released when these bacteria die or their cell membranes are damaged; thus, these toxins are always found in the outdoor environment. High-level exposures to airborne endotoxins in agricultural and waste-disposal industries contribute to acute and chronic bronchitis that may lead to decreased lung function (Vogelzang *et al.*, 1998). In indoor environments, lower airborne endotoxin concentrations have been associated with both adverse health effects (increased asthma symptoms and medication use) and protective health effects (decreased prevalence of allergy) (IOM, 2004). Many factors appear to interact to modulate health effects associated with endotoxin exposure (Song *et al.*, 2003).

Viruses

Viruses are the smallest and simplest infectious agents, unable to survive well outside their plant, animal or human host. In outdoor air, viral survival depends on season, moisture content and temperature of the air, wind conditions, sunlight and presence of atmospheric pollutants (Cox, 1995). In the indoor environment, the infectivity of airborne viruses is affected by factors such as relative humidity and room temperature (Otten *et al.*, 1999). Most viral infections are

spread from person to person in the indoor environment. Rarely a virus such as Hantavirus (also known as Sin Nombre or Four Corners virus), endemic in some populations of deer mice in the Southwestern United States, can be transmitted to humans. Indoor air exposure to this virus can occur when housecleaning activities disturb dust or nests resulting in aerosolized mouse saliva or excreta (Mills, 2002).

Damp buildings

Moisture is common in buildings, with most studies reporting signs of dampness in at least 20% of homes examined (IOM, 2004). Moisture problems originate from rainwater, groundwater, plumbing, construction, water use by occupants and condensation of water vapor. Although some moisture is present in all buildings, excessive dampness is more likely to occur in buildings that are older, lack central heating, are poorly insulated and overcrowded (IOM, 2004). The assessment of building dampness is complicated by the absence of a generally accepted definition of "dampness" or what constitutes a "dampness problem".

A recent report by the National Academy Institute of Medicine, entitled *Damp Indoor Spaces and Health* (IOM, 2004), reviewed the scientific literature regarding indoor dampness and its relationship to the various health outcomes that have been attributed to damp or moldy indoor environments. Key findings from this report are shown in Table 2.10. The report concludes that although many details require clarification through future research, the currently available scientific evidence is sufficient to regard excessive indoor dampness as a health threat to building occupants.

A review of the medical literature has shown that occupants of damp buildings are twice as likely to experience coughing, wheezing and asthma attacks as those in dry buildings (Bornehag *et al.*, 2001). The underlying causes and mechanisms of these illnesses are not completely understood. Some studies have associated occupant health problems with fungal or bacterial growth on building materials such as drywall and carpeting (Husman, 1996; Verhoeff and Burge, 1997; Peat *et al.*, 1998). However, building dampness is also known to increase emission of volatile organic compounds (VOCs) from decomposition of flooring materials, even without microbial growth (Pasanen *et al.*, 1998). Damp concrete floors are known to increase the rate of chemical degradation of polyvinyl chloride floor coatings and glues, resulting in emissions of ammonia and other VOCs into the indoor air (Gustavsson and Lundgren, 1997; Wiglusz *et al.*, 1998; Tuomainen *et al.*, 2004). Damp buildings also encourage the growth and allergen production capacity of cockroaches, house dust mites and other arthropods, as well as the survival of respiratory viruses.

2.3.4.3 Indoor Mold

The remainder of this section discusses indoor mold in detail, because of its increasing occurrence at problem levels in indoor environments.

Molds are simple, microscopic organisms, present virtually everywhere, indoors and outdoors. Molds, along with mushrooms and yeasts, are fungi, which play a critical role in nature by breaking down dead plant and animal matter and recycling nutrients in the environment. For molds to grow and reproduce, they need only moisture and a food source – organic plant material, such as leaves, wood or paper or animal products such as leather. Because molds grow by digesting organic material, they gradually destroy whatever they grow on. Visible mold growth on cloth or building materials or furnishings, sometimes referred to as "mildew", often has a wooly or cottony appearance that is frequently green, gray, brown, or black but may also

be white or a range of other colors. Many molds that can grow indoors release countless tiny, lightweight spores that travel easily through the air.

Health Effects of Mold

In recent years, media attention to indoor mold has surged, leading to rising concern about mold-related health effects. Because mold spores are ubiquitous in air or dust, everyone contacts them on a daily basis, usually without evident harm. However, persons who are allergic to mold and those with compromised immune systems may develop serious health problems from exposure to routine types and amounts of indoor mold.

Table 2.10. Health Outcomes and Indoor Dampness[1]

Sufficient Evidence of a Causal Relationship • None (the evidence was not sufficient to link dampness as a clear cause of any health outcome)
Sufficient Evidence of an Association • Upper respiratory tract (nasal and throat) symptoms • Asthma symptoms in sensitized asthmatic persons • Wheeze • Cough
Limited or Suggestive Evidence of an Association • Shortness of breath • Lower respiratory illness in otherwise healthy children • Asthma development
Inadequate or Insufficient Evidence to Determine Whether an Association Exists • Airflow obstruction in otherwise-healthy persons • Skin symptoms • Chronic obstructive pulmonary disease • Mucous membrane irritation syndrome • Inhalation fevers (non-occupational exposures) • Lower respiratory illness in otherwise healthy adults • Rheumatologic and other immune diseases • Acute idiopathic pulmonary hemorrhage in infants • Gastrointestinal tract problems • Fatigue • Neuropsychiatric symptoms • Cancer • Reproductive effects

1. Not applicable to immunocompromised persons, who are at increased risk for fungal infections.
Source: IOM, 2004

Fungi produce a very large number of allergens, with each fungus potentially producing several different allergenic compounds depending on its growth pattern and environmental conditions. More than 80 fungal species have been associated with allergic diseases (Day *et al.*, 2001). Unfortunately, the limited number of standardized materials available to allergists to test patients for mold allergies complicates our ability to determine the frequency of mold allergy in U.S. or California populations. Humans are known to potentially encounter approximately 200 different species of fungi outdoors and perhaps 50 species indoors. Typical symptoms that mold-allergic persons report (alone or in combination) include: respiratory problems, such as wheezing, difficulty breathing and shortness of breath; nasal and sinus congestion; eye irritation (burning, watery or reddened eyes); dry, hacking cough; nose or throat irritation; and skin rashes. In rare instances exposures to fungi may illicit more intense immunological responses such as allergic bronchopulmonary aspergillosis and allergic fungal sinusitis. These conditions involve fungal colonization of the airways or sinuses (Storey *et al.*, 2004).

Serious lung infections from a few fungal groups such as *Aspergillus* and *Fusarium* species are common in persons being treated with high-dose cancer chemotherapy, recent solid-organ transplant recipients or those who are otherwise immunocompromised (Summerbell, 2001). Children in damp or moldy buildings sometimes report having more respiratory infections, including colds and ear infections (Rylander, 2000). Some investigators have suggested that this increase is due to an immunosuppressive effect of exposure to indoor fungal growth (Johanning *et al.*, 1996). While some fungi have been shown to cause immune suppression in experiments with laboratory animals, it is not yet clear if damp or moldy building exposures can cause significant changes in the human immune system.

Organic dust toxic syndrome is a general term used in reference to illnesses related to inhalation of bacterial endotoxins or fungal toxins, typically resulting from occupational exposures to bioaerosols. The symptoms are similar to those of hypersensitivity pneumonitis (Storey *et al.*, 2004). One other possible health outcome from exposure to mycotoxins is pulmonary hemorrhage (Storey *et al.*, 2004).

In studies of health effects associated with indoor fungal exposure, some people without allergies report respiratory and other symptoms similar to those experienced by mold-allergic individuals. In addition, occupants of moldy buildings have reported some health outcomes that are not usually associated with allergy (such as memory loss, depression, fatigue, mood swings, and "hemorrhage in the mucous membranes of the intestinal and respiratory tracts") (IOM, 2004). Investigators are exploring whether these effects are associated with exposure to one or more fungal constituents such as:

- Fungal toxins – chemicals known to be produced under certain fungal growth conditions by more than 400 fungal species and capable of producing a toxic response in animals or other microbes.
- Some structural components of fungi – for example, glucans (chemicals that make up all fungal cell walls) may affect the activity of immune cells in the lung.
- Microbial volatile organic compounds – gaseous substances that account for the odors identified when mold is growing indoors. While often associated with respiratory symptoms in damp buildings, the specific contribution of these compounds to health complaints is still unclear.

Key findings regarding indoor mold growth and health outcomes from the National Academy Institute of Medicine report, *Damp Indoor Spaces and Health* (IOM, 2004), are summarized in Table 2.11. The report concludes that although more research is needed to define the role of molds, mycotoxins and other fungal components, as well as the potential for synergistic

interaction between molds and other microbial or chemical agents in damp buildings, the currently available scientific evidence is sufficient to conclude that indoor mold is associated with upper respiratory symptoms, cough, wheeze, asthma symptoms in sensitized asthmatic individuals and, rarely, hypersensitivity pneumonitis in susceptible persons.

Table 2.11. Presence of Indoor Mold and Health Outcomes[1]

Sufficient Evidence of Causal Relationship • None (the evidence was not sufficient to link mold as a clear cause of any health outcome)
Sufficient Evidence of an Association • Upper respiratory tract (nasal and throat) symptoms • Asthma symptoms in sensitized asthmatic persons • Wheeze • Cough • Hypersensitivity pneumonitis (a rare immune-mediated lung condition) in susceptible persons[2]
Limited or Suggestive Evidence of an Association • Lower respiratory illness in otherwise healthy children
Inadequate or Insufficient Evidence to Determine Whether an Association Exists • Shortness of breath • Airflow obstruction in otherwise healthy persons • Skin symptoms • Asthma development • Chronic obstructive pulmonary disease • Mucous membrane irritation syndrome • Inhalation fevers (non-occupational exposures) • Lower respiratory illness in otherwise healthy adults • Rheumatologic and other immune diseases • Acute idiopathic pulmonary hemorrhage in infants • Gastrointestinal tract problems • Fatigue • Neuropsychiatric symptoms • Cancer • Reproductive effects

1. Not applicable to immunocompromised persons, who are at increased risk for fungal infections.
2. For mold or bacteria in damp indoor environments.

Source: IOM, 2004

Mold Concentrations

It is common in indoor air quality investigations to find mold spores in the air and dust inside homes, with most of these originating outdoors. Many studies have tried to differentiate

buildings with problem mold growth from non-moldy buildings by evaluating the type and concentration of indoor airborne fungal spores. The concentrations and types of mold spores in indoor air typically are directly related to those in outdoor air. If there is a serious mold problem in a building, the indoor types and concentrations of mold may, or may not, differ from those outdoors at the time of sampling. Numerous studies have found that spore concentrations vary widely in both outdoor and indoor air. Outdoor concentrations vary with geographical location, climate, season, relative humidity, wind direction and types of vegetation in the immediate area of the sampling device (IOM, 2004). Outdoor airborne mold spore concentrations may reach levels as high as 10,000 spores/m^3 (Mullins, 2001). Indoor mold spore concentrations are usually lower than corresponding outdoor levels, but are also quite variable, ranging from 0-10,000 colony forming units/m^3 (Shelton *et al.*, 2002).

Different types of molds are identified and their concentrations measured either by directly examining a sample under a microscope or by culturing the spores and allowing them to grow into colonies that are then counted. There are many reliable methods for collecting and analyzing fungi although no single method can identify all the fungi present in environmental samples (AIHA, 1996; ACGIH, 1999). Thus, different sample collection and analysis techniques often lead to different fungal count or concentration estimates. No standard method of mold identification or spore counting has been proven effective in a wide range of building applications or is mandated for environmental assessment by any federal or California state government agency.

In a recent review of studies aimed at identifying buildings with mold problems, an expert panel of the Institute of Medicine concluded "...fungal counts alone provide little information about the microbial status of an indoor environment" (IOM, 2004). Currently, government and professional industry groups recommend that building investigations for mold include a thorough visual inspection of the premises, documentation of visibly moldy areas, and the use of professional judgment in determining whether mold sampling is appropriate for that particular investigation (U.S. EPA, 2001b; AIHA, 1996; ACGIH, 1999; Miller, 2001).

2.3.5 Pesticides

Pesticides can be naturally occurring or synthetic chemicals designed to control and kill insects, weeds, and disease carrying organisms in the home and surrounding landscaping. Ninety% of American homes use pesticides (Gurunathan *et al.*, 1998). Schools commonly apply pesticides in or around the classroom (Addiss *et al.*, 1999; Kaplan *et al.*, 1998; Volberg *et al.*, 1993). Pesticides can be tracked in on clothing or drift in from outdoors, later becoming resuspended in air and accumulating in dust.

2.3.5.1 Health Effects of Pesticides

Pesticides differ in their levels of toxicity and mechanisms of action. Some classes of insecticides are highly toxic, especially to the nervous system. Pesticides with relatively low acute toxicity are not necessarily safe, as they may have the potential to cause cancer or other chronic effects. Other health effects of pesticide exposure include irritation of the skin and eyes, and hormone or endocrine disruption.

Two classes of widely used insecticides in the U.S. are the organophosphates and pyrethroids; both are neurotoxins. Pesticide workers have experienced nausea, headaches, dizziness, and general weakness after exposure to agricultural organophosphates. Typical indoor air concentrations of organophosphates previously approved for home use (i.e., chlorpyrifos and

diazinon) generally do not result in these symptoms. As a class, pyrethroids are less toxic than organophosphates. Their neurotoxic effects have not been reported for humans exposed to typical levels in the home. Pesticides are often measurable in house dust and carpet dust; levels of contamination are discussed later in this section. The effects of pesticides on children are a particular concern because their behavior can lead to greater exposure than to an adult. Children spend time on the floor where they contact dust that may contain pesticides. The hand-to-mouth behavior of young children may lead to ingesting pesticides. Furthermore, research has shown that low-level chronic exposure to organophosphates can adversely affect children's developing nervous systems (Eskenazi *et al.,* 1999); chronic exposure to pyrethroids has been linked to possible hormonal disruption (Landrigan *et al.*, 1999). There is insufficient evidence to determine if pesticides cause or exacerbate asthma (IOM, 2000).

2.3.5.2 Sources of Pesticides

Chlorpyrifos and diazinon, two organophosphate insecticides, were the most widely used pesticide ingredients in common household ant and roach killers and lawn-care sprays (U.S. EPA, 2001a; U.S. EPA, 2000d). The U.S. EPA banned the indoor use of chlorpyrifos and diazinon for non-agricultural settings in December 2000 and March 2001, respectively (U.S. EPA, 2001a; U.S. EPA, 2000d). Chlorpyrifos is persistent in the environment and has been observed in many indoor house dust samples since it was banned.

Other pesticides that are persistent in the environment and have been banned for some time include, DDT, and Dieldrin. DDE is a breakdown product of DDT, a widely used insecticide that was banned in 1972 (U.S. EPA, 2000f). Environmental sources of DDE include soil, atmospheric dispersion, sediment runoff, contaminated plants and animals, and improper use and disposal. Dieldrin was widely used from 1950 to 1974 to control insects on cotton, corn and citrus crops (U.S. EPA, 2000g). Also, dieldrin was used to preserve wood, control termites, and control locusts and mosquitoes. Most uses of dieldrin were banned in 1987. Environmental sources of dieldrin include soil surrounding wooden structures treated for termites; soil or sediment; improper use or disposal; contaminated fish and shellfish; and contaminated dairy products and meat.
Since the ban on indoor use of chlorpyrifos, a class of insecticides called pyrethroids has been used as substitutes for chlorpyrifos and other organophosphate pesticides. Permethrin, the most common pyrethroid, acts on a broad spectrum of insects, and is less persistent than chlorpyrifos in dust and soils.

2.3.5.3 Pesticide Concentrations

To date, only one published study has examined indoor pesticide concentrations in California homes. In September 1992, Bradman *et al.* (1997) measured pesticide levels in house dust samples from five farmworker homes and five non-farmworker homes in an agricultural area south of Fresno. This study was conducted before the ban on indoor use of chlorpyrifos and diazinon. However, it cannot be determined if the indoor levels were from infiltration of outdoor air and soil track-in, or from the use of indoor pesticide. Of the ten pesticides detected in house dust, only chlorpyrifos and diazinon levels frequently exceeded 1 µg/g of dust in the homes. In general, farmworker homes had higher levels of chlorpyrifos and diazinon than non-farmworker homes. The highest detectable level of pesticide in non-farmworker homes was 2.5 µg/g of diazinon, and the highest in farmworker homes was 169 µg/g of diazinon.

During 2001-2002 (after the ban on indoor use of chlorpyrifos and diazinon), ARB and DHS funded a comprehensive statewide study of the environmental health conditions in portable

(relocatable) classrooms. As part of the study, investigators summarized floor-dust pesticide concentration data for twenty different pesticides. Chlorpyrifos, *cis*- and *trans*-permethrin, *ortho*-phenylphenol, piperonyl butoxide (PBO), and esfenvalerate were detected in over 80% of the classrooms. Excluding ortho-phenylphenol, these pesticides measured 95th percentiles above 1 µg/g. Portable classrooms did not differ significantly from traditional classrooms in their mean pesticide concentrations (Whitmore *et al.*, 2003).

The pesticides detected in California and their concentrations are similar to those found in other studies in other areas of the U.S. A summary of indoor and personal air concentrations and house dust levels is presented in Table 2.12. The following paragraphs provide a brief discussion of some of the table content.

The Non-Occupational Pesticide Exposure Study (NOPES) examined inhalation exposures for 32 different pesticides in 208 residences in Jacksonville, Florida, and 101 residences in Springfield/Chicopee, Massachusetts, over three different seasons from 1986 to 1988 (Whitmore *et al.*, 1994). Seven pesticides – chlordane, chlorpyrifos, diazinon, dichlorvos, heptachlor, *ortho*-phenylphenol, and propoxur – had the highest mean concentrations across seasons in Jacksonville for both indoor and personal air. Mean concentrations of all detectable pesticides captured in carpet ranged from 0.01 µg/g to 15.4 µg/g. The mean concentrations for chlorpyrifos and diazinon found captured in the carpet were 5.8 µg/g and 1.7 µg/g, respectively.

During the 1992 spray season, Simcox *et al.* (1995) vacuum-sampled house dust for four organophosphorus (OP) insecticides, including chlorpyrifos, in 59 homes in Wenatchee, Washington (48 agricultural, 11 non-farming families). All four compounds were detected in 62% of households. Pesticide levels were significantly lower in non-farming homes than in agricultural homes (Table 2.12). OP levels ranged from non-detectable to 17 µg/g in agricultural homes and non-detectable to 0.82 µg/g in non-farming homes. Two-thirds of the agricultural homes contained pesticide levels above 1 µg/g for at least one OP compound.

During May to September 1997, the Minnesota Children's Pesticide Exposure Study (MNCPES) characterized indoor and personal air levels for children 3 to 12 years old in 102 homes in Minneapolis/St. Paul, Minnesota (Clayton *et al.*, 2003). Although the median values for indoor air were slightly higher than personal air (Table 2.11), there was a highly significant correlation between personal air and indoor air samples for the organophosphates chlorpyrifos (0.81), malathion (0.51), and diazinon (0.62). Air levels for chlorpyrifos and diazinon were measurable for >90% and >65% of the participants, respectively. Permethrin was also detectable in air with a median concentration of <0.2 ng/m^3.

In the National Human Exposure Assessment Survey (NHEXAS) performed in Arizona, Gordon *et al.* (1999) measured residential levels of chlorpyrifos and diazinon. Chlorpyrifos was found in dust more often than diazinon (88% vs. 53%), and had nearly the same occurrence in indoor air as diazinon (65% vs. 63%). There was a small association between chlorpyrifos in dust and air (Pearson = 0.096, Spearman = 0.773). Although the NHEXAS results are generally comparable to those from other studies, Gordon *et al.* (1999) found a higher maximum level for pesticides in residential samples than previously reported.

In a recent study, Rudel *et al.* (2003) analyzed 39 air and 38 dust samples (collected by vacuum cleaner) for pesticides in 120 homes in Cape Cod, Massachusetts. Of the 23 pesticides detected in air and 27 detected in dust, chlorpyrifos was one of the most abundant with 90th percentile concentrations of 12 ng/m^3 in air and 1.87 µg/g in dust. Although not as pervasive in dust, diazinon had one of the highest 90th percentile concentrations in air (9.0 ng/m^3). *Cis*- and *trans*-permethrin and PBO were also detected at relatively high levels in dust (7.04 µg/g, 16.5 µg/g, and 15.1 µg/g, respectively).

Table 2.12. Comparison of Indoor-air, Personal-air, and House-dust Levels in U.S.

Location	Season	Ranges and median and mean values					
		Chlorpyrifos			Diazinon		
		Range	Median	Mean	Range	Median	Mean
Indoor air		***Concentration (ng/m³)***					
Minneapolis, MN[a]	All	NL[b]	1.742	NL	NL	0.29	NL
NHEXAS Arizona[c]	All	<3.2-3280	8	NL	<2.1-20,500	4.6	NL
Brownsville, TX[d]	Spring	2.5-115	7.6	NL	1.6-60	1.4	NL
	Summer	5.7-67	24	NL	2.5-78	3.5	NL
Jacksonville, FL[e]	Summer	<2.5-2170	182	366.6	15-13,700	73	420.7
	Spring	NL	NL	205.4	NL	NL	109.2
	Winter	<2.5-1043	69	120.3	30-1080	21	85.7
Springfield, MA[e]	Spring	<4.5-252	<4.5	9.8	<22-1810	<22	48.4
	Winter	<3.5-291	<3.5	5.1	<28-28	<28	2.5
Raleigh, NC[f]	Fall	NL	NL	0.08	NA[g]	NA	NA
New Jersey area[h]	Fall	151.2-154.2	NL	NL	5.7	NL	NL
Cape Cod, MA[i]	All	<1-92	<1	NL	<1-550	<1	NL
Personal air		***Concentration (ng/m³)***					
Minneapolis, MN	All	NL	1.577	NL	NL	0.275	NL
NHEXAS Arizona	All	<19-175	NL	NL	<15	NL	NL
Jacksonville, FL	Summer	NL	NL	280.4	NL	NL	321.6
	Spring	NL	NL	182.8	NL	NL	112.7
	Winter	NL	NL	118.2	NL	NL	89
Springfield, MA	Spring	NL	NL	7.5	NL	NL	10.1
	Winter	NL	NL	5.9	NL	NL	1.4
House dust		***Concentration (μg/g)***					
Fresno, CA[i]	Fall[j]	0.2-33	NL	NL	0.7-169	NL	NL
	Fall[k]	<1	NL	NL	0.2-2.5	NL	NL
NHEXAS Arizona	All	<0.004-119	0.16	NL	<0.020-66	0.13	NL
Brownsville, TX	Spring	0.1-1.7	0.3	NL	0.1-1.8	0.06	NL
	Summer	0.2-1.7	0.56	NL	0.1-0.8	0.07	NL
Wenatchee, WA[l]	Spring[j]	<0.02-3.6	0.27	0.43	NA	NA	NA
	Spring[k]	<0.02-0.5	0.05	0.17	NA	NA	NA
Jacksonville, FL	Winter	NL	4.7	5.8	NL	0.4	1.7
Raleigh, NC	Fall	NA	NA	1.6	NA	NA	NA
New Jersey area[h]	Fall	0.53-15	NL	NL	0.08-0.74	NL	NL
PCS[m]	All	NL	0.308	0.607	NL	0.035	0.358
Cape Cod, MA[i]	All	<0.2-228	<0.2	NL	<0.2-51.0	<0.2	NL

Source: Modified from Gordon *et al.*, 1999.

[a]Clayton *et al.*, 2003.

[b]NL, not listed.

[c]Gordon *et al.*, 1999.

[d]Mukerjee *et al.*, 1997b.

[e]Whitmore *et al.*, 1994.

[f]Lewis *et al.*, 1994.

[g]NA, not applicable.

[h]Roinestad *et al.*, 1993.

[i]Rudel *et al.*, 2003.

[j]Bradman *et al.*, 1997.

[k]Samples from agricultural family homes.

[l]Samples from non-agricultural family homes.

[m]Simcox *et al.*, 1995.

[n]Whitmore *et al.*, 2003.

2.3.6 Lead

Lead is a toxic metal that has been used in paints, gasoline, pipes, and ceramic glazes, and to a lesser extent, in caulk. Lead paint chips and soil contaminated with lead poses a major hazard to children when they ingest them. Over the long term, exposure to lead can lead to brain damage, decreased growth, hyperactivity, impaired hearing, and reproductive effects.

2.3.6.1 Health Effects of Lead

The body mistakes lead for calcium and concentrates it in the bones, where it can leach into the blood. Both pre-natal and post-natal exposure, and exposure during childhood results in toxicity to the nervous system (Thacker *et al.*, 1992; Needleman *et al.*, 1979). Children, especially those younger than five, are more susceptible to lead than adults. Chronic, low-dose exposure to lead, often occurring in early childhood, can lead to decreased learning ability due to brain damage. Acute health effects, seldom seen, include seizures, paralysis, anemia, abdominal pain, constipation, vomiting, and decreased appetite.

Lead not only concentrates in bones, it also accumulates in the liver and kidneys. Lead has a half-life of 25 to 40 days in the blood and organs, and a half-life of 25 years in the bones. During pregnancy, lead mobilized from the bones and released into the bloodstream can pass through the umbilical cord and adversely affect the developing fetus. The blood lead level of concern for children, established by the U.S. Centers for Disease Control and Prevention in 1991, is 10 µg/dL (CDC, 1997). Significant decreases in IQ, behavioral problems, and learning impairment have been documented at blood-lead levels above this threshold. Recent research showing health effects at levels below 10 µg/dL now supports the growing consensus that there is no safe level for lead exposure.

2.3.6.2 Sources of Lead

In the U.S., lead was eliminated from paints and gasoline in the late 1970s. Today, major sources of lead include old paint in homes built before 1978, lead pipes placed before the 1930s, lead solder in copper piping installed up to 1986, and soil by busy roads. Activities that disturb lead-based paint, such as remodeling or paint removal, can release large amounts of lead-bearing particles into the air, which may later settle in dust. Even without remodeling, deteriorating lead paint can accumulate in house dust. Often overlooked sources of lead include home remedies, cosmetics, hobby materials, and foreign-made, vinyl mini-blinds (ATSDR, 1999a; CPSC, 1996).

Next to resuspension of lead dust from lead-based paint, much of the lead present in indoor air appears to result from the infiltration of lead particles in outdoor air. Infiltrated and tracked-in lead dust, brought into the home from the workplace, can accumulate in carpets that can serve as a reservoir for lead-laden dust (U.S. EPA, 1997a). Research has shown that lead-dust loadings and concentrations per unit mass of dust are correlated with blood-lead levels, but no causal effect can be inferred from this association. Children's mouthing behaviors and activities that put them in direct contact with lead-contaminated surfaces increase their probability for exposure to lead by ingestion.

Candles containing a lead/metal core in the wick can emit lead into the air when burned. Van Alphen (1999) calculated the mean rate of lead release from seven candles was 770 µg/hour. In homes where candles are burnt once per week for several hours, lead air levels would be sufficient to drive the child's blood lead levels over the CDC level of concern of 10 µg/dl.

2.3.6.3 Lead Concentrations

In a 1997 report, the U.S. EPA summarized findings from a literature review on the amount of lead in dust within carpets, furniture, and forced air ducts. A major drawback across studies was the inconsistency in the methods used to collect and analyze lead. In general, the average loadings are lower for furniture than for floors, window components, walls, and air ducts. Furniture, including upholstery and window treatments, has an averaging loading of less than 100 μg/ft^2. The average loadings for forced air ducts exceed 100 μg/ft^2, and can be as high as 1,000,000 μg/ft^2.

Dust lead levels at schools are an important factor when determining children's total exposure to lead. In a recent comprehensive study of the environmental health conditions in California portable (relocatable) classrooms, ARB and DHS summarized floor-dust metal concentration data for 18 different metals (Whitmore *et al.*, 2003). All the elements, except copper, had higher dust loading in portable classrooms than traditional classrooms, though the differences were not statistically significant. As stated in the report: "Since the portable classrooms are generally newer, the lower concentration of lead may reflect the number of years' accumulation of the particles in the classroom." Table 2.13 reports the median and 95th percentile concentrations for lead in the floor dust of all classrooms sampled.

Table 2.13. California Portable Classrooms Study Results for Lead in Floor Dust

Element	Room Type	Concentrations (μg/g)		Loadings (ng/cm^2)	
		Median	95th Percentile	Median	95th Percentile
Lead	All	61.6	189.5	6.6	58.4
	Portable	57.5	151.6	5.8	57.9
	Traditional	66.8	200.6	7.1	57.5

Between 1994 and 1998, DHS surveyed the lead content in paint and soil for a random sample of 200 California public elementary schools and child care facilities (DHS, 1998). Results were used to make predictions about the lead content in all of California's 5,000 schools. The study found: "As in California's housing, lead-containing paint is estimated to be in most California public elementary schools and child care facilities." DHS estimated that one-third of schools contain deteriorating lead paint, and nearly 6% have soil that is contaminated with lead exceeding U.S. EPA Lead in Soil Guidelines. The highest levels of lead in soil were found close to school buildings and near buildings built prior to 1940.

2.3.7 Mercury

There are three types of mercury: elemental (or metallic), inorganic (or mercury salts), and organic. In the past, inorganic mercury was added to skin-lightening creams and medicinal products. Today, compounds containing inorganic mercury are used as fungicides and antibacterials (ATSDR, 1999b). Metallic mercury can be found in thermometers, electric switches, fluorescent lights, thermostats, and other products. The primary source of organic mercury, specifically methylmercury, is fish and other types of seafood.

2.3.7.1 Health Effects of Mercury

Workers exposed to mercury levels ranging from 25 to 60 μg/m^3 have experienced tremors, changes in personality, loss of sensation and muscle coordination, vision and hearing

impairments, deficits in cognitive function, respiratory tract irritation, severe stomach comfort, skin rashes, and elevations in blood pressure and heart rate (OEHHA, 2000c). In combination with data from other published research, Carpi and Chen (2001) estimated that 10% of U.S. homes may have indoor mercury levels that exceed the U.S. EPA Reference Concentration of 0.3 $\mu g/m^3$. Exceeding the U.S. EPA Reference Concentration may increase the likelihood of health impacts, but it might take years for the effects to be seen and they may not occur at all. Typical mercury exposures in indoor environments are not likely to produce developmental neurotoxicity.

2.3.7.2 Sources of Mercury

Indoor air is the second most common route of exposure to mercury in the general U.S. population (fish consumption is first) (NJ Mercury Task Force, 2002; Carpi and Chen, 2001; WHO, 1990). Mercury is an effective fungicide and bactericide, so it has been used as an intentional additive in many household products, including latex paints (interior use banned in 1990), contact lens solutions, and nasal sprays (ATSDR, 1999b). Mercury may also be found as an accidental contaminant in detergents and cleansers due to its extensive use in the chlor-alkali industry (U.S. EPA, 1999b). Elemental mercury's unique physical and chemical properties are the basis for its use in many household items. It expands uniformly with increases in temperature, does not cling to glass, and is liquid and can vaporize at room temperature. Mercury workers are exposed to greater levels of elemental mercury than the general population. Workers' families are also at increased risk; workers may bring home clothing and shoes contaminated with mercury in the workplace (ATSDR, 1999b). Elemental mercury is also used in some cultural practices, either carried as a good luck charm, or sprinkled in cars and homes or over infants (ATSDR, 1999b). With the removal of mercury in paint and the ban on the sale of mercury thermometers in California (SB633, 2002), exposures to mercury in indoor air in California should decrease over time. Table 2.14 lists the sources of mercury in homes.

Table 2.14. Sources of Mercury in the Residential Environment

Type of Mercury	Sources
Metallic	Thermometers, electric switches, fluorescent lights, thermostats, barometers, batteries, ethnic remedies, spiritual practices
Inorganic and organic	Latex paints, medicines, disinfectants, detergents, antiseptics

2.3.7.3 Mercury Concentrations

There is limited information on airborne exposures in the non-occupational environment. From June 2000 through March 2001, Carpi and Chen (2001) measured mercury concentrations in indoor air for nine residences and three businesses in the New York metropolitan area. Nearly all of the sites showed higher indoor levels than outdoor levels. The average indoor mercury concentration for all buildings was 69 ng/m^3, and was highly dependent on season.

Mercury is found in many household items, but unless the mercury-containing device is broken or disturbed, mercury does not pollute the air. Once released, however, mercury persists in the indoor environment for months or years after its first release, especially if the spill is not properly cleaned (ATSDR, 1999b). According to the New Jersey Mercury Task Force (2002): "Exposure to Hg^0 (such as in certain cultural practices) can be significant with respect to health effects. As

little as one drop (0.05 ml) of liquid Hg^0 in a sealed bedroom-sized room (assuming a room volume of about 33 m^3 and no air exchange) can result in an air concentration equal to the U.S. EPA Reference Concentration."

Several nationwide incidences of non-occupational exposure to mercury have been documented. The Ohio Department of Health, in cooperation with the federal ATSDR, analyzed the indoor mercury concentrations in nine school gymnasiums (ATSDR, 2002). The highest detectable level of mercury in indoor air was 1.6 $\mu g/m^3$. A *Morbidity and Mortality Weekly Report* summarized findings from the contamination of homes and schools with Hg^0 in Palm Beach County, Florida (CDC, 1995). The report illustrated how quickly mercury can spread and how difficult it is to contain. The local authorities evacuated 50 homes, and closed schools until mercury levels dropped below 10 $\mu g/m^3$. Seventeen homes contained vapor concentrations greater than 15 $\mu g/m^3$. Table 2.15 shows the guideline levels for mercury.

Table 2.15. Recommended Airborne Concentrations of Mercury

Values	Mercury Vapor
ATSDR Minimum Risk Level (MRL)	0.2 $\mu g/m^3$
ATSDR recommended screening level	1 $\mu g/m^3$
ATSDR action level	10 $\mu g/m^3$
U.S. EPA reference concentration (RfC)	0.3 $\mu g/m^3$

2.3.8 Polycyclic Aromatic Hydrocarbons

Polycyclic aromatic hydrocarbons (PAHs) are a complex class of semi-volatile organic compounds and consist of two or more fused benzene rings. The larger PAH species are associated with fine or ultrafine particles; humans become exposed to a complex mixture of PAHs, not individual compounds (IARC, 1983; U.S. EPA, 1987c; Atkinson and Arey, 1989). Benzo(a)pyrene, a PAH with five fused benzene rings, is often considered to be the representative compound for PAHs (U.S. EPA, 1987c; IARC, 1983). PAHs are produced from incomplete combustion; indoor sources are primarily tobacco smoking, aerosols from hot cooking oil, woodburning, and infiltration of outdoor air.

2.3.8.1 Health Effects of PAHs

Several individual PAHs are believed to be carcinogenic to humans. The U.S. Environmental Protection Agency (U.S. EPA) classifies benzo(a)pyrene as a possible human carcinogen; the International Agency for Research on Cancer (IARC) classifies benzo(a)pyrene as a probable human carcinogen (IARC, 1983; U.S. EPA, 1984). Other PAHs characterized by either IARC or U.S. EPA as possible human carcinogens include: chrysene, certain benzofluoranthenes, and indeno(1,2,3-cd)pyrene (IARC, 1983; U.S. EPA, 1984, ARB/OEHHA, 1994; ARB, 1994a). For most of the PAH species, there is currently insufficient evidence to classify these compounds as to their carcinogenicity.

2.3.8.2 Sources of PAHs

Tobacco smoking, when present, was identified as the strongest indoor source of benzo(a)pyrene and other PAHs indoors in a study conducted in California (Sheldon *et al.*, 1993; Sheldon *et al.*, 1992b). Investigators have measured as many as 56 PAH species and derivatives in tobacco smoke; hundreds more may exist (Vu-Duc and Huynh, 1989; Wynder and Hoffman, 1964).

Cooking activities were found to be the greatest contributor to PAH concentrations, second to infiltration of traffic -polluted outdoor air in a small study conducted by Dubowski *et al.* (1999). These investigators also determined candle- and incense burning contribute to indoor levels of PAHs. Siegman and Sattler (1996) determined that PAH concentrations in the fumes from hot cooking oil ranged from 1.08 to 22.8 $\mu g/m^3$, compared to PAH concentrations found in a road tunnel with heavy traffic (2.6 $\mu g/m^3$) and an office with heavy cigarette smoking (1.2 $\mu g/m^3$). However, they caution that the oil droplets may not have the same carcinogenic potential as PAH from combustion aerosols.

Woodburning appliances contribute to indoor PAH levels. Fireplaces can emit soot and PAHs directly into the indoor air environment (Traynor *et al.*, 1987). While newer, more efficient airtight stoves appear to emit less than older, leakier woodstove models, poor maintenance and certain practices, such as operating the woodstove with the door open, can raise indoor air PAH levels substantially (Traynor *et al.*, 1987).

Transport of outdoor air into the home can introduce PAHs from outdoor sources such as traffic, diesel engines, power plants and agricultural burning (IARC, 1983; U.S. EPA, 1987c; Dubowski, 1999).

2.3.8.3 PAH Concentrations

The Air Resources Board funded two large studies to characterize indoor exposures to PAHs in California residences. In a northern California study, investigators measured PAHs inside 280 homes during the winter of 1992, evaluating the relationship between PAH levels and common indoor combustion sources (Sheldon *et al.*, 1993). Investigators reported average concentrations of 2.2 ng/m^3 of benzo(a)pyrene in smoker's homes, but only 0.83 ng/m^3 in nonsmoker's homes. Investigators reported that cigarette smoking significantly raised levels of 12 of 13 PAH species when compared to levels in nonsmokers' homes.

In the same study, fireplace and woodstove use raised average benzo(a)pyrene levels to about twice the levels found in homes with no obvious combustion sources. Researchers reported average benzo(a)pyrene concentrations of 1.2 ng/m^3 compared to 0.83 ng/m^3 measured in "no source" homes. The benzo(a)pyrene level of 0.83 ng/m^3 was primarily attributed to infiltration of smoke from community woodburning, a value well above the typical average outdoor levels in California (Atkinson and Arey, 1989).

The second ARB-funded study was conducted in Southern California homes. In this study, Sheldon *et al.* (1992b) found polluted outdoor air to be a major source of indoor PAHs when other combustion sources were absent in the home. Other investigators have reported similar findings (Waldman *et al.*, 1989; Lioy and Greenberg, 1990; Naumova *et al.*, 2002). In two Los Angeles area communities, Naumova *et al.* (2002) measured PAHs in homes located near busy traffic areas and reported significantly lower levels of particle-bound PAHs indoors than outdoors. Concentrations were dominated by coronene, an indicator of motor vehicle-related pollution. Investigators stated that these data suggested that indoor concentrations were driven by outdoor PAH pollutant levels. Cigarette smoking was also a major contributor to indoor PAH levels in the study of southern California homes (Sheldon *et al.*, 1992b). In smokers' homes, investigators reported statistically significant increased concentrations relative to nonsmoking homes of nine PAH species, including benzo(a)pyrene during the daytime, and six PAH species during the night.

Investigators in other states have reported substantial increases in benzo(a)pyrene indoors as a result of tobacco smoking in the home (Kanarek *et al.*, 1985; Chuang *et al.*, 1988; Turk *et al.*, 1987). In a study of PAH levels in 15 public buildings in the Pacific Northwest, Turk (1987) reported average levels of benzo(a)pyrene of 1.07 ng/m^3 smoking areas compared to 0.39 ng/m^3 in nonsmoking areas. The mean indoor-to-outdoor ratio was 7.6.

2.3.9 Radon

Radon is a naturally-occurring radioactive gas derived from the decay of radium-226 (a decay product of uranium-238) present in small amounts in some soils, rocks, and water. Radon itself is relatively harmless, but its decay products, called radon daughters or radon progeny, can accumulate in the lung and cause cancer. A statewide survey (Liu *et al.*, 1991) indicates that only 0.8% of California residences have annual radon levels above 4.0 pCi/l, U.S. EPA's recommended mitigation level, and the statewide radon average is about 1 pCi/l. However, due to the potency of radon daughters, a preliminary estimate based on an extrapolation from national data indicates that radon contributes to about 1500 excess cancer deaths per year in California. There is substantial uncertainty surrounding this estimate, however, in part due to its inseparability from the risk from exposure to tobacco smoke, and as explained further below.

2.3.9.1 Health Effects of Radon

Causal associations between exposure to radon and it progeny and lung cancer have been demonstrated in many epidemiological studies of underground miners. The National Research Council (NRC) Committee on the Biological Effects of Ionizing Radiations has updated estimates of radon potency and lung cancer deaths over a number of years, at times increasing or decreasing the estimated potency or deaths (NRC, 1988; NRC, 1990; NRC, 1999a). In the most recent assessment, NRC (1999a) estimated excess lung cancer deaths in the U.S. due to residential radon exposure using two different models. The results range from 15,400 to 21,800 deaths, for the age-duration and age-concentration risk models, respectively. The U.S. EPA (2003c) modified the NRC models and reported 21,000 excess lung cancer deaths (90% confidence interval or CI: 9,000 to 50,000) due to residential radon exposure nationally. Concurrent exposure to radon and smoking show synergistic effects, that is, the risk of lung cancer is higher than predicted by adding the individual risks (NRC, 1999a). It is difficult, if not impossible, to separate lung cancer risk from ETS and that from radon (NRC, 1999a).

To provide a rough estimate of the risk from radon in California for the purposes of this report, the first-order estimation of radon-induced lung cancer deaths in California was derived using simple linear extrapolation by population number, smoking prevalence, and average indoor radon concentrations. The national estimates (21,000 deaths/year) were multiplied by the fraction of the U.S. population in California (34 million out of 281 million, or 12%; U.S. Census 2000 data); the ratio of average radon concentrations (CA: 1 pCi/l; U.S.: 1.25 pCi/l; ratio of 0.8); and the smoking prevalence in California (CA: 16.4%; U.S.: 22.5%, ratio of 0.73; CDC, 2004). This preliminary calculation yields an estimate of about 1,500 (90% CI 600-2,100) excess cancer deaths per year in California attributable to radon exposure.

However, this estimate is likely to be an over-estimate, for several reasons. First, a recent study in the Sierra foothills region found that the percentage of households exceeding 4 pCi/l is far less than the percentage found for that region in the previous statewide survey (3% vs. 25%), so the actual statewide radon average may be notably lower than the previous estimate of 1 pCi/l (Tsai and Waldman, 2005). Additionally, current indoor radon levels may be lower than the measurements taken 15 years ago because new construction may be more radon resistant.

These factors would lead to a lower statewide radon average; thus the risk estimate presented here may be an overestimate, and should be viewed as preliminary. Finally, as discussed in an earlier section, smoking rates and exposure to secondhand smoke have decreased substantially in California as a result of state regulations and public education efforts. This may reduce the opportunity for synergistic effects of radon and tobacco smoke to occur, resulting in fewer cases of cancer.

Although the toxicity of radon is relatively certain, based on strong epidemiological studies, many uncertainties associated with the estimation of risk in California remain to be explored. Detailed information for region-specific residential radon exposures, in combination with demographic data (e.g., age, gender) and smoking status (current and previous smoking; non-smoking) would be required to perform a more accurate radon risk assessment for California, but such an effort is beyond the scope of this report. Additionally, a more refined estimate may not be useful, for several reasons. A statewide solution in a state with such low average levels of radon is not useful for addressing radon. The most effective approach to reducing risk from radon is to avoid tobacco smoke (Mendez *et al.*, 1998; NRC, 1999a), and California has aggressive state programs to promote reductions in smoking. Local building codes to prevent radon intrusion in regions with elevated radon are another preventive measure that could provide some protection. However, following the simplified estimation approach above, even if the statewide indoor radon average were reduced to the ambient level, 0.5 pCi/l, about 750 (300-1,050) lung cancer deaths would still be expected to occur per year due to natural radiation.

While the majority of radon risk is associated with inhalation exposure from air, naturally occurring radon in water also poses a risk. About 168 cancer deaths per year are estimated to occur in the U.S. from radon in drinking water – 89% from lung cancer caused by breathing radon released from water (e.g., showering or flushing the toilet), and 11% from stomach cancer caused by drinking radon-containing water (NRC, 1999b).

2.3.9.2 Sources of Radon

Indoor radon derives from various sources in the vicinity of a building: (a) release from soils underneath the building (generally the most important source), (b) building materials enriched with uranium-238 (e.g., granite or concrete blocks used in the foundation or walls), (c) use of radon-enriched local well water (especially for bathing and appliance use), and (d) outdoor air. Elevated indoor radon levels are generally due to the combination of a rich source, a driving force into the building, and a relatively low dilution rate for fresh air (Eichholz, 1987).

2.3.9.3 Radon Concentrations

Samplers are available to test indoor radon levels at reasonable cost. Measurement techniques include charcoal canisters, alpha track detectors, electret ion chamber detectors or scintillation flasks. Radon concentrations are reported as the number of radioactive decays per time in a volume of either air or water, or picoCuries per liter (pCi/l). The U.S. EPA has issued recommendations to reduce the health risks from radon exposure in homes. Immediate corrective action should be pursued when indoor air radon levels are above 200 pCi/l. For radon levels between 20 and 200 pCi/l, corrective action should be pursued within a few months. For radon levels between 4 and 20 pCi/l, corrective action should be pursued within a few years (U.S. EPA, 2002a).

A statewide survey in California found that only 0.8% of California residences have annual radon concentrations exceeding 4 pCi/l (Liu *et al.*, 1991). This study examined annual average radon concentrations in over 300 homes in a population-based survey and determined that the average radon level in California homes is 1 pCi/l. Areas with higher than average radon levels include the Central Valley, the Sierra Nevada mountains and foothills, the valleys east of the Sierra Nevada, and Ventura and Santa Barbara counties. Surveys of California public elementary schools demonstrated similar geographic patterns for areas with elevated indoor radon (Churchill and Youngs, 1993; Zhou *et al.*, 1998).

The National Residential Radon Survey was conducted by the U.S. EPA in 1989-90 to assess annual average radon concentrations in U.S. residences (Marcinowski *et al.*, 1994). The survey estimated an arithmetic annual average radon concentration in U.S. homes of 1.25 ± 0.12 pCi/L, and about 6.0% of homes had radon levels greater than the U.S. EPA action level of 4 pCi/l.

Homes with elevated levels of radon are more commonly found in geographical areas with higher natural levels of soil radioactivity (i.e., uranium). However, predicting residential radon concentrations is difficult due to factors associated with home construction and operation (Churchill, 1997). Nonetheless, building factors, such as ventilated crawl space, and age of the buildings may affect the indoor radon (Tsai and Waldman, 2005).

2.3.9.4 Activities and Policies to Address Radon in California

The Department of Health Services' (DHS) Environmental Management Branch, within the Division of Drinking Water & Environmental Management, manages the state's Radon Program. The Radon Program goals are to increase the public's awareness of the risks of radon in indoor air and reduce exposure by encouraging mitigation in buildings with elevated levels of radon. The program (ongoing for 14 years) promotes radon testing of homes, businesses, and schools through outreach programs and distribution of free radon test kits; maintains a statewide database; and is responsible for listing individuals certified to perform radon services in California. DHS encourages all homeowners to test their home for radon, and provides test and remediation information to the public.

In addition, yearly intensive radon screening studies of selected counties identify areas with the potential for elevated indoor levels of radon based on short-term indoor radon measurements. Selection of counties and sampling sites is based primarily on geology because uranium occurrence is not random: it is largely influenced by geology. There are three primary benefits arising from the screening studies: 1) outreach and public education: approximately 25,000 recruitment/radon information letters are sent to residents of each selected county; 2) identification of areas of high indoor radon potential: mitigation and construction of radon resistant homes are encouraged in these areas; and 3) production of detailed radon potential maps. To date, three such maps have been produced (Santa Barbara, Ventura and, soon to be released, Los Angeles) and are available through the Program's web page at www.dhs.ca.gov/radon and distributed to interested city and county agencies.

2.3.10 Asbestos

Asbestos is a naturally occurring mineral fiber with strong commercial appeal due to its physical properties. It is a poor conductor, but a good insulator, and is strong, flexible, non-corrosive, and flame-resistant. Asbestos is a commercial term for a number of naturally-occurring minerals: chrysotile belongs to the serpentine group, while amosite, crocidolite, tremolite, actinolite, and anthophyllite are in a group called amphiboles. When inhaled, asbestos fibers penetrate deep

into lung tissues where they cannot be expelled or destroyed by the body. Federal and International agencies recognize that asbestos is a carcinogen. In 1986, asbestos was identified as a toxic air contaminant under California's Toxic Air Contaminants Program (AB 1807). Exposure to asbestos is associated with increases in non-malignant respiratory symptoms, and may cause asbestosis, lung cancer, and mesothelioma (ATSDR, 2001). Indoor asbestos is primarily found in older homes in pipe and furnace insulation, shingles, millboard, textured paints and other coating materials, and floor tiles. Due to the success of remediation efforts, asbestos concentrations are generally quite low in most buildings today.

2.3.10.1 Health Effects of Asbestos

Asbestos fibers are so small and thin that they can penetrate to the small alveoli in the lungs. Once the fibers are inside the lungs, the body's defense mechanisms attempt to break them down and remove them. Despite these attempts, many fibers remain in the body and are potential disease-causing agents. Each fiber is treated as a foreign body; inflammations develop as the body tries to neutralize, break down or remove the sharp, irritating fibers. Fibers longer than 5 μm in length tend to have the greatest health impact. These fibers and the bodies defense mechanisms lead to the development of the various kinds of asbestos-caused diseases.

Lung cancer is the predominant asbestos-related disease, accounting for the majority of deaths from asbestos exposure. Asbestos workers who smoke are at greater risk of developing lung cancer than workers who do not smoke. Asbestosis, a diffuse fibrous scarring of the lungs characterized by shortness of breath, mainly arises after long-term heavy exposure to asbestos. Mesothelioma, a rare cancer of the lining of the chest and abdomen, does not appear to be influenced by smoking. A study of vermiculite miners in Libby, Montana revealed a 4.2% death rate due to mesothelioma (McDonald *et al.*, 2004). Based on the high-end estimate of the potency of mixed asbestos fibers, breathing air that contains 100 fibers/m^3 (0.0001 fibers/ml) poses a one to two in ten thousand excess lifetime risk of cancer (ARB, 1986). Others (U.S. EPA, 1994) have made similar estimates.

Non-occupational exposures are generally low compared to occupational exposures. Workers' families have developed asbestos-related pulmonary disease (Whitehouse, 2000; Peipins *et al.*, 2003), lung cancer and mesothelioma (Magnani *et al.*, 2001) from para-occupational exposure to asbestos, such as inhalation of asbestos fibers released by clothing and equipment brought home from job sites. People living in the vicinity of asbestos mines and factories may be at risk from neighborhood exposure to asbestos. Although the risk to the general population is minimal, no safe exposure level for asbestos has been established.

2.3.10.2 Sources of Asbestos

For 30 years following World War II, asbestos was extensively used in the renovation and construction of homes, schools, and public buildings. Asbestos was once found in nearly 3,000 different types of commercial products, including older plastics, paper products, brake linings, floor tiles, textiles, sealants, cement pipe, cement sheets, and insulation (NTP, 2002). It is now prohibited in the manufacture, processing, and importation of most products by the 1989 Asbestos Ban and Phase Out Rule (40 CFR 763 Subpart I, Sec. 762.160 - 763.179). Some asbestos paper products and asbestos-cement products remain in use today.

Friable asbestos-containing material (ACM) can be crumbled by hand pressure when dry, releasing fibers to air for potential exposure by individuals. Non-friable ACM can become friable

during abatement and maintenance activities, and with age. The three most common asbestos forms used in construction are amosite, crocidolite, and chrysotile (DHS, 2000). When the minerals are crushed, they break into tiny fibers invisible to the naked eye, but distinguishable under light microscope. Chrysotile fragments into curly fibers; the other types release tiny, needle-like fibers.

Homes built in areas of naturally occurring asbestos may become contaminated with tracked-in asbestos and infiltration of fibers that are suspended in outdoor air. Once such fibers are indoors, they can be resuspended by normal household activities, such as vacuuming (OEHHA, 2000b).

2.3.10.3 Asbestos Concentrations

For the purposes of counting asbestos fibers in samples, regulatory agencies commonly count as fibers those particles of asbestos minerals at least 5 µm in length and with length:width ratios of 3:1. For other purposes, such as detecting fibers in bulk building materials, asbestos particles with length:width ratios of 5:1 are counted.

Asbestos concentrations in most buildings are quite low. Crump and Farrar (1989) examined indoor asbestos concentrations for 49 public buildings located in six cities across the U.S. The concentration of all fibers was 0.00073 f/ml, and for fibers greater than or equal to 5 µm, the average concentration was 0.00007 f/ml. The mean indoor concentration was 0.00020 f/ml for buildings with no known asbestos, 0.00059 f/ml for buildings with asbestos in good condition, and 0.00073 f/ml for buildings with damaged asbestos. There were no statistically significant differences in airborne asbestos levels among the three categories of buildings. For the 43 buildings that contained ACM, the average indoor levels for fibers greater than or equal to 5 µm were 0.00005 f/ml; these levels were indistinguishable from outdoor levels (Lee *et al.*, 1992).

In a nationwide study conducted by Lee *et al.* (1992), airborne asbestos concentrations were made for 315 public, commercial, residential, school, and university buildings. The mean indoor level was 0.02485 f/ml for total asbestos structures and 0.00013 f/ml for fibers longer than 5 µm. Indoor total asbestos was significantly higher than outdoor total asbestos for all building types. In comparing indoor and outdoor levels of fibers greater than or equal to 5 µm for building types, indoor levels were higher than outdoor levels only for schools ($p=0.003$). Only 52% of all indoor samples contained asbestos fibers. Approximately 92% of fibers were shorter than 5 µm in all buildings, and 2% of fibers were amphiboles. 90% of the samples for fibers longer than 5 µm had levels less than 0.00071 f/ml and 0.00054 f/ml in schools and public buildings, respectively.

Corn *et al.* (1991) reported asbestos concentrations for a portion of the study conducted by Lee *et al.* (1992) involving 71 occupied schools in eight different states. Schools from different states did not differ statistically in their levels of airborne asbestos. The type of asbestos-containing materials (ACM) present (i.e., chrysotile or amphibole) and the condition of ACM were not significantly correlated with levels of indoor asbestos. Most of the asbestos fibers were chrysotile (95%) with an average of 0.018 f/ml, and few were longer than 5 µm (7.9%). The mean indoor concentration of fibers over 5 µm long was 0.00024 f/ml. Total asbestos structures found indoors averaged 0.20 f/ml.

2.3.11 Polybrominated Diphenyl Ethers and Other Endocrine Disrupters

Endocrine disrupters are substances that alter the normal function(s) of the endocrine systems of animals and humans and adversely affect growth, development or reproduction. They can act

like a natural hormone, bind to a receptor and prevent a normal response, or interfere with the way natural hormones and receptors are synthesized or controlled. Public attention has been drawn to endocrine disrupters that mimic or block the natural effects of female sex hormones (estrogens), but they can also affect male sex hormones, development and behavior. The range of substances that cause endocrine disruption is wide and varied, and includes both natural and synthetic chemicals.

Polybrominated diphenyl ethers (PBDE), a chemical class of endocrine disrupters, are added to plastics and textiles to meet fire safety regulations. The PBDE class is comprised of 209 possible congeners, differentiated by the position and number of bromine atoms on the two phenyl rings. U.S. EPA has classified deca-BDE as a possible human carcinogen since it has been linked to development of liver tumors in rats and mice. PBDEs have been identified as HAPs and California (TACs).

Phthalates are another group of chemicals that have been investigated for their potential endocrine-disrupting activities and many members of this group of chemicals, e.g., di-butyl phthalate (DBP), di-(2ethylhexyl) phthalate (DEHP), have been implicated as endocrine disrupters based on sufficient evidence in laboratory animals. Many are used to provide flexibility to plastic products and have been used in the manufacture of many products including toys, vinyl upholstery, shower curtains, raincoats, garden hoses, surgical gloves, medical tubing, and blood storage bags.

2.3.11.1 Health Effects of PBDEs

Research has shown that PBDE compounds are toxic and bioaccumulate similarly to polychlorinated biphenyls (PCBs) and dioxins and have genotoxicity profiles similar to PCBs. PBDEs have similar chemical structures to PCBs and the human thyroid hormone, thyroxine (T4). Toxicological endpoints of PBDEs are suspected to be thyroid hormone disruption, neurodevelopmental deficits, and cancer (McDonald, 2002).

Neurodevelopmental deficits have been linked to PBDE exposure (Eriksson *et al.*, 1998, 1999; Viberg *et al.*, 2000, 2001). Other results indicated deficits in brain development and possibly changes in the cholinergic system. Changes in the cholinergic system interfere with choline acetyltransferase activity and contribute to motor and mental impairments in animals. A no-observable-effects level for neurobehavioral effects has not been established for PBDEs (McDonald, 2002).

Evidence shows that exposure to PBDEs can lead to disruption of endocrine function in a number of wildlife species. Effects suggesting endocrine disruption have been reported in mollusks, crustaceans, fish, reptiles, birds, and mammals in various parts of the world. There is limited evidence in humans that adverse endocrine-mediated effects have resulted from either intentional or accidental exposure to chemicals. Many of these chemicals, even at relatively low levels, are known to affect growth, reproduction, and development of organisms in the ecosystems. The impact of these substances on human health is still under investigation.

2.3.11.2 Sources of PBDE

PBDEs are added to plastics and textiles to meet fire safety regulations. The production of PBDEs has steadily increased since the 1970s. Commercial forms of PBDEs include deca-, octa- and penta-BDE. In 1999, its use in the United States was estimated at 25,000 tons or 44%

of the total global use (Hale *et al.*, 2002). California is a major user of fire retardants due to strict regulations concerning consumer safety.

Commercial penta-BDE is added to soft polyurethane foam cushions, upholstery textiles and mattresses. Commercial octa-BDE is incorporated into plastics used for hard casings of office equipment, fax machines, computers, telephone handsets and car trim. Commercial deca-BDE is utilized in high-impact plastics found in televisions, computers, stereos and other electronic equipment.

Historically, di-2-ethylhexylphthalate has constituted approximately 50% of all the phthalate ester plasticizers used. In 1994, production of DEHP was approximately 258 million pounds (IARC, 2000). DEHP is the single largest volume member of the di-octyl phthalates. However, in recent years the use of DEHP has declined because of health concerns. It is no longer used in plastic food packaging or baby teethers. Many toy manufacturers have discontinued its use in toys, and it is being replaced by linear phthalates and other plastomers in other applications (ATSDE, 2000).

2.3.11.3 PBDE Concentrations

The air and dust inside U.S. homes are likely to contain a wide variety of chemicals, many of which are identified as endocrine disrupting substances. A recent study (Rudel *et. al.*, 2003) found numerous endocrine disrupting compounds in indoor air and dust obtained from 120 homes in Cape Cod, Massachusetts. The most abundant compounds in air included bis(2-ethylhexyl)phthalate (DEHP), (median=77 ng/m^3, max= 1000 ng/m^3); *o*-phenylphenol (disinfectant) (median=70 ng/m^3, max=590 ng/m^3); 4-nonylphenol (detergent metabolite) (median=110 ng/m^3, max=420 ng/m^3); and 4-*tert*-butylphenol (adhesive) (median=16 ng/m^3, max=290 ng/m^3). The penta- and tetrabrominated diphenyl ethers used as flame retardants were frequently detected in dust. Numerous pesticides were detected in air and dusts from these homes, the most abundant being permethrins and the synergist piperonyl butoxide. The banned pesticides heptachlor, chlordane, methoxychlor, and DDT were also frequently detected. The median concentration of DEHP in the homes studied was 590 ng/m^3 in the air and 4.98 µg per gram of dust collected. The authors noted that "detected concentrations exceeded government health-based guidelines for 15 compounds, but no guidelines are available for 28 compounds, and existing guidelines do not consider endocrine effects."

PBDE congeners were quantified in samples of human breast milk collected in Sweden during the period from 1972 to 1997 (Noren and Meironyte, 1998). This Swedish study reported that levels of PBDEs in breast milk had increased 40 fold since 1972, indicating a doubling every five years. In a 2003 U.S. study, levels of PBDEs in women's breast milk measured in Texas were 10 to 100 times higher compared to levels found in Europe (Schecter *et al.*, 2003). Two recent U.S. studies in California (Petreas *et al.*, 2003) and Indiana (Mazdai *et al.*, 2003) also report similar results with levels from 10 to 100 times higher than levels reported in Europe (Schecter *et al.*, 2003).

PBDEs have been found in a wide variety of environmental samples, including fish, birds, soil sediments, air, marine mammals, and human blood (Strandberg *et al.*, 2001, Darnerud *et al.*, 2001). Penta-BDE has been detected among 89% of the fish collected from two large Virginia watersheds (Hale *et al.*, 2001a).

An air study found that PBDE compounds were widely distributed and that PBDEs with lower molecular weight, such as penta-BDE (which is found mostly in the vapor phase), can be transported through the atmosphere to remote areas (Strandberg *et al.*, 2001).

2.3.12 Non-Industrial Workplace Exposure to Air Pollutants

Non-industrial workplaces provide unique situations for exposure to indoor air pollutants. Products and activities associated with non-industrial workplaces such as beauty salons, hospitals, dry cleaners, medical laboratories, retail shops, copy shops, and other workplaces can lead to elevated levels of air pollutants. Despite regulations for pollutant levels and ventilation requirements, some workers in these environments experience adverse health effects related to indoor environmental quality.

Under CCR Title 8, Section 5155, the California Occupational Safety and Health Standards Board sets permissible exposure limits (PELs) and other requirements to limit employee exposure to airborne contaminants. The PELs legally apply to both industrial and non-industrial workplaces. The Cal/OSHA PELs include 8-hour exposure limits designed to protect healthy working adults, as well as short-term and ceiling exposure limits when they are necessary to prevent acute effects. However, they are not sufficient to prevent all health impacts for all working individuals, and they do not address possible impacts on those with asthma or reactions by sensitive individuals to low levels of chemicals. Cal/OSHA's Indoor Air Quality Policy and Procedure (C-48) states: "Most complaints about the quality of indoor air arise from employees who work in non-industrial environment. Approaches using traditional industrial hygiene techniques usually demonstrate compliance with 8 CCR section 5155 (PELs) despite the persistence of IAQ complaints from the building occupants."

Cal/OSHA also has a regulation designed to provide adequate ventilation while workers are at work. Title 8, Section 5142, *Mechanically Driven Heating, Ventilating and Air Conditions (HVAC) Systems to Provide Minimum Building Ventilation*, requires HVAC systems to be operated during hours of occupancy to provide at least the amount of outside air required by the State Building Code at the time of permitting. It also contains maintenance and record-keeping requirements.

<u>Cal/OSHA data on workplace investigations</u>
A review of Cal/OSHA inspection data indicates ventilation requirements are often not met. From 1995 through 2001 data show that citations for violations of Title 8, Section 5142 were issued during 514 on-site inspections (Gold, 2005). Of these citations, the majority (260) were in services, with 114 in schools and 33 in health care. Citations for violations of Section 5142 were issued in 112 inspections in Public Administration.

Indoor air quality problems in non-industrial workplaces range beyond inadequate ventilation. A review of data collected by Cal/OSHA from 1997 through 1999 reveals that 849 cases were coded as being related to poor indoor air quality (Gold, 2005). However, because all inspections that included IAQ concerns were not necessarily coded as IAQ, the actual number of inspections involving indoor air issues may have been more than twice this number (Gold, 2005). Of the 849 cases, 353 inspections were in service workplaces and 128 were in public administration (typically offices). These numbers indicate that problems exist with indoor air quality in these locales, but information on specific pollutants or conditions leading to the inspections and possible citations are not readily available.

In addition to the indoor air quality problems documented in Cal/OSHA records, the Hazard Evaluation Section and Information Service (HESIS) within DHS receives many inquiries from individuals who are experiencing illnesses associated with poor indoor air quality (Katz, 2005). Problems are largely associated with several types of personal-services and community-services environments. Typically, the individuals affected in these environments include employees, clients or customers, students, patients, and self-employed workers. The following discussion includes some non-office, non-industrial indoor environments with widespread indoor air problems documented by the Occupational Health Branch of DHS, the National Institute of Occupational Safety and Health (NIOSH) within CDC, and other scientific investigators.

Schools, preschools, nursery schools, daycare

Teachers and other school staff frequently report illnesses associated with poor indoor air quality. Often the problems contributing to those illnesses are not adequately remedied by school administrators for months or even years. At the request of the Legislature, ARB and DHS (2004) conducted a study of the environmental conditions in both portable and traditional classrooms throughout California. They found that a number of indoor air quality problems were widespread in California. The most serious indoor air quality problems included:

- Inadequate ventilation with outdoor air during 40% of classroom hours, and seriously deficient ventilation 10% of the time, largely due to teachers turning off the ventilation systems due to excess noise produced by the systems.
- Formaldehyde concentrations above guidelines for preventing acute eye, nose, and throat irritation in 4% of the classrooms, and exceeding guidelines for preventing long-term health effects. including cancer, in all classrooms.
- Obvious mold in about 3% of classrooms, and water stains, excess wall moisture, and other potential mold indicators in about one-third of classrooms.

Other studies have identified air quality problems in classrooms as well:

- Defresne *et al.*, (2002) found that students in a 3-hour biology lab were exposed to average formaldehyde levels of 0.25 mg/m^3 (200 ppb) in one classroom, and 0.632 mg/m^3 (510 ppb) in another classroom. These values are well above the OEHHA 8-hour guideline level of 27 ppb.
- In another study (Ryan *et al.*, 2002), investigators measured personal exposures of art students to VOCs in a university art building. Concentrations of methylene chloride, a carcinogen, were elevated near the print cleaning operation, at 27.2 µg/m^3 (std. dev. 48 µg/m^3). Concentrations were well below existing occupational limits for 40-hour exposures, but the elevated area exceeded the Proposition 65 no significant cancer risk level of 10 µg/m^3.

Photocopy centers

Numerous VOCs are emitted during the photocopy process (see Section 2.3.2.2). Stefaniak *et al.*, (2000) measured VOC concentrations in three copy centers on a university campus. 38 VOCs were detected in the personal air samples, with toluene being the highest at 690 ppb. The maximum area air concentration measured was 1,132 ppb toluene. The time-weighted average personal exposures to VOCs were 100 times less than OSHA PELs. However, the toluene levels exceed OEHHA's chronic REL of 75 ppb, a guideline designed to protect against the effects of long-term exposure, and exceed the California Proposition 65 warning level of 172 ppb (650 µg/m^3) for reproductive toxicity. Real-time total VOC (TVOC) concentrations ranged from < 71 to 21,300 ppb. The authors conclude that "even though concentrations of individual VOCs are well below regulatory limits, the time-weighted average TVOC concentration can be well above suggested levels known to cause perceived and physiological health effects in controlled laboratory studies".

Indoor sporting events

Recreational events are attended by a wide cross section of the population, including individuals with heart conditions who are more susceptible to the effects of carbon monoxide (CO). Indoor CO levels can exceed the PEL of 25 ppm at indoor sporting events such as motocross competitions, tractor pulls, monster truck shows, and automobile demolition derbies, where internal combustion engines are operated in indoor environments (Levesque *et al.*, 2000; Levesque *et al.*, 1997; MMWR, 1994). Monster truck shows and tractor pulls in Cincinnati, Ohio during 1992-1993 were found to have average levels of 79-140 ppm CO during events compared to pre-event average levels of 13-23 ppm, and a peak observation of 283 ppm (Boudreau *et al.*, 1994). The authors noted that the observed concentrations varied inversely with the arena seating level (lower levels with greater height of seats). Similar CO concentrations were observed by Levesque *et al.* (2000) during monster truck shows and demolition derbies in Canada, and in motocross events (Levesque *et al.*, 1997).

Elevated CO and nitrogen dioxide (NO_2) concentrations are often present in indoor ice-skating rinks. Pelham et al. (2002) found that, in 33 investigations of indoor skating rinks, the majority of the reviewed studies reported CO levels in excess of 25 ppm, and several studies reported NO_2 concentrations in excess of 1 ppm, well above occupational levels (STEL = 1 ppm) and well above the one-hour state ambient air quality standard of 0.25 ppm. The two most identified causes for high CO and NO_2 levels were the ice surface refinisher (Zamboni) and inadequate ventilation systems.

Hair salons and nail salons

Hair and nail products typically contain toxic and irritating chemicals, though air concentrations tend to be well within the applicable occupational standards (Labreche *et al.*, 2003; Leino *et al.*, 1999; NIOSH, 1992). Many are highly volatile or are sprayed as aerosols, yet there is often inadequate ventilation to remove the airborne chemicals. Many salons are leased facilities where the business owner has little or no control over the ventilation system, which may distribute the pollutants to adjoining businesses. HESIS has received numerous inquiries from cosmetologists and cosmetology students about this problem. Health concerns include asthma and other respiratory problems, dizziness with headache, and potential effects on pregnancy. Because of the high number of complaints and the seriousness of the health concerns, HESIS has conducted several field investigations in this field of personal-service businesses (HESIS, 2004).

Hiipakka and Samimi (1987) measured several VOCs in personal air samples of nail salon operators. Reported values were below OSHA PELs; however, workers reported health symptoms. The incidence of throat irritation was significantly elevated relative to a control group. Nail sculptors consistently reported more nose and skin irritation, drowsiness, dizzy spells, and trembling of the hands than the control group. The mean time-weighted average concentration of chemicals reported were: ethyl methacrylate, 4.5 ppb; isopropyl alcohol, 15.6 ppb; butyl acetate, 0.4 ppb; toluene, 0.8 ppb; and polymethacrylate dust, 0.9 mg/m^3 for respirable dust.

Other non-industrial workplaces with unique indoor exposure scenarios

Workers in many types of workplaces and businesses are exposed to air pollutants associated with the type of work they perform. Large air quality studies of these businesses are lacking; however various small studies, complaint records, and investigations have shown that there are indoor air quality problems in many other types of non-industrial workplaces as well. Some of these workplaces or services include:

Dry Cleaners - Residual solvents used in dry cleaning volatilize off the clean clothes leading to elevated levels in dry cleaner shops. Perchloroethylene has been the mostly widely used dry cleaning solvent; however other solvents (ethylbenzene, *m,p*-xylene, and *o*-xylene) have been used to some extent. In studies in New York, concentrations of perchloroethylene in 13 apartments connected to or near dry-cleaning shops ranged from 7.6 to 23,000 $\mu g/m^3$, averaged 5000 $\mu g/m^3$, and showed a median level of 1400 $\mu g/m^3$, well above the New York Department of Health guideline of 100 ug/m^3 (NY DOH, 2003) and the OEHHA chronic REL of 35 $\mu g/m^3$ and any acceptable cancer risk levels.

Janitors - Occupational asthma and other respiratory problems can be caused or aggravated by chemical cleaning products used inside schools, hotels, medical settings and other service facilities. California has nearly 400,000 people who regularly work with cleaning products. The Occupational Health Branch (OHB) of DHS found that the rate of work-related asthma in janitors is nearly double the rate in the overall workforce. Bystanders in health care and other industries are often indirectly exposed to cleaning chemicals as well. Nurses and nursing aides constituted 20% of the bystanders who developed asthma in the OHB study (DHS, 2004b; DHS, 2004c).

Hospitals, jails, etc.- Tuberculosis, a bacteria transmitted by airborne particles, is a substantial risk for persons in group-living environments. These environments include hospitals, prisons, jails, homeless shelters, drug treatment clinics, residential facilities for HIV-infected persons (e,g.,hospices or group housing), and residential facilities for the elderly. (DIR, 1997).

Construction workplaces - Construction workers may encounter heavy concentrations of plaster/wallboard dust, adhesive VOCs, lead dust from surface preparation for painting, and fungal particles from mold remediation or flood-damaged buildings. Several organizations have published detailed guidelines for the protection of mold remediation workers (U.S. EPA, 2003b). However, very few studies have been conducted to determine the actual effectiveness of the guidelines in preventing health problems among these workers. A few case reports document illnesses among renovation workers.

Roofing work - Application of roofing asphalt is known to cause indoor air quality problems when contaminated air enters a building, often through ventilation system intake vents (Lynch and Kipen, 1998). Health complaints often continue well beyond the time of exposure.

Aircraft cabins - The pesticide permethrin is routinely sprayed on some international airliner routes to eradicate insects inside cabins. Flight attendants have become ill due to the exposure to permethrin; there is also concern about passenger safety. Airlines are not currently required to tell passengers of pesticide use (DHS, 2003). Aircraft exposures to biological contaminants and ozone also have been a concern (CITATIONS).

Semiconductor industry

Although exposures experienced by workers in the semiconductor industry are essentially industrial exposures (as opposed to non-industrial), they are included here to illustrate concerns regarding exposures in non-traditional industrial environments, and the suspected health effects of some glycol ether compounds. These same glycol ethers have been detected in some consumer products and residential environments. Ethylene-based glycol ethers used in this field have been related to reproductive problems in animal species (Eskenazi *et al.*, 1995). Eskenazi *et al.* monitored reproductive activities and pregnancy rates of female workers in the semiconductor industry. The authors concluded that women employed in certain areas (fabs, dopant, and thin-film processes) have fewer pregnancies than other workers. Women exposed to ethylene glycol ethers had somewhat lower fecundability (probability of conception) than

women unexposed to ethylene glycol ethers. Glycol ether levels were not recorded. In a similar study, glycol ethers and fluoride compounds used by workers in fabrication-rooms and photoresist processes accounted for increased risk of spontaneous abortions (Shanna *et al.*,1995). Hammond *et al.* (1996) measured time-weighted average personal exposures for semiconductor workers. Workers that poured solvent or cleaned a coater cup experienced a higher exposure with a geometric mean concentration of 81 ppb of 2-ethoxyethanol. Workers who loaded and unloaded cassettes experienced a lower exposure with a geometric mean of 14 ppb 2-ethoxyethanol.

In summary, workplaces can pose a risk not experienced in residences or offices due to the nature of the activity that occurs there, or the type of sources present. While industrial exposures are moderated through the use of engineering controls, administrative controls, and personal protection gear when needed, indoor air quality problems in non-industrial workplaces have been less recognized and less studied. Nonetheless, some exposures in non-industrial workplaces have been shown to exceed guidelines for protecting the health of the general population, although levels of pollutants in those environments generally do not exceed occupational standard levels. Indoor air quality problems in non-industrial workplaces are not unique to California: in the past 25 years, the percentage of health evaluations that NIOSH (at the Centers for Disease Control and Prevention) has conducted related to indoor-air quality has increased from 0.5 percent of all evaluations in 1978, to 52 percent of all evaluations since 1990. This means that in those years, the evaluations related to air quality concerns have increased from one of every 200 evaluations to one of every two evaluations (Office of the Surgeon General, 1-13-05 news release).

3. COSTS OF INDOOR AIR POLLUTION

The health effects from indoor air pollution impose large costs on society, both economic and personal. The loss of human life, due to indoor air pollution constitutes a large economic and social cost. In addition, the medical costs of increased disease due to indoor air pollution, such as for cancer treatments, hospitalizations, chronic respiratory disease, and emergency room visits for asthma attacks and CO poisoning, are substantial. Student absenteeism, reduced worker productivity, and associated costs also result from indoor air pollution.

However, only very limited quantitative estimates on the costs of indoor pollution are available to date. Most of the available estimates are for a few specific indoor air pollutants that have been well studied, such as ETS and radon. There are many more indoor air pollutants that pose a significant health risk to the public, but the lack of cost information or other data for California precludes us from making even rough cost estimates here.

Estimates of the economic costs of some aspects of indoor air pollution have been made for the United States (Fisk, 2000; Mendell *et al.*, 2002). These studies allow order-of-magnitude estimates of the economic costs for California due to increased asthma symptoms and lost worker productivity in schools and office buildings. In addition, cost or value estimates can be derived for some of the health effects discussed in Chapter 2, in this report.

This information is used below, along with national and California-specific cost estimates for medical treatment and other costs (where available), to estimate the economic costs or value of specific health effects from indoor pollutants in California (U.S. EPA, 2002b; Thayer *et al.*, 2003). However, only the costs for selected indoor air pollutant exposures that have been reasonably well characterized in California are used for the final estimate. To account for inflation, cost estimates are adjusted to 2000 dollars using the relative increase in the urban consumer costs of the Consumer Price Index; for adjusting health care costs, the changes in medical care costs are used instead (USCB, 2002). Table 3.1 presents the unit medical costs (direct and indirect) and economic valuations of premature deaths used in the following analyses.

3.1 PREMATURE DEATH

Exposure to some air pollutants, primarily carbon monoxide, ETS, other carcinogens, and PM, can lead to near-term death or significantly increase the risk of premature death. The economic impact of premature death, or the value of a human life, has been estimated in the scientific literature and in environmental regulatory settings. The estimation methods include those based on: 1) an individual's willingness to pay to avoid a health risk, 2) the additional compensation demanded in the labor market for riskier jobs, and 3) society's willingness to pay to avoid a health or safety risk.

U.S. EPA (2002b) reviewed 26 value-of-life studies that used either the first or second method above, and confirmed the finding of a previous literature review that "most of the reasonable estimates of the value of life are clustered in the $3.7 to $8.6 million range."

Table 3.1. Unit Costs for Health Effects.

Outcome	Method[1]	Unit Cost in Year 2000 Dollars [2]
Premature death	WTP	$6,330,000[3]
Cancer medical costs (various types, survivor only)	COI	$94,600
Lung cancer medical costs (weighted average for all cases)	COI	$64,900
Asthma patient, direct medical and indirect costs	COI	$640[4]
Cardiovascular hospitalization	COI	$15,200[6]
Low birth weight, lifetime	COI	$118,000
Otitis media (middle ear infection)	COI	$360[5]
Asthma, emergency room visit	COI	$310[7]
Asthma, chronic	WTP	$33,000[7]
Bronchitis, acute	WTP	$59[7]
Respiratory hospitalization (U.S.)	COI	$11,000[6,7]
Acute respiratory hospitalization (CA) Age < 18 Age ≥ 18	COI	 $11,800[6,7] $23,500[6,7]

1. WTP: willingness to pay method; obtained from surveys. COI: cost of illness method; based from medical cost data and estimates of indirect costs such as lost workdays.
2. Unless noted otherwise, all values are averages from *Cost of Illness Handbook* (U.S. EPA, 2002b), after adjustment to 2000 dollars using the Consumer Price Index for medical care cost.
3. Midpoint value, adjusted for inflation using urban consumer costs.
4. From Weiss and Sullivan (2001), including direct medial and indirect costs. The 1998 U.S. estimates have not been adjusted to 2000 dollars.
5. From Gates (1996). The estimated annual U.S. cost of $5 billion per year (in 1993 dollars) was divided by the estimated caseload of 14 million children under 5 years of age.
6. From a study of California costs by Thayer *et al.* (2003). Cost adjusted for inflation using the Consumer Price Index for medical care costs.
7. These values were not used in this analysis due to inability to separate hospitalization cases from doctor's visits in the literature.

Based on its review and input from the scientific community, U.S. EPA selected $6 million (1999 dollars) as a point estimate for the "value of a statistical life," along with caveats for applying the study results to the general population. U.S. EPA (2000c) has used this point estimate in assessing the impact of regulations for diesel fuel and other pollutant sources. This point estimate is equivalent to $6.33 million in 2000 dollars, and is used in the following sections to estimate the economic valuation of excess premature deaths due to indoor air pollution. The estimated valuation of premature death from indoor air pollution in California is discussed below and summarized in Table 3.2.

Another approach to estimating the economic cost from deaths due to indoor air pollution is to base the estimate on court decisions and insurance awards, such as those from CO poisoning. However, nearly all cases of fatal and non-fatal CO poisoning are settled out of court, and utility companies are not required to track such cases; thus, there is little reliable data available on these legal settlements.

3.1.1 Deaths From CO Poisonings

Based on the DHS review of coroners' reports in the 1980's, an average of 30-40 Californians die each year from accidental CO poisoning (Girman *et al.*, 1998; Liu *et al.*, 1993a, 2000). On average, about 2/3 of those deaths (about 20-26, or 23 average) documented in the DHS study were due to appliance-related causes such as faulty furnaces, gas ovens used for space heating, and charcoal grills used indoors. This estimate has a high degree of certainty relative to other pollutant risk estimates because the CO death estimates are based on coroners' reports that clearly identify CO poisoning deaths. The economic value of these lost lives in California is estimated to be $130-160 million per year (2000 dollars), or about $150 million on average, as shown in Table 3.2.

Although the rate of CO deaths in the U.S. population has declined over the years (CPSC, 1997), the actual numbers of CO poisoning cases in California may have increased since 1990. Based on California's population growth alone – from 30.38 million in 1990 (USCB, 1990) to 33.87 million in 2000 (DOF, 2002ab), a 14% increase – one might expect the number of deaths to have increased by a similar percentage. In addition, much of this population growth has occurred in the inland and foothill regions of California, which have longer, colder winters than coastal areas, and thus, would have a greater likelihood of CO poisoning due to more frequent use of combustion appliances for space heating.

However, trends toward increased use of CO alarms and testing of combustion appliances could potentially have reduced the number of appliance-related CO poisonings in California since 1990. CO poisonings at the national level have declined (CPSC, 1997), which may be due to lower pollution emissions from motor vehicles and the increased heavy marketing of inexpensive CO alarms for use in homes, motor homes, and boats. The state's program for weatherizing existing, low-income homes has required installation of CO alarms and combustion appliance safety testing, as discussed in Section 4.3.2.

Overall, based on the factors in both directions discussed above, the current number of fatal CO poisonings in California due to appliance-related causes is assumed similar to that in 1990. It is likely that the risk of CO poisoning remains higher than average in older homes with older gas appliances, in homes with propane appliances, and in some socioeconomic groups that use unvented gas stoves or charcoal grills indoors more frequently.

Table 3.2. Estimated Valuation of Mortality Attributed to Indoor Air Pollution in California

End Point	Excess Premature Deaths (cases/yr)[1]			Valuation per case[2] ($ Million)	Cost per Year[3] ($ Billion)		
	Low	Average	High		Low	Average	High
CO: Poisoning[4]	20	23	26	6.33	0.13	0.15	0.16
VOCs: Lung cancer[5]	115	115	115	6.33	0.73	0.73	0.73
ETS: Lung cancer[6,7,8]	380	380	1,020	6.33	2.4	2.4	6.5
ETS: Heart disease[7,8]	1,700	3,600	5,500	6.33	11	23	35
Radon: Lung cancer[9]	600	1,500	3,500	6.33	3.8	9.5	22
Mold and Moisture: Asthma and allergy[10]	NA	NA	NA	NA	0.031	0.031	0.031
TOTAL[11]					**18**	**36**	**64**

1. Low and high estimates are based on a range or confidence intervals, where available. Average estimates are based on mean values from available estimates or ranges.
2. From *Cost of Illness Handbook* (USEPA, 2002b), adjusted to 2000 dollars.
3. Costs per year are rounded to 2 significant figures.
4. Case estimate from coroner's data in California (Liu *et al.*, 2000).
5. Case estimate from average values in California Comparative Risk Project (CCRP, 1994), updated. See Section 2 and Appendix II of this report.
6. From best risk estimate rather than average risk estimate; for spousal smoking only (OEHHA, 2004). Adjusted to 2000 population total. This risk estimate is probably an overestimate because it assumes ETS exposure and death rates in California are the same as those for the U.S.
7. From OEHHA (2004).
8. OEHHA (2004) is currently incorporating comments from the independent Scientific Review Panel on this report.
9. From Waldman (2004), based on USEPA (2003c), with adjustments for smaller population, lower average residential radon concentrations, and lower smoking prevalence rates in California.
10. Cost estimates taken from national cost estimated by Weiss and Sullivan (2001) of $2 billion (in year 2000 dollars), scaled to 12% for California estimate, and multiplied by fraction of asthma risk attributed to mold and moisture in three large epidemiology studies (0.13).
11. These totals do not include death from exposures to other types of indoor PM, which could be substantial but are not currently quantifiable due to lack of appropriate studies.

3.1.2 Deaths From Volatile Organic Compounds (VOC) Exposure

As discussed in Section 2 and Appendix II, about 230 excess cancer cases due to VOCs from indoor exposures are estimated to occur in California each year. This estimate is based on the results of the California Comparative Risk Project (CCRP, 1994) that have been updated to reflect reductions in formaldehyde exposures and increased population growth as of the year 2000. These are conservative estimates relative to the total cancer burden from indoor carcinogens, because they do not include the additional, significant cancer risk from radon, and the risk from many other carcinogens also found in indoor air and house dust, such as acetaldehyde, PAHs other than B(a)P, phthalates other than DEHP, and asbestos.

Half of the VOC-related cancer cases attributed to indoor air (115) per year are estimated to result in premature death. U.S. EPA (2002b) assumed this same fraction of fatal cases within five years of diagnosis when estimating the costs of typical cancer treatment. Actual premature death rates could be higher than one-half of the cases because cancers caused by these air pollutants typically result in lung, organ, or blood cancers, which are more difficult to detect and treat, and thus, are more deadly. The estimated value of premature deaths from the 115 excess cancer deaths in California totals $730 million per year, as shown in Table 3.2. This estimate does not explicitly include the much smaller costs of cancer treatment, which are discussed below.

3.1.3 Deaths from Environmental Tobacco Smoke Exposure

ARB/OEHHA (2004-2005) estimate that exposure to ETS from spousal smoking in California in 2003 produced 400 premature deaths (range 411-1,064) due to lung cancer. These estimates are based on the best risk estimate rather than the average risk estimate. They also are based on smoking prevalence rates in the U.S., and scaled down to reflect that California contained 12% of the U.S. population.

To adjust the ARB/OEHHA estimate back to the year 2000 population, the lung cancer risk estimate was multiplied by 0.96. This yields an estimated number of 380 excess lung cancer deaths cases per year from ETS exposures. The estimated value of premature deaths from the 380 excess lung cancer deaths (range 390-1,020) in California totals $2.4 billion per year, as shown in Table 3.2. This estimate does not explicitly include the much smaller costs of cancer treatment, which are discussed below

For ETS effects on ischemic heart disease, ARB/OEHHA (2004-2005) estimated that ETS exposure resulted in 1,700-5,500 (average = 3,600) premature deaths from heart disease. This estimate was based on 1999 ETS exposure data and 2000 mortality data for California. The estimated value of 3,600 deaths is $23 billion, as shown in Table 3.2.

The estimated numbers of premature deaths from ETS exposure, and the morbidity effects discussed below, probably overestimate current risk levels for two reasons. First, ETS exposure is likely much lower in California compared to the U.S. The CDC (2004) found adult and adolescent smoking prevalence in California was about 16% in 2001, while the U.S. prevalence rate was 22%. In addition, cigarette consumption by California adults was found to be about half of the U.S. average in 2001-2002 (DHS, 2002a). Secondly, the estimates include home and workplace exposure, but California's workplace smoking ban has virtually eliminated ETS exposure in enclosed workplaces, with a few exceptions. By 1999 over 93% of indoor workers reported having a smoke-free workplace (DHS, 2002b). On the other hand, newer data on the health effects of ETS may increase the unit risk of ETS for death and disease. For example,

ARB/OEHHA (2004) estimate that ETS increases the risk of breast cancer by about 30%. ARB/OEHHA currently revising the report to reflect the comments of the external Scientific Review Panel on breast cancer and other topics, and those revised estimates may be included in this report at a later date.

These costs of ETS-related health effects could be prevented if people smoked only outdoors away from other people, or stopped smoking. However, to achieve these potential cost savings, further efforts are needed to change peoples' smoking behaviors, and to improve awareness of and control of other indoor and outdoor asthma triggers in California (Meng *et al.*, 2003).

3.1.4 Deaths from Radon Exposure in Homes

The DHS has estimated that 1,500 excess lung cancer deaths per year are attributed to the residential radon exposure in California (Waldman, 2004), as discussed in chapter 2. This first-order estimate is based on the U.S. EPA's (2003c) national estimate of 21,000 excess lung cancer deaths (90% CI: 9,000 to 50,000). To calculate the residential radon risk for excess lung cancer in California, the national estimates were multiplied by the fraction of the U.S. population in California (34 million out of 281 million, or 12%; U.S. Census 2000 data). The national estimates were also multiplied by the ratio of average radon concentrations (CA: 1 pCi/l; U.S.: 1.25 pCi/l; ratio of 0.8) and the smoking prevalence in California (CA: 16.4%; U.S.: 22.5%, CDC, 2004). This yielded an estimate for the radon-induced lung cancer death in California of about 1,500 (90% CI: 600-3,500) deaths per year. This may overestimate the excess deaths because recent measurements in the Sierra Nevada region indicate that this region has much lower radon levels than that reported from the previous statewide survey (3 vs. 25%), and that the statewide average may be lower than previously estimated (Tsai and Waldman, 2005). The estimated value of premature deaths from the 1,500 excess lung cancer deaths in California attributable to radon totals $9.5 billion per year, as shown in Table 3.2.

3.1.5 Deaths from Mold and Moisture-related Problems in Homes

Weiss and Sullivan (2001) estimated that the costs of excess death in children and adults due to asthma in the U.S. was $2 billion, in year 2000 dollars. Scaling this to reflect that 12% of the U.S. population resides in California gives an estimated cost for asthma deaths in California of $0.24 billion. As discussed below in the Medical Costs section, the fraction of these health effects attributed to mold and moisture problems in homes is 0.13. This fraction is an average of four large, well-conducted epidemiology studies of adults and/or children (shown in Table 3.4). Multiplying the estimated cost of asthma in California by this fraction yields an estimated cost of $31 million attributable to excess death due to residential mold and moisture problems. This estimate does not include other indoor allergen sources such as dust mites, pets, cockroaches, and chemical emissions.

3.2 MEDICAL COSTS

Illness and disease caused by indoor air pollution include the production of new asthma cases (induction), exacerbation of asthma symptoms, development of other respiratory disease and symptoms, and induction or exacerbation of allergies. These impacts of indoor air pollution also affect the quality of a person's life in terms of reduced or limited activities, limited employment opportunities, and reduced productivity.

The cost of illness and disease has been estimated in various ways. These include methods based on medical costs, work-related costs, education-related costs, and willingness of

individuals to pay to avoid the anxiety, pain, suffering, and other health risks (U.S. EPA, 2002b). The following section uses medical costs shown in Table 3.1 to estimate the medical costs of indoor air pollution in California, but generally excludes the indirect costs of reduced productivity of individuals and employees. The estimated medical costs are summarized in Table 3.3.

3.2.1 CO-related Hospitalization

About 175-700 emergency room visits and hospitalizations (midpoint of 438) due to non-fatal CO poisoning are estimated to occur in California each year, on average (see Section 2.2.4.2). Emergency room visits cost about $442 per visit, based on costs for asthma room visits to emergency rooms and adjustment to 2000 dollars (U.S. EPA, 2002b). Therefore, the annual cost of emergency room visits due to CO poisoning in California ranges from $77,000-310,000, or an average of about $190,000, as shown in Table 3.3.

The estimated number of emergency room visits due to CO is conservative because CO poisoning can be misdiagnosed as a viral flu illness or general fatigue. The estimated cost for each case of non-fatal CO poisoning is conservative because it does not include several types of costs that could be substantial, such as the costs for lost school and work days, hyperbaric oxygen treatment for severe poisoning, long-term neurological and developmental damage to many victims of sub-lethal CO poisoning, and the estimated hundreds to thousands more CO poisoning cases per year that do not result in hospitalization.

3.2.2 VOC-related Cancer Treatment

The number of excess cancer cases from VOC exposures was estimated above to be 230 cases per year, and one-half of those cases (115) were assumed to survive, as discussed above. The average cost of medical treatment in the U.S. for the 13 most common types of cancer, when assuming a 50% death rate at five years and no discounting, is $82,581 in 1996 dollars (U.S. EPA, 2002b) or $94,619 in 2000 dollars. This treatment cost estimate is conservative because it does not consider recent changes in cancer treatment that can be very expensive, such as the bone marrow transplant procedure and new pharmaceutical treatments. At this treatment cost per cancer case, the total cost for medical treatment for 115 cases from VOC exposure was estimated to be $11 million, as shown in Table 3.3.

3.2.3 ETS-related Illness and Hospitalization

As discussed above, ETS exposure was estimated to result in 380 excess deaths per year from lung cancer. To estimate the medical costs of this impact, the approach used above for VOCs was used, except that the average cost of medical treatment for lung cancer alone was used instead: $56,624 in 1996 dollars (U.S. EPA, 2002b) or $64,900 in 2000 dollars. Costs for lung cancer only were used because ETS exposure is primarily associated causally with lung cancer, while VOCs are causally associated with several other types of cancer. This cost is based on the total costs for non-survivors and survivors over 10 years, weighted annually for survival rates and discounted at 5%. The total cost of cancer treatment for the 380 cancer cases from ETS exposures was estimated to be $25 million (in 2000 dollars), as shown in Table 3.3.

For estimating costs due to hospitalization for heart disease due to ETS exposure, an approach similar to that used above for VOCs was used. However, in this case, the average cost of medical treatment for heart disease ($15,200) from Table 3.1 was used. The total cost of

hospitalization for 3,600 heart disease cases attributed to ETS exposures was estimated to be $55 million (in 2000 dollars), as shown in Table 3.3.

ETS exposure in California was estimated to have resulted in at least one asthma episode in 31,000 (range of 24,000-40,000) non-smoking children under 18 years of age over the last 12 months (ARB/OEHHA, 2004-2005). This estimate was based on a meta-analyses of 29 epidemiological studies, and an attributable fraction based on California data for population and ETS exposure prevalence for 2000. The estimate includes exacerbation of asthma in both new and existing asthma patients in the last 12 months. These health effects were primarily observed in infants and older children, but they may also increase the risk of health effects later in life. The average unit cost for each asthma patient in the U.S. was estimated to be $640 per year, based on national data for 1998 (Weiss and Sullivan, 2001). This unit cost includes direct medical and indirect costs for all age groups, and is about twice the unit cost for asthma treatment in an emergency room ($310). Actual costs for asthma episodes are likely to be higher because the medical costs of asthma treatment are rising rapidly and the costs of upper and lower respiratory conditions that occur along with asthma are not included (Weiss and Sullivan, 2001). Multiplying the number of asthma episodes by the unit cost for asthma patients ($640) from Table 3.1, yields an estimated cost of $19 million per year (1998 dollars) in direct medical costs and indirect costs, as shown in Table 3.3.

ARB/OEHHA (2004-2005) estimate that ETS exposure resulted in 1,600 cases of low birth weight. Again, the case numbers multiplied by the unit medical cost shown ($118,000) in Table 3.1. The estimated cost of the 1,600 cases of low birth weight infants was $190 million. This unit medical cost is likely to be an underestimate of actual costs because credible data are lacking for the first two years of life when intensive medical care and additional hospitalization are most likely, and for some age groups where costs were extrapolated from others (U.S. EPA, 2002b). In addition, cost data for non-medical expenses over a lifetime were lacking; these costs could be substantial as well.

ARB/OEHHA (2004-2005) estimated that 4,700 cases of premature delivery result from ETS exposure in California each year. Premature delivery is a major cause of infant mortality and infant medical expenses in the U.S., especially among African-Americans (MOD, 2003). Because the large majority of premature infants have low birth weights (Mattison *et al.*, 2001), most of the medical costs of premature delivery are included in the cost estimate above for low birth weight due to ETS exposure. However, the increased costs of premature death, long-term disability, and indirect expenses due to premature delivery are not available. OEHHA is currently revising the report to reflect the comments of the external Scientific Review Panel on developmental effects and other topics, and those revised estimates will be included in this report at a later date.

ARB/OEHHA (2004-2005) also estimated that 51,700 cases of otitis media (middle ear infection) in children were associated with ETS exposure in California. This estimate is based on ETS exposure of California children in 1999, and 2000 California population data. Gates (1996) estimated that the direct and indirect cost of otitis media totaled $5 billion dollars per year in 1993. Dividing this cost by the estimated caseload of 14 million children under the age of 5, the primary patients for this diagnosis, yields a unit case cost of $360 per year (1993 dollars). This unit cost is an underestimate because it does not include all medical costs, the potential cost of impacts on children's learning and development, and the increase in medical costs and consumer costs since 1993. This unit cost is relatively minor compared to, and somewhat redundant with, the costs estimated above for new cases of chronic asthma ($33,000); it is provided here to address the impacts on persons with pre-existing asthma. Multiplying the

estimated number of cases by the unit cost ($360) yields an estimated cost of $19 million per year (1993 dollars), as shown in Table 3.3.

Table 3.3. Estimated Annual Medical Costs of Indoor Air Pollution in California.

Indoor Pollutant and Health End Point	Average Cases per Year [1] (Range)	Medical Cost per Case[1,2] ($)	Average Cost per Year[3] ($ millions)
CO: poisoning	438 (175-700)	442	0.19
VOCs: cancer	115	94,600	11
ETS: lung cancer[4,5]	380	64,900	25
ETS: heart disease[4,5]	3,600 (1,700-5,500)	15,200	55
ETS: asthma episodes[4,6]	31,000 (24,000-40,000)	640	20
ETS: low birth weight[4]	1,600	118,000	190
ETS: otitis media visits[4]	51,700	360	19
Radon: lung cancer[7]	1,500	64,900	97
Mold & moisture: asthma and allergy[8]	NA[9]	NA	190
Total[10]			**610**

1. Original data were adjusted to 2000 dollars and 2000 population where necessary.
2. Medical cost values were taken from Table 3.1 and do not include indirect costs, unless noted otherwise. Thus, cancer treatment cost does not include costs for outpatient prescription medications and nursing home care below the skilled level.
3. Rounded to two significant figures.
4. Case estimate from OEHHA (2004).
5. Medical costs for fatal cases of cancer or heart disease also are implicitly included in death valuations, but are generally an insignificant amount relative to the total valuations.
6. The case estimate is for children under 18 years of age with at least one asthma episode in the past 12 months.
7. From Waldman (2004), based on U.S. EPA (2003c) with adjustments for smaller population, lower average residential radon concentrations, and lower smoking prevalence rates in California.
8. Cost estimates taken from national estimates for medical and indirect costs by Weiss and Sullivan (2001), adjusted to 2000 dollars, scaled to 12% for California estimate, and multiplied by fraction of asthma risk attributed to mold and moisture in four large epidemiology studies (0.13). Includes estimate of $60 million for indirect costs, such as lost workdays, lost school days, and reduced housekeeping.
9. NA = not available.
10. These totals do not include disease from exposures to other indoor PM, which could be substantial but are not currently quantifiable.

3.2.4 Radon-Related Cancer Treatment

As discussed above, residential exposure to radon was estimated to result in 1,500 excess deaths per year from lung cancer. To estimate the medical costs of this impact, the approach used above for ETS was used; $64,900 in 2000 dollars was used for the average cost of medical treatment for lung cancer (U.S. EPA, 2002b). Only costs for lung cancer were used because radon exposure is primarily associated causally with lung cancer. The total cost of cancer treatment for the 1,500 cancer cases attributed to indoor radon exposures was estimated to be $97 million (in 2000 dollars), as shown in Table 3.3.

3.2.5 Medical and Indirect Costs Associated with Indoor Mold and Moisture-related Problems in Homes

Available data demonstrate a relatively strong and consistent association of dampness and mold in buildings with an increase in lower respiratory symptoms that are often considered evidence of asthma exacerbation (IOM, 2004). The specific biological, chemical, or physical agent responsible for the health-relevant exposures from indoor dampness and mold are not fully understood. Few large studies of mold, allergens, and other biological contaminants have been conducted in office buildings, schools, and other non-residential buildings, so this section focuses on residential exposures.

To estimate the cost of asthma attributable to dampness or mold, the fractions of asthma attributable to dampness or mold in apartments and homes in the U.S. and California are multiplied by estimates of the cost of asthma. Four large studies of the health risks of building dampness and mold for adults and/or children have been conducted in the U.S. and Canada, as shown in Table 3.4. Each of these studies controlled for the effects of many factors other than dampness and mold. Most of the study areas have moderate or cold climates similar to California, and the three studies from Canada or the U.S. had very similar odds ratios for dampness or mold. In addition, southern California homes in the ARB's Children Health Study (Taylor, 2004) were found to have reported dampness or musty odors at rates similar to those reported in the U.S. and Canadian studies (Spengler *et al.*, 1994; Dales *et al.*, 1991ab) – over 30%. Thus, the results of the studies shown in Table 3.4 can be used to make estimates for California.

Equation 1 was used to estimate the fraction of asthma attributable (AF) to dampness and mold, using odds ratios (OR) and prevalence rates (p) specific to each study population:

$$AF = p(OR-1) / (p(OR-1)+1)$$ *[Equation 1; Lilienfeld and Lilienfeld, 1980]*

Table 3.4 shows the calculations of attributable fraction[1] using odds ratios, rather than relative risks, from the results of these four major studies. The average attributable fraction of asthma due to dampness or mold in homes is estimated to be 0.13.

[1] The attributable fraction is normally calculated using the relative risk value, but odds ratios can be used instead where the outcome prevalences are relatively low. For example, with prevalence rates of 11.4% for asthma symptoms and 16.9% for lower respiratory symptoms in the study by Spengler *et al.* (1994), the relative risks are approximately equal to the odds ratios. Thus, the attributable fraction can be estimated using odds ratios (ORs) in place of relative risks (RRs).

Table 3.4. Fraction of Asthma Attributable to Mold or Dampness in Houses

Study and Condition	Study Population	Prevalence of Condition (%)	Asthma Symptoms	
			Adjusted Odds Ratio (95% CI)	Estimated Attributable Fraction
Spengler *et al.* (1994), mold or mildew	12,842 U.S. children ages 9-11	36	1.39 (1.23-1.57)	0.12
Dales *et al.* (1991a), dampness or mold	13,495 Canadian children ages 5-8	38	1.45 (1.23-1.71	0.15
Dales *et al.* (1991b), dampness or mold	14,700 Canadian adults	38	1.56 (1.25 -1.95)	0.18
Zock *et al.* (2002), mold or mildew last year	19,218 adults, 38 centers in U.S., Europe, Australia, India, New Zealand	22	1.28 (1.13-1.46)	0.06
AVERAGE				**0.13**

To estimate the national cost of asthma, two estimates of asthma costs (Weiss and Sullivan, 2001; Smith *et al.*, 1997) were reviewed. The estimates of direct costs from the two studies are similar. However, the estimate of medical and indirect costs based on Weiss and Sullivan (2001) are more recent than those from Smith *et al.* (1997), and they do not exclude persons in the military, schools, and other institutions. Thus, the Weiss and Sullivan results may better reflect the current costs of treatment and medications, and are used here.

The Weiss and Sullivan estimates were adjusted to 2000 dollars and 2000 population using the data on population growth and medical care and general inflation. No adjustment was made for changes in asthma prevalence because it is not clear that asthma prevalence has changed since 1994 (Mannino *et al.*, 2002). It was also assumed that the prevalence of asthma in California is similar to the average prevalence of asthma in the U.S. Data in IOM (2004) indicate that self-reported asthma prevalence in California was 7.1% versus 6.4% for the U.S.; however, more recent data (Rhodes *et al.*, 2002) indicate that the prevalence of asthma in 2002 was 6.4% in California versus 7.5% for the full U.S. Thus, it is not clear that asthma prevalence in California differs from that for the U.S. at this time.

The updated costs for the full U.S. were then multiplied by the 0.12, the percentage of U.S. population that resided in California in 2000. As shown in Table 3.5, the estimated premature death costs are $240 million, as discussed in the premature death section above. The estimated medical costs and indirect costs (such as lost work days and school days) of asthma in California are $980 million and $460 million, respectively.

To estimate the asthma costs attributed to indoor mold and moisture problems, these costs of asthma in California were multiplied by 0.13, the attributable fraction of asthma from mold and dampness that was calculated above. This yields the estimated medical and indirect costs of $130 million and $60 million, respectively, for a total of $190 million, shown in Table 3.3. This estimate does not include the costs of other indoor allergen sources such as dust mites, pets, cockroaches, and chemical emissions.

Table 3.5. Updated Estimates of Annual Cost of Asthma.

Population Group	Cost in U.S. ($ Billions)[1]				Cost in California ($ Billions)[1]			
	Death	Medical	Indirect[2]	Total	Death	Medical	Indirect[2]	Total
Children	–	–	1.2	–	–	–	0.14	–
Adults	–		2.6	–	–	–	0.31	–
Both	2.0	8.2	3.8	13.9	0.24	0.98	0.46	1.7

1. In 2000 dollars. Row and column totals may not add precisely due to rounding of numbers. Source of U.S. estimates: Weiss and Sullivan (2001). California costs were estimated to be 12% of U.S. costs, based on the relative population sizes.
2. Indirect cost elements include loss of work, loss of school, and reduced housekeeping.

Extensive data on are not available on the costs of asthma that are preventable, but a large portion would appear to be readily preventable. The particle sources considered in this analysis are tobacco smoking, pets, use of gas stoves for heat, dust mites, cockroaches, and dampness/mold. Behavioral changes can eliminate all indoor tobacco smoking, indoor pets in homes of people with pet allergies, and use of gas stoves for heat in the homes of asthmatics. Dust mite allergen levels can be diminished by reducing indoor humidity and by surface cleaning; however, studies of dust mite remediation measures have had only moderate success. Cockroach infestations can be reduced substantially using pest management methods. Mold contamination in buildings can be reduced by preventing and remediating dampness problems.

Many dampness problems, probably a majority of serious problems, result from water leaks that could be prevented through better building maintenance and improved design and construction. These measures would also reduce the costs of dampness-caused mold contamination and degradation of building materials. Better ventilation and use of dehumidifiers could reduce dampness problems that result from high indoor humidity. Thus, with proper measures, it is probably feasible to eliminate at least 50% of the particle exposures that contribute to asthma exacerbation, and likely more.

3.3 PRODUCTIVITY COSTS OF INDOOR AIR POLLUTION

Sick building syndrome is a collection of non-specific symptoms such as eye, nose, skin, and throat irritation; headache; fatigue; and skin rash that have no known cause. Inadequate building ventilation, as indicated by elevated indoor CO_2 concentrations, as well as elevated indoor levels of VOCs, elevated levels of biological contaminants, and other environmental stressors have been implicated as potential causes of sick building syndrome.

Fisk (2000) estimated the economic impacts of sick building syndrome in the U.S. due to reductions in worker productivity. Several field and laboratory studies of office buildings and school buildings were reviewed; the performance reduction for specific tasks ranged from 3-5%. The midpoint value of 4% was reduced to 2% to estimate the overall productivity reduction throughout the day. Using this conservative estimate of a 2% preventable reduction in worker productivity due to sick building syndrome, the estimated cost savings for the U.S. in 1996

dollars were $10-30 billion ($11-33 billion in 2000 dollars). An independent group of scientists who reviewed the literature and assessed the impacts of indoor air quality on worker health and productivity for the National Institute of Occupational Safety and Health estimated the same costs (Mendell *et al.*, 2002).

To estimate the costs of sick building syndrome in California, this $11-33 billion U.S. estimate by Fisk can be scaled to reflect California's portion of the U.S. population, or 12%. This would yield a California estimate of $1.3-4.0 billion per year, for an average of $2.6 billion per year.

Kats (2003) used the 2% preventable reduction in worker productivity estimate by Fisk (2000) to estimate an avoidable cost of sick building syndrome in California. Using California-specific data where available, Kats estimated this avoidable cost to be $9 billion per year ($6 billion in wages and $3 billion in worker benefits). Using only a 1% lost productivity value, Kats calculated that sick building syndrome reductions in State of California buildings were equivalent to a present value of $37-$55 per square foot of building space over 20 years, compared to average energy costs of $1.47 per square foot for state buildings. The avoidable cost estimate of $9 billion for California is much larger than the estimate derived from Fisk because it includes employee benefits to reflect the workers' full market value, and it reflects the higher percentage of office workers and the higher salaries in California compared to the U.S.

For estimating the costs of sick building syndrome in California's school and office buildings, we use the more comprehensive and California-specific approach of Kats. We modified his estimate by using more recent data and conservative estimates of worker benefits costs, as follows. California has 7 million workers in offices or schools, and their average salary is $43,000 per year in 1998 dollars (Kats, 2003). Sick building syndrome symptoms are again conservatively estimated to cause a 2% decrease in worker productivity. Multiplying these values together yields a cost of about $6 billion per year. For U.S. white collar workers in 1999, salary accounted for 72.8% of their total compensation on average, while benefits such as health insurance and retirement accounted for 27.2% (USCB, 2002). Dividing this benefit percentage by the salary percentage yields a ratio of 0.37 as the additional fraction for compensation as benefits. Multiplying 0.37 by the salary estimate of $6 billion yields an estimated overhead cost of $2.2 billion. This is a conservative estimate because it does not include other overhead costs to the employer such as training, equipment, and travel. Combining these salary and benefit cost estimates yields a total cost estimate of $8.2 billion, or $8.5 billion in 2000 dollars, due to the avoidable impacts of sick building syndrome on worker productivity in California.

3.4 SUMMARY OF ESTIMATED COSTS

The combined cost of both fatal and non-fatal impacts due to indoor air pollution in California homes, schools, and non-industrial workplaces is substantial; it is estimated at **$45 billion per year**, as shown in Table 3.6 below. The annual valuation of premature death attributable to indoor air pollution is estimated to total about $36 billion. The costs attributed to ETS and radon dominate the total cost. However, this is not because they are necessarily the predominant health hazards from indoor air in California, but because those are the two pollutants out of many indoor air pollutants for which there is sufficient data to estimate risk and costs. The cost estimates in this report do not include other indoor air pollutants that can increase the risk of premature death, and any synergistic affects among indoor and outdoor pollutants. Examples of these other pollutants include: PM from wood smoke, other toxic substances emitted from materials and products, and biological pollutants such as mold, bacteria, pollen, and animal

allergens. Therefore, the actual total valuation of death and disease costs are likely to be even higher than estimated here.

The quantifiable medical costs (direct and some indirect) due to some indoor air pollutants total more than $0.6 billion per year, with a large portion of the costs attributable to mold and other moisture-related allergens and sum of all ETS-related medical costs. Again, this is certainly a very low estimate: the cost estimates for disease do not include of the potential losses due to other indoor allergens, CO poisoning's long-term effects, reduced student performance, lost earnings opportunity, unpaid caregivers, and human suffering. Finally, the cost of reduced worker productivity due to indoor air pollution (sick building syndrome) that could be prevented is estimated to be $8.5 billion per year. As discussed in the next section, case studies have documented that measures to reduce indoor air pollution in homes and schools can have immediate and cost-effective benefits on human health and student performance.

3.5 BENEFITS OF IMPROVING INDOOR AIR QUALITY

Several case studies and demonstration programs have documented the economic, productivity, and health benefits that can accrue by improving IAQ. The following examples and other available examples provide empirical data on the benefits achieved in different types of buildings.

Residential Buildings

The Seattle Healthy Home Program has found that home visits to low-income households by a trained community health worker can reduce asthma symptom significantly and cost-effectively (Takaro *et al.*, 2004; Krieger *et al.*, 2005). This program reduced medical costs for asthma treatment significantly, in both the low-intensity version (one visit over the year) and the high-intensity version (multiple visits over one year). The observed marginal cost savings were $1,316 to $1,849 per patient over four years. These savings do not reflect the reduced number of emergency room visits and the reduced risk of asthma-related deaths.

The "Healthy Neighborhoods Program" of New York State inspected over 45,000 homes for health and safety problems, and intervened where necessary (HUD, 1999; NYS, 1999). Interventions addressed lead-based paint hazards, fire safety, sources and detection of carbon monoxide, and asthma-related conditions. Interventions were generally simple and low cost, such as providing working smoke detectors and batteries, CO detectors, and furnace filters. The program results showed that the visits to each home cost $132 per unit. However, the benefits for lead poisoning prevention, asthma reduction, and burn prevention alone were worth at least $285 per unit, excluding estimated benefits associated with reduced injury, CO poisoning, and fire.

Schools

Office visits for asthma inhaler use dropped by 50% at two elementary schools in San Francisco Unified School District (USD) after the *IAQ Tools For Schools* program was pilot tested (U.S. EPA, 2000a). In addition, fewer asthma episodes occurred, and fewer students brought asthma medications or inhalers to school.

A study of Chicago and Washington, DC schools found that better school facilities can add three to four percentage points to a school's standardized test scores, even after controlling for demographic factors (Schneider, 2002). This and other studies reviewed by Kats (2003) and Fisk (2000) confirm a widely held, common sense perception that the physical quality of the classroom environment greatly affects how well children learn.

Table 3.6. Summary of Estimated Costs of Some Indoor Air Pollution in California

Health End Point	Health Valuation: Premature Death[1-3] ($ Billions/yr)	Medical Cost[2-4] ($ Billions/yr)	Lost Productivity Cost[2,3] ($ Billions/yr)	Total Cost ($ Billions/yr)
CO: poisoning	0.15	<0.001	NA	0.15
VOCs: cancer	0.73	0.011	NA	0.74
ETS: lung cancer	2.4	0.025	NA	2.4
ETS: heart disease	23	0.055	NA	23
ETS: asthma episodes	NA	0.020[5]	NA	0.020
ETS: low birth weight	NA	0.19	NA	0.19
ETS: otitis media	NA	0.019[5]	NA	0.019
Radon: lung cancer	9.5	0.097	NA	9.6
Mold and moisture: asthma and allergies	0.031	0.19[5]	NA	0.22
Sick building syndrome	NA	NA	8.5	8.5
TOTAL[6]	**36**	**0.6**	**8.5**	**45**

1. From Table 3.2.
2. Estimates are based on average or mid-point of incidence rates of mortality and morbidity from previous tables, and estimates of productivity discussed in the text. Values are rounded to two significant figures.
3. Original data were adjusted to year 2000 dollars and year 2000 population, except where noted otherwise in previous tables.
4. From Table 3.3.
5. Includes indirect costs such as lost work days, lost school days, and travel expenses.
6. Totals are rounded to 2 significant figures. These totals are likely low because conservative cost estimates were used, and quantitative information is not readily available for many known impacts of indoor air pollution, such as for indoor PM and many indirect costs of health effects.

An analysis of two school districts in Illinois, one small and one large, found that student attendance improved by 5% after incorporating cost effective indoor air quality improvements – regardless of school district size (Healthy Schools Campaign, 2003).

Clovis USD near Fresno, and Everett USD near Seattle, have used the *IAQ Tools for Schools* program and a rapid complaint response approach using a portable indoor air monitoring kit. Additional staff were not needed to implement the program. his program has quickly paid for

equipment by reducing the number of complaints by up to 60%, and by reducing the cost for environmental consultants (Poytress, 2003; Jefferis, 2004). It has also greatly improved the credibility of the school maintenance program with school staff and parents of students.

Indoor air quality is one of several important indoor environmental factors affecting student performance. In a study of over 8,000 students in third through sixth grade in Fresno, Heschong (2003) investigated the effect of daylight and other indoor environmental quality effects on student test scores over an academic year. As expected, commonly recognized factors such as teacher characteristics, number of computers, or attendance rates were found to be significant in predicting student performance. However, physical characteristics such as indoor air quality, ventilation, acoustics, and especially daylighting were found to be equally significant, if not more significant.

The economic benefits of improved indoor air quality in schools, in terms of improved student productivity and health has not been estimated. The potential benefits of improved productivity, even assuming a slight increase in test scores and intelligence quotients (IQ), could be quite large.

Office Buildings

Recent experiments in office buildings have shown that office worker performance could be significantly increased over the short term by removing common indoor sources of air pollution, such as floor coverings, used supply air filters, and personal computers (Wyon 2004). Alternatively, keeping these pollutant sources in place while increasing the ventilation rate from 1.5-3 liters per second (lps) to 10-30 lps per person (3-6 cfm/person to 20-60 cfm/person) also increased worker performance. These short-term effects on worker performance were demonstrated repeatedly, even at pollutant levels that increased SBS symptoms such as headache and poor concentration but had no measurable effects on the occupants' perception of air quality. These short-term effects have been validated recently in field intervention experiments in call centers in northern Europe and the tropics, and the effects were larger than those found in the laboratory.

The potential benefits of these improvements in workplace indoor air quality are substantial. Economic calculations based on this series of experiments indicate that benefits for improving indoor air quality beyond the minimum level acceptable to visitors would exceed improvement costs by a factor of about 60 (Wargocki and Djukanovic, 2003). Typical payback periods were estimated to be about two years. In another study, the economic benefits of reduced employee sick leave in offices can be achieved by increasing ventilation rates over the minimum recommended rate, as shown in epidemiological research in the U.S. (Milton *et al.*, 2000).

4. EXISTING REGULATIONS, GUIDELINES, AND PRACTICES

Despite the ubiquitous presence of toxic pollutants in the indoor environment, there are no government air quality regulations that are intended to protect the general public in residences, schools, or public buildings. Workplace regulations address indoor air quality, but they are designed for 8-hour exposures of healthy adults, and are not designed to be protective for longer periods nor for some of the more sensitive subgroups of the population, such as children and the elderly. Ambient air quality standards are focused on outdoor air quality, and are not designed to protect indoor air quality. Other regulations, such as California's Proposition 65, and AB 13, which prohibits cigarette smoking in workplaces, are applicable to indoor air quality only in a limited way and do not prevent indoor emissions and exposures.

There are a few examples of government regulations for emissions from specific sources of indoor pollutants that are intended to protect the general public in indoor envronments. In addition, a variety of government agencies and private organizations have established voluntary guidelines and practices that can be applied to indoor environments to assist in the assessment and control of health hazards from air pollutants. The following sections summarize the pertinent regulations, guidelines, and practices for the following categories:

- Indoor and outdoor air quality in general.
- Emission limits for consumer products, appliances, and building materials.
- Building design.
- Building operation and maintenance.

4.1 STANDARDS AND REGULATIONS

4.1.1 Workplace Air Quality Regulations

The California Occupational Safety and Health Program (Cal/OSHA) in the Department of Industrial Relations (DIR) has jurisdiction over most private and public employers and employees in California, with the exception of U.S. government employees. Cal/OSHA has regulatory authority to develop, promulgate, and enforce air pollutant exposure limits, ventilation regulations, and other standards for the workplace that directly impact indoor air quality. The California Occupational Safety and Health Standards Board is the unit within the Cal/OSHA program with authority to adopt standards and regulations to protect workers. Labor Code Section 144.6 requires the Standards Board to adopt standards that "most adequately assures, to the extent feasible, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to a hazard regulated by such standard for the period of his working life."

Under CCR Title 8, Section 5155, the Standards Board sets permissible exposure limits (PELs) and other limits for airborne contaminants. The PELs legally apply to both industrial and non-industrial workplaces. However, they are not sufficient to prevent health impacts for all working individuals, such as individuals with pre-existing heart or respiratory disease, and they do not address possible impacts on those with asthma or reactions by sensitive individuals to low levels of chemicals. The Cal/OSHA PELs are 8-hour exposure limits designed to protect healthy working adults, and may be based in part on technological and economic feasibility considerations (non-health related criteria). These standards are not developed to protect infants, the elderly, or other sensitive groups who may frequent non-industrial workplaces (such as public buildings and retail establishments), nor are they intended to be protective for exposures greater than eight hours per day, five days a week. Additionally, PELS have not been

developed for a number of known indoor air contaminants, and they are not designed to evaluate the health and comfort risks posed by the complex mixtures of pollutants found in modern buildings such as offices, schools, and homes. Generally, lower exposure limits would be necessary to protect the general population.

Both Cal/OSHA and others are aware that PELs do not fully address indoor air quality concerns in all workplaces. Cal/OSHA's Indoor Air Quality Policy and Procedure (C-48) states: "Most complaints about the quality of indoor air arise from employees who work in non-industrial environments...Approaches using traditional industrial hygiene techniques usually demonstrate compliance with 8 CCR section 5155 (PELs) despite the persistence of IAQ complaints from the building occupants." The Hazard Evaluation Section and Information Service (HESIS) within DHS also affirms that non-industrial workers experiencing indoor air quality problems are seldom exposed to contaminant levels approaching PELs.

Cal/OSHA also has regulations concerning the operation and maintenance of HVAC systems, and the control of moisture, vermin, and other sanitation concerns, as discussed later in Section 4.5. In addition, Section 3203, Illness and Injury Prevention Program, requires employers to have written plans for hazard identification, evaluation, and correction, for communication with employees, and for training. Other Cal/OSHA requirements reduce or prevent employee exposures to asbestos (Sections 5208, 1529), lead (Section 1532.1), and environmental tobacco smoke (Labor Code 6404.5, and Section 5148).

4.1.2 Ambient Air Quality Standards

National and State ambient air quality standards (AAQS), established by the U.S. EPA and the ARB, respectively, are developed to protect the general public from the harmful effects of "traditional pollutants" in outdoor air, for specified averaging times (exposure times). California's AAQS are often more protective than the national AAQS. Currently, the State AAQS are under review to ensure that they are protective of sensitive populations, especially infants and children (ARB/OEHHA, 2000). In the absence of indoor air quality standards or guidelines, the AAQS serve as useful guidelines for indoor air quality, because they are based on specified averaging times and incorporate a margin of safety. Outdoor standards for PM are often exceeded in indoor environments, and standards for CO, NO_2, and ozone are sometimes exceeded. National and state AAQS are available at http://www.arb.ca.gov/research/aaqs/aaqs.htm.

4.1.3 Proposition 65

In 1986, California voters approved Proposition 65, the Safe Drinking Water and Toxic Enforcement Act of 1986, an initiative to address concerns about exposure to toxic chemicals. Proposition 65 requires the State to publish a list of chemicals known to cause cancer, birth defects, or other reproductive harm. The list includes approximately 750 chemicals, many of which are additives or ingredients in pesticides, common household products, food, drugs, dyes, solvents, building materials, and other sources found indoors. Businesses are required to provide a "clear and reasonable" warning when their products or actions may result in a release of chemicals above a specified threshold level, so that members of the public are aware they may be exposed to harmful chemicals. Warnings have evolved to include labeling of consumer products, posting signs at the workplace or on new housing, and publishing notices in a newspaper. OEHHA develops numerical guidance levels, known as "safe harbor" levels, for determining whether a warning is necessary. For potential carcinogens, the Proposition 65 "no significant risk level" is one excess case of cancer per 100,000 individuals exposed over a 70-year lifetime; for reproductive toxicants, the "no significant risk level" is one-thousandth of the no

observable effect level (NOEL). Proposition 65 pollutants and safe harbor levels are available on the OEHHA website at http://oehha.ca.gov/prop65.html.

4.1.4 Tobacco Control in the Workplace and Public Buildings

Cigarette smoking is a major source of indoor pollution that is now prohibited in most public buildings in California. Passage of a statewide smoke-free workplace law in 1995 (AB 13, Friedman; Labor Code 6404.5, Section 5148) led to a reduction in smoking by the California population and eliminated smoking at nearly all California indoor workplaces, including restaurants, bars and gaming clubs. This statewide prohibition is primarily enforced at the local level. Cal/OSHA is required to respond to complaints of workplace cigarette smoke after the employer has been found guilty at the local level three times in the previous year. Prior to passage of the statewide law, numerous city and county ordinances had been implemented to restrict cigarette smoking.

The smoke-free workplace law contains fourteen exceptions to the smoking ban, each with additional explanations and stipulations. Exclusions include some hotel/motel guest rooms, hotel/motel lobbies, private meeting rooms, tobacco shops, truck cabs, warehouse facilities, gaming and bingo clubs, bars and taverns until January, 1997, theatrical productions, medical research sites, private residences, patient smoking areas, employee breakrooms, and small businesses. Despite the exclusions, the ban has been very successful in reducing worker exposure to cigarette smoke. In 1999, 93.4% of California's indoor workers reported working in a smoke-free environment, compared to only 45% in 1990 (Gilpin *et al.*, 2001).

The workplace prohibition of smoking has had far reaching benefits. The percentage of Californians with children under the age of 18 who do not allow smoking in the household has increased substantially. In 1994, 63.0% of Californians with children did not allow smoking in the house. By 2001, 77.9% did not allow it (Gilpin *et al.*, 2001). Fewer Californians are smoking as a result of this legislation and the DHS Tobacco Control Program. Smoking rates among California adults have declined from 26% in 1984 to 17% in 2001 (BRFSS, 2001). Californians who still smoke are smoking fewer cigarettes than they did in the past (Gilpin *et al.*, 2001).

4.1.5 Radon In Drinking Water

Radon levels in typical groundwater concentrations pose higher risks than those posed by the other drinking water contaminants that have been subjected to regulation (e.g., disinfection by-products). U.S. EPA was directed under the Safe Drinking Act (as amended in 1996) to attempt to regulate radon in drinking water. Though it has no authority to regulate indoor air radon, which is dominated by the soil gas infiltration, the U.S. EPA recognized that it would be far more cost effective to mitigate indoor air radon. The Safe Drinking Act gave U.S. EPA the latitude to allow higher concentrations of radon in drinking water if efforts were established to reduce indoor air radon to achieve risk reduction *equal to or greater* than the risk reduction that would be achieved by reducing the concentration of radon in drinking water (U.S. EPA, 1996).

The U.S. EPA is in the process of promulgating a maximum contaminant level (MCL) for radon, which is the allowable concentration based only on the contaminant risk in drinking water (U.S. EPA, 2000e). The proposed MCL is 300 pCi/l. An alternative MCL, 4000 pCi/l for states that had multimedia radon mitigation programs, and would allow utilities not to treat radon concentrations between the MCL and AMCL. Multimedia programs would aim to reduce indoor air radon risk, using a combination of approaches allowed in the regulations: public education; testing; training; technical assistance; remediation grants, loan or incentive programs; or other regulatory or non-

regulatory measures. EPA (2000e) plans to make their multimedia program regulations effective in December 2005.

4.1.6 Reference Exposure Levels for Air Toxics

The Air Toxics "Hot Spots" Information and Assessment Act of 1987 (Assembly Bill 2588 as codified in Health and Safety Code Section 44300 *et seq.)* requires stationary sources of air pollutants (e.g., industrial plants) to report the types and quantities of substances their facilities routinely release into the air, ascertain health risks associated with the release, and notify nearby residents of significant risks. An amendment to the statute in 1992, SB 1731 (Calderon), requires OEHHA to prepare and adopt risk assessment guidelines, and requires facilities with a significant risk to prepare and implement risk reduction plans. Under this act, OEHHA develops acute and chronic reference exposure levels (RELs) as guidelines to prevent harm from toxic air pollution (http://www.arb.ca.gov/toxics/toxics.htm).

RELs are used by Cal/EPA agencies as indicators of potential adverse health effects other than cancer. RELs are generally based on the most sensitive adverse health effect reported in the medical and toxicological literature. They are designed to protect the most sensitive subgroups of the population by the inclusion of margins of safety. Because uncertainty factors are incorporated into the REL, an air concentration greater than the REL does not necessarily mean that the exposed public will suffer adverse health impacts. The methodology and studies used to develop the REL health standards are detailed in two documents available on the OEHHA website (http://www.oehha.ca.gov/): the *Air Toxics Hot Spots Program Risk Assessment Guidelines, Part 1: The Determination of Acute Reference Exposure Levels for Airborne Toxicants,* March 1999 and the *Air Toxics Hot Spots Program Risk Assessment Guidelines, Part III: The Determination of Chronic Reference Exposure Levels for Airborne Toxicants, February 2003.*

OEHHA has established chronic RELs for approximately 71 air pollutants. Chronic RELs are indicators of healthy versus potentially unsafe levels resulting from long-term exposure, exposure lasting at least 8% of a lifetime or 12 years and possibly longer. Chronic RELs are intended to indicate levels that will not cause adverse health effects in individuals with high susceptibility for chemical injury as well as identifiable sensitive subpopulations (high-risk individuals). However, chronic RELs may not necessarily be indicative of safe levels in hypersensitive individuals.

Acute RELs are indicators of healthy versus potentially unsafe levels of exposure for short periods of time, such as one hour. Because exposure is for a short time period, acute RELs are always higher than chronic RELs. OEHHA has established acute RELs for 51 chemicals (OEHHA, 2000a). OEHHA recommends that acute RELs be used to evaluate exposures that occur no more frequently than every two weeks in a given year.

In addition to providing an indication of healthful versus potentially unsafe levels outdoors near stationary sources, the acute and chronic RELs are also being used to identify healthful versus unhealthful pollutant levels in indoor air. Formaldehyde is a very common indoor air contaminant that has both cancer and non-cancer health effects. Because of the frequent use of RELs for indoor application, OEHHA developed an additional REL for formaldehyde, an interim REL (IREL) based on an 8-hour exposure period. The IREL established for formaldehyde is 27 ppb. The IREL identifies the level below which irritant effects such as eye, nose, and throat irritation would not be expected to occur during typical daytime (8-hour) occupancy of buildings. Other 8-hour IRELs are not yet available. Chronic RELs have been used in developing building material

emission limits for use by DGS in specifying requirements for new state buildings and furnishings. These are known as Section 01350 requirements, and are discussed in detail in Section 4.3.3.2.

4.2 INDOOR AIR QUALITY GUIDELINES

Various governmental and private organizations issue guidelines to promote healthful indoor environments. Such guidelines cannot be enforced; compliance with the recommended levels is voluntary. Guidelines mentioned here are generally based on health or comfort endpoints.

4.2.1 ARB Indoor Air Quality Guidelines

The ARB has published three indoor air quality guidelines – for formaldehyde, combustion pollutants, and chlorinated hydrocarbons. ARB's indoor air quality guidelines discuss the indoor sources and potential health effects of various pollutants, and provide information to the public on ways to limit or reduce their exposure to those pollutants. The guideline levels were developed in consultation with DHS and OEHHA, and are set to protect sensitive subgroups of the population. Because a number of the chemicals covered in the guidelines may cause cancer or other significant health problems, and no absolutely safe levels have been identified, recommendations are made to take action to prevent emissions of those chemicals in the home and to reduce exposure to the greatest extent feasible (ARB, 1991; ARB, 1994b; ARB, 2001b; ARB, 2004). Guidelines for traditional pollutants are based on the ambient air quality standards for California, because the basis of those standards is the health impacts seen at different air concentrations for specified exposure durations, which would be the same regardless of whether the individual is standing indoors or outdoors. An indoor air quality guideline for ozone is under development and is expected to be released in 2005.

4.2.2 Air Quality Guidelines For Europe

"Air Quality Guidelines for Europe" were developed by the World Health Organization's (WHO, 2000) Regional Office for Europe, a specialized agency of the United Nations. The guideline value indicates a concentration of air pollutant below which no adverse effect to human health is expected, based on consideration of both carcinogenic and other health effects. They are intended to be applicable to both indoor and outdoor air exposures. Guideline values have been established for 38 non-carcinogenic compounds and some carcinogens for various exposure periods ranging from 15 minutes to one year. They address public health and are intended to be protective of the entire population. Uncertainty factors are used to extrapolate from animals to humans and from a small group of individuals to a large population. These factors are based on experience, wisdom, and judgement.

The latest edition of the WHO guidelines is more recent than the ARB indoor air quality guidelines. Some of the WHO guideline levels differ from the ARB guideline levels in part because they are based on more recent studies. For example, the current WHO NO_2 guideline is 100 ppb for 1 hour and 20 ppb for an annual average, while ARB's guideline is 250 ppb for 1 hour (a guideline for an annual average is not included). The ARB guideline coincides with the ARB ambient air quality standard for NO_2, currently under review. If a new California standard is adopted, the guideline value may better align with the more recent WHO guideline.

Table 4.1. ARB Indoor Air Quality Guidelines

Pollutant	Measurement Period	Outdoor Air Quality Standards		ARB Recommended Maximum Indoor Levels
		ARB	U. S. EPA	
Formaldehyde (HCHO)	24-hour 8-hour	—	—	Lowest level feasible to reduce cancer < 27 ppb to avoid acute irritant effects
Carbon Monoxide (CO)	8 hours	9 ppm	9 ppm	9 ppm
	1 hour	20 ppm	35 ppm	20 ppm
Nitrogen Dioxide (NO_2)	24 hours	—	—	0.08 ppm avoid repeated high exposures
	1 hour	0.25 ppm	—	0.25 ppm
Particles – (PM10)*	24 hours	50 µg/m^3	150 µg/m^3	50 µg/m^3
Particles – (PM2.5)*	24 hours	—	65 µg/m^3	65 µg/m^3
Polycyclic Aromatic Hydrocarbons (PAHs)	—	—	—	Lowest levels feasible to avoid cancer risk. Avoid or minimize exposure.
Chlorinated Hydrocarbons, e.g., chloroform Trichloroethylene *p*-dichlorobenzene methylene chloride perchloroethylene methyl chloroform	—	—	—	Lowest levels feasible to avoid cancer risk. Avoid or minimize personal exposures.

4.2.3 DHS Mold Guidelines

The California Department of Health Services through the Division of Environmental and Occupational Disease Control (DEODC) is engaged in many types of activities regarding indoor mold. DEODC staff members provide current information about mold health effects and remediation recommendations through educational outreach to the general public, health care professionals, city and county government staff, and private environmental assessors and mold remediators. Staff members also conduct research to improve understanding of how indoor microbial growth affects human health, especially in sensitive groups such as pregnant women, children, and persons with asthma. DEODC Occupational Health Branch staff conducts health consultations in conjunction with Cal/OSHA to prevent harmful microbial exposures in California workplaces.

DHS Indoor Air Quality (IAQ) Program staff members have developed a website, www.dhs.ca.gov/iaq, that provides information about mold-related health effects and voluntary guidelines for remediation of damp/moldy buildings through fact sheets and links to other good sources of mold-related information. Two fact sheets, "Mold in My Home: What do I do?" and "Mold in My School: What do I do", produced by DEODC staff have been used by other state agencies. The latter was selected by the National Clearinghouse for Educational Facilities to be

distributed to teachers nationwide and included on the Clearinghouse website, www.edfacilities.org. The DHS IAQ website also posts a "Listing of Consultants Offering IAQ Services in California" along with guidance for hiring IAQ consultants. This listing is a service allowing interested individuals to contact consultants in their geographic area. Becasuse California does not license, certify, or endorse mold assessment or remediation professionals, individuals seeking assistance for moldy buildings are encouraged to use these and other reliable web resources to learn about this topic and find experienced consultants.

The DHS IAQ Program also provides staff to address inquiries from the public about mold and other indoor environmental problems through the Indoor Air Quality Telephone Assistance Hotline. Staff also organize and conduct meetings of the California Interagency Working Group on Indoor Air Quality (CIWG-IAQ), with representatives from State, Federal, local, and non-governmental agencies having IAQ oversight or interests. The CIWG-IAQ provides a unique forum for discussion, collaboration, and education on issues of indoor environmental quality, building engineering, and related health effects among the professionals working in the field.

The Toxic Mold Protection Act of 2001 (SB 732 Ortiz; California Health and Safety Code Sections 26100 et seq.) was enacted to address increasing concerns regarding health effects from exposure to indoor molds and to provide Californians with guidelines or standards for the safe and effective removal of molds from buildings. Among other tasks, this statute directs the California Department of Health Services (DHS) to determine the feasibility of identifying permissible exposure limits for indoor molds. DHS scientists have addressed this issue, and their conclusions are included in a report currently undergoing internal review. When approved for public release, this report will be posted to the DHS Indoor Air Quality website.

DEODC staff collaborates with other nationally recognized experts in developing guidance documents for mold assessment or remediation professionals, including:

- American Conference of Governmental Industrial Hygienists. *Bioaerosols: Assessment and Control.* ACGIH. Cincinnati, OH. 1999
- American Industrial Hygiene Association. *Field Guide for the Determination of Biological Contaminants in Environmental Samples,* 2nd Edition. In production. AIHA. Fairfax, VA.

4.2.4 American Society of Heating, Refrigerating, and Air Conditioning Engineers

The American Society of Heating, Refrigerating, and Air Conditioning Engineers (ASHRAE) provides professional guidance on minimum building ventilation rates for human health and comfort. While not regulatory, ASHRAE Standards, especially Standard 62, *Ventilation for Acceptable Indoor Air Quality* (ASHRAE, 2003b), and Standard 55-2004 *Thermal Environmental Conditions for Human Occupancy* (ASHRAE, 2004) are important references for California's ventilation standards and recommended comfort levels for nonresidential buildings. ASHRAE and other groups have also developed guidelines using indoor carbon dioxide concentrations as an indoor air quality indicator, ranging from about 800 to 1200 parts per million (ppm) as a "not to exceed" level.

4.2.5 U.S. EPA's *IAQ Tools For Schools Program*

Although it does not provide indoor pollutant guideline levels, U.S. EPA's *IAQ Tools for Schools Program* (http://www.epa.gov/iaq/schools/) provides schools with information they need to understand IAQ issues, prevent IAQ problems, and assure healthful indoor air quality. The

program uses a team approach to school IEQ management and emphasizes staff and occupant training, communication, and improved routine operation and a district.

In California, U.S. EPA has trained more than 2000 individuals from districts throughout the state. Despite the outreach, awareness and use of the *IAQ Tools for Schools* program among California schools is still relatively low: about 11% of school districts use all or part of the program (ARB/DHS, 2003). U.S. EPA is partnering with organizations of school officials (e.g., ACSA, the Association of California School Administrators, and CASBO, the California Association of School Business Officers) to promote increased use of this program.

California Assembly Concurrent Resolution No. 75 (Chan), enrolled in June 2003, recognizes the significance of school indoor environments to the childhood asthma problem, and "encourages California school districts to implement the *Indoor Air Quality Tools for Schools Program* for the benefit of asthmatic children and for the health, well-being, learning, and productivity of the entire school population".

4.3 EMISSION LIMITS

Prevention or reduction of emissions from pollutant sources is the preferred control technique for most pollutants. This section will discuss known regulations, guidelines, and practices for limiting emissions from consumer products, appliances, and building materials.

4.3.1 Consumer Products

4.3.1.1 Government Regulations For Consumer Products

Consumer Product Safety Commission (CPSC)

The federal Consumer Product Safety Commission (CPSC) has broad jurisdiction over consumer products, which are defined as any articles which are produced or distributed for sale to, or use by, consumers in or around the home or in schools, recreation areas, or other non-occupational settings. Certain products are exempted from CPSC authority, including pesticides, cosmetics, tobacco and cigarettes, food, drugs, automobiles, airplanes and firearms. In addition, houses and other buildings are specifically excluded by legislative history and judicial review. However, CPSC does have jurisdiction over home building materials that are sold or distributed as separate products to consumers.

CPSC has the authority under certain conditions to: (1) ban a product; (2) establish mandatory safety standards for products; (3) recall products for repair, replacement or refund: (4) mandate warning labels for products; and (5) cooperate with manufacturers in the development of voluntary product standards. The Federal Hazardous Substances Act (FHSA), enacted in 1960 and administered by CPSC requires labeling of "hazardous substances" if they are "intended, or packaged in a form suitable, for use in the household or by children". A hazardous substance is further broadly defined as a "toxic" substance that may cause substantial personal injury or illnesses through reasonably foreseeable or customary use. In 1992, a supplemental definition was added to the Act to require appropriate labeling if a substance has chronic hazards of cancer, neurotoxicity, and developmental or reproductive toxicity.

CPSC has made a few notable contributions in areas related to IAQ regulation and research. Indoor pollutants regulated to some extent by CPSC through product bans or labeling requirements include asbestos, vinyl chloride, and combustion pollutants from appliances. However, voluntary product standards, rather than product bans and mandatory standards, are

generally preferred by CPSC for several reasons. Voluntary standards take less time to develop and implement and are less likely to be challenged legally, so that public protection should occur sooner; they tend to be less intrusive, more responsive to technological change, and cheaper for all concerned.

The CPSC has focused on regulations for safety more than for health. For example, a policy on methylene chloride led to labeling regulations rather than a ban on its use in household products (CPSC, 1987a). The mandate of the California Department of Consumer Affairs (DCA) differs from that of the CPSC in that the DCA does not regulate consumer products directly, but rather regulates services provided to consumers and those who provide them, and addresses consumer complaints.

ARB Consumer Products and Architectural Coating Programs

The focus of ARB's Consumer Products and Architectural Coating Programs is to reduce the amount of reactive volatile organic compounds (VOCs) that are emitted from the use of consumer products and architectural coatings. The driving force behind the state and local regulations developed under these programs is not reducing exposure to VOCs in indoor air, but limiting the release of VOCs that have the potential to react with other pollutants under sunlight to form ground-level ozone and particulate matter (PM10), the main ingredients in smog. As a result of these regulations, indoor air quality has improved in some situations where these products are used. For example, ARB prohibited the use of three TACs, perchloroethylene, methylene chloride, and trichloroethylene in 13 categories including general purpose degreasers, brake cleaners, all spray paints, all aerosol adhesives and adhesive removers. Additionally, antiperspirants and deodorants are not allowed to contain any compounds identified as TACs. Overall, these prohibitions have reduced TAC emissions in consumer products by over 10 tons per day (tpd). ARB performs an annual survey to determine remaining emissions of perchloroethylene and methylene chloride used in consumer products. The purpose of the survey is to determine if further mitigation measures are necessary to reduce potential health risks from outdoor exposures.

State air pollution regulations define "consumer product" as a chemically formulated product used by household and institutional consumers, including, but not limited to, detergents; cleaning compounds; polishes; floor finishes; cosmetics; personal care products; home, lawn, and garden products; disinfectants; sanitizers; aerosol paints; and automotive specialty products. Excluded from this definition are architectural coatings such as other paint products and furniture coatings. The VOC compositions of these excluded products are regulated by the local air pollution control and air quality management districts (districts), although the ARB provides considerable technical assistance to the districts and develops Architectural Coatings Suggested Control Measures (i.e., model rules) for adoption by the local districts.

The California Clean Air Act (CCAA) enacted by the Legislature in 1988 added Section 41712 to the California Health and Safety Code (HSC), which requires the ARB to adopt regulations to achieve the maximum feasible reduction in reactive organic compounds emitted by consumer products. In enacting Section 41712, the Legislature gave the ARB clear new authority to control emissions from consumer products, an area that had previously been subject to very few air pollution control regulations.

To date, ARB has adopted the following regulations to fulfill the requirements of the California Clean Air Act as it pertains to consumer products:

- Antiperspirants and Deodorants Regulation

- Consumer Products Regulations
- Alternative Control Plan
- Aerosol Coating Products Regulation
- Hairspray Credit Program Regulation

Manufacturers are given flexibility in their approach to meeting the regulations. They can modify their product formulas to reduce VOC content or employ innovative products that may actually increase the amount of "active ingredients" and change the dispenser to lower the amount of VOC emitted per application. An alternative control plan allows manufacturers to average their emissions from noncomplying products with those from products that more than meet the standard. Variances provide temporary relief from the VOC limits in a product regulation if a company cannot comply for reasons beyond their control.

Although each consumer product may seem to be a small source of emissions, the cumulative use of these products by over 35 million Californians results in significant emissions. Consumer products accounted for approximately 267 tons per day (tpd) of VOC emissions in the year 2000, which comprised about 8% of the total man-made VOC emissions statewide. In 2005, without ARB's actions to control VOC emissions in consumer products those emissions would exceed 400 tpd. Even with significant reductions from control measures adopted by ARB factored in, due to population growth, consumer products emissions are projected to total 260 tpd by 2010, and at that time make up about 12% of the VOC emissions projected to be emitted. Further reductions in VOC emissions from consumer products and other VOC sources are needed if ozone standards are to be achieved.

4.3.1.2 Government Guidelines for Consumer Products

A statewide sustainable building program can promote sustainable building concepts and challenge manufacturers to produce more environmentally friendly products. Recognizing this opportunity, Governor Davis issued Executive Order D-16-00 in August 2000. This Executive Order directed the Secretary of the State and Consumer Services Agency to develop a strategy for how the State could design, build, operate, and maintain buildings that are models of efficiency while providing healthy indoor environments. As part of that strategy, the Department of General Services (DGS) directed the development of specifications for environmentally preferable janitorial products for the cleaning and maintenance of state-owned buildings. Green Seal, a product certification organization, has recently developed a standard for cleaning materials (GS-37) that relies primarily on chemical content. The specification reduces emissions to the general work area, and reduces exposures to the individuals using the products. The specification is currently under revision to provide greater health protection. These specifications can result in reduced exposure to cleaning chemicals; however, the use of these products in state-owned buildings is voluntary.

4.3.2 Appliances

Indoor appliances that burn fuel (combustion appliances) or heat food but lack a venting system to remove combustion by-products, moisture, and odors, such as kerosene heaters, unvented gas logs, and unvented gas stoves, can be a major source of indoor air pollution. Wood stoves and fireplaces that burn gas or wood are nearly always vented by design, but they can also be a source of indoor air pollution due to leakage, re-entrainment of air pollutant emissions, or blockage of the flue. Building standards also require gas furnaces and gas water heaters to be vented, but the emissions of these appliances can sometimes leak or be drawn into a building due to blocked or malfunctioning vent systems.

4.3.2.1 Government Regulations for Appliances

The design of combustion appliances is usually regulated through product standards, outdoor air pollution regulations, and indirectly through energy efficiency standards for appliances and buildings. The installation of combustion appliances in California is usually regulated through building standards that are enforced by local building officials, and through local air quality regulations that are implemented by local air pollution control districts. However, the performance testing or inspection of combustion appliances in California homes is not regulated unless the home is participating in a low-income weatherization (energy efficiency) program managed or approved by the State. These programs use a performance test method entitled the Combustion Appliance Safety protocol, or a modified version of it. Therefore, inspection of vented and unvented combustion appliances in California homes occurs mainly during home construction, major remodeling, and resale, but there are no emission or indoor air quality testing regulations for California homes unless the home is participating in certain weatherization programs.

Unvented Appliances

The sale of unvented combustion heaters designed for residential use, such as kerosene heaters, decorative gas logs and fireplaces, and gas-fired space heaters, has been banned in California since the 1980s (HSC Sec. 19881[a]). These appliances are still marketed for other uses such as workshops and barns, and are marketed by out-of-state sellers via mail order and the Internet. It is not clear how effective this regulation has been in preventing the residential use of unvented kerosene heaters, since 1 to 3% of California households use them as primary or secondary heat sources (Phillips *et al.*, 1990).

This regulation was amended in 1997 to allow the sale and indoor use of "natural gas fueled unvented decorative gas logs and fireplaces," provided a series of steps were completed (SB 798, Haynes, HSC 19881[b-c]). The first step, the development of recommended standards by DHS and the Department of Housing and Community Development, was begun by funding a contractor study of the indoor air quality and health impacts of unvented gas logs. The contractor recommendations for unvented gas log standards included restrictions on emission rates for NO_2 and CO, with adjustments for different house volumes (Traynor, 1999). DHS recommended not setting a California standard for these appliances, and the manufacturers of unvented gas logs agreed with this approach. The sale of these devices is thus still banned under current regulations.

Cooking appliances that are unvented, especially gas-fired models, can also produce significant emissions of indoor air pollutants and water vapor, but these emissions are generally not regulated by any government agency. In addition, California regulations do not require cook stoves or ovens to have exhaust ventilation, nor do they limit the flow rate of the range hood to avoid excessive depressurization. The State Building Standard only requires that the range hood be able to supply the ventilation that is required by the stove manufacturer, but installation ofa range hood or testing of the actual air flow rate for the installed hood is not required.

Residential building standards in some states have required that both electric and gas cook stoves have vented range hoods (State of Washington, 2003; MDC, 2004). In California, local building departments may adopt different building standards for the design and installation of appliances, as long as the local standards are at least equivalent to the state building standard code in performance, safety, and the protection of life and health (HSC 17951[d]2). For example, in its housing rehabilitation program, the City of Hayward (2002) requires range hoods to be exhausted to the outside.

Vented Appliances

Most of the 35 California air districts have rules governing outdoor emissions from vented gas appliances such as water heaters and residential central furnaces (ARB, 2003). Most of these rules require certification that natural gas-fired water heaters and central fan-type gas furnaces sold or installed in the district meet the 2002 emission limitations for NOx in South Coast Air Quality Management District's Rule 1121 (SCAQMD, 1999). This rule limits NOx emission to no more than 40 ng/J from gas water heaters and gas furnaces (the gas furnace rule does not apply to models for mobile homes). However, the SCAQMD emission limits for gas water heaters in homes will be lowered in 2005 to 10 ng/J of NOx. This reduction in NOx also reduces the potential magnitude of indoor pollutant exposure when water heater emissions enter a home due to backdrafting.

Outdoor emissions from wood burning devices have come under increasing control by local air pollution control districts in California. For example, in the Northern Sonoma County and San Luis Obispo County Air Pollution Control Districts, PM emissions from non-catalytic and catalytic wood fired appliances must be less than or equal to 7.5 g/hr and 4.1 g/hr, respectively. The districts also require U.S. EPA-Certified Phase II wood burning devices for new sales and installations (40 CFR, Part 60, Subpart AAA, Section 60.530 through 60.539b). State and federal regulations do not directly address the operation and maintenance of woodstoves to ensure that catalysts, door seals, and flue connections are operating properly.

Recently the San Joaquin Valley Unified Air Pollution Control District adopted fairly stringent regulations for fireplaces and woodstoves. These regulations: 1) ban the installation of fireplaces in most new residential development, 2) require U.S. EPA Phase II wood stoves in any sale, supply, transfer, and installation, and 3) require the removal of older woodstoves, with the option to install U.S. EPA Phase II or pellet-burning wood stoves, in any real estate sale or transfer (SJVUAPCD, 2003). The district can also curtail wood burning in the region when outdoor PM levels are predicted to exceed an Air Quality Index level of 150.

In the San Francisco Bay area, 24 cities have ordinances that prohibit conventional fireplaces in new construction. The mountain town of Truckee has a more aggressive policy that states that existing unapproved wood burning appliances must be removed from all properties by July 15, 2006.

California's energy efficiency standards for new home construction have provisions aimed to ensure proper venting and reduce indoor emissions. These standards require vented fireplaces, decorative gas appliances, and gas logs to be installed with closable doors over the opening, a combustion air intake for outside air, and a flue damper with a readily accessible control (CCR, Title 24, Pt. 6, Sec. 150[e] http://www.energy.ca.gov/title24/residential_manual/res_manual_chapter2.PDF). In addition, gas-fired logs, lighters, or decorative appliances must have a flue damper that is permanently blocked open, as required by the California Mechanical Code or the manufacturer. Emissions from gas fireplaces are not regulated in California or the U.S. However, Canada has recently adopted an energy efficiency standard for both vented and unvented gas fireplaces. This standard is based on an industry consensus standard of the Canadian Gas Association (NRCan, 2004a,b).

Home Weatherization Programs and the Combustion Appliance Safety (CAS) Test

The California Department of Community Services Development (DCSD, 2003) manages the State Low-Income Home Weatherization Program, which provides insulation and air leak sealing for low-income homes. The program also requires completion of the Combustion

Appliance Safety (CAS) test and appliance inspection before and after weatherization is completed, and cleaning and adjustment of the gas appliances to meet program specifications. The CAS test protocol was adopted to improve the health and safety of program participants, and to avoid unnecessary liability for contractors and government agencies. It is required by state weatherization programs in California and many other states.

The CAS test protocol specifications in California include the following:

- CO levels in the flue of the furnace and water heater cannot exceed 100 ppm, 100 ppm above the cooktop, or 225 ppm above the oven vent for both the oven and broiler burner. Cleaning, adjustment, and repair or replacement are required to bring the appliances within these limits.
- Clothes dryers must be vented outside with metal ductwork, and CO must not exceed 100 ppm in the exhaust vent.
- The flue draft pressures in furnaces and water heaters appliances must meet certain minimums
- House depressurization cannot exceed limits that can cause backdrafting of emissions from vented combustion appliances.
- Indoor CO cannot exceed 9 ppm in the room.
- An operable exhaust fan is required in the cooking area.

The California Pubic Utilities Commission has recently allowed investor-owned utilities to use a modified CAS for their low income weatherization programs (CPUC, 2003). For example, flue measurements of CO and installation of CO alarms are not required, while CO concentration in room air must be measured. If CO levels in a particular room (or rooms) are 10 ppm or more above nearrby outdoor levels, then the utility may (but is not required to) conduct flue tests as a diagnostic tool to identify the source(s) of the problem. If the incremental indoor CO levels reach or exceed a 35 ppm threshold, the home must be ventilated, the occupants advised to evacuate, and the offending appliance made inoperable pending repair/replacement. CPUC will issue its final decision on testing requirements in early 2005.

4.3.2.2 Government Guidelines for Appliances

ARB's (1994b) Indoor Air Quality Guideline, "Combustion Pollutants in Your Home", recommends actions to reduce combustion pollutants in homes, including emissions from combustion appliances. These actions include removing unvented combustion sources from the home, using and maintaining vented combustion appliances properly, and ensuring adequate and balanced ventilation for the whole house. Also recommended are annual inspections and adjustments to combustion appliances, installation of CO alarms, and testing of combustion appliance safety. This guideline has been widely distributed in California and other states, especially through low-income weatherization groups, of which many are community-based organizations.

ARB (1997b) has also published a "Woodburning Handbook", which recommends measures for reducing emissions and exposures from residential woodburning. These measures include using a gas-fired furnace or gas fireplace insert instead, increasing the efficiency of the house and wood stove, and improving the operation and maintenance of the wood stove. This handbook has been widely distributed in California, mainly through local air pollution control districts.

4.3.2.3 Professional and Industry Guidelines and Practices for Appliances

Combustion Appliances

Groups of high performance home builders and remodelers currently recommend using the CAS test protocol discussed above to prevent indoor air quality and moisture problems from combustion appliances. They also recommend providing exhaust ventilation for gas cooking appliances, and using sealed combustion or power-vented gas appliances that are immune to backdrafting. Examples of such building groups are the Energy Efficient Building Association (EEBA, 2003), the California Building Performance Contractor Association (CBPCA, 2003a,b), the U.S. Department of Energy's Build America program (BSC, 2003), and the National Association of Home Builders Green Building Program (NAHB, 2004).

Manufacturers of gas appliances currently test gas stoves, cooktops, furnaces, and other gas appliances using the industry-promulgated test standards and CO emission limits of the American National Standards Institute Standards Committee Z21/83. The ANSI standards for CO emissions from gas stoves can allow excessive levels of indoor air pollution to build up in homes when gas stoves are used for extended periods without adequate venting to the outside (Tsongas and Hager, 1994; Tsongas, 1995). ANSI is developing a test method for NO_2 emissions from gas stoves (Traynor, 1999).

For testing and assessing gas stoves installed in homes, "No industry standards or uniform government requirements currently exist..." (American Gas Association [AGA], 2003). Some, California utilities have been using the CAS test protocol to test existing installations (Hosler, 1998; see discussion of Weatherization programs above). For installing new stoves, the gas industry has adopted standards, but it is not clear how those standards prevent indoor air quality problems, how well they are incorporated into the State building standard, and how well contractors and homeowners comply with any such standards.

Carbon Monoxide Alarms

The standards organizations of the United States (Underwriter's Laboratory) and Canada (Canadian Standards Association International) have coordinated the writing of CO standards and product testing for CO alarms. The standards currently prohibit showing CO levels of less than 30 ppm on digital displays of alarms. The new standards also require the alarm to sound at higher levels of CO than with previous editions of the standard. New CO alarms will not sound at CO concentrations up to 70 ppm, concentrations that are significantly in excess of the health-based guidelines from ARB, WHO, and other groups. The reasoning behind these changes is to reduce calls to fire stations, utilities and emergency response teams when the levels of CO are not life-threatening. The CO alarm standards are not designed to protect the public from expsoure to low-level, long-term exposures to CO, or from brief exposures to CO emissions from vehicle traffic or properly operating combustion appliances (UL, 2002).

Detectors with a digital display and a history option can provide the true CO concentrations in a house. A low-level display would be useful for people with existing respiratory problems or for those who like to spot evolving problems, rather than having to wait for the situation to become serious. Low-level CO detection products are becoming commercially available. They will not be certified to CSA or UL standards, as these standards currently prohibit low level displays.

Air Cleaners

The American Home Appliance Manufacturers (AHAM, 2004) has developed test protocols to certify the performance of room air cleaners in removing tobacco smoke particles, pollen, and dust. Testing is conducted by a third-party independent laboratory, and the test results are

published. Certified air cleaners must also meet an ANSI/Underwriter Laboratories safety standard for ozone emissions of 100 ppb ozone. Unlike ozone generators, most models of room air cleaners are not designed to intentionally emit large quantities of ozone; however, ionizers and electrostatic precipitators can emit ozone as a by-product. The performance of air cleaners is also evaluated by consumer research organizations (Consumers Union, 2004a).

Vacuum Cleaners

Vacuum cleaners can be an essential tool in controlling indoor pollutants that accumulate on interior surfaces. In recent years, vacuum cleaners have begun to include features such as high efficiency particle attenuation (HEPA) filters and electronic dust sensors that are advertised to improve the removal of allergens and dust from floors and furnishings (Consumers Union, 2003, 2004b).

To date, there are no industry consensus standards for testing and performance of vacuum cleaners. However, the Carpet and Rug Institute (CRI), discussed later in Section 4.3.3.3, has developed a voluntary "Green Label Vacuum Cleaner" program that includes tests for soil removal, dust containment, and carpet appearance retention. CRI provides a list of vacuum cleaner models that have passed these tests and received the Green Label on their website at http://www.carpet-rug.com/. CRI also encourages the proper use and maintenance of vacuum cleaners.

4.3.3 Building Materials

Conventional building materials such as vinyl flooring, carpet, paint, cabinets, and composite wood products can be strong indoor sources of chemical pollutants with a major impact on human health. In the absence of clear regulatory authority, governmental purchasing guidelines are being pursued to recommend low-emitting products in new construction. Also, certain segments of industry have initiated voluntary programs to reduce product emissions, and label their products accordingly.

4.3.3.1 Government Regulations for Building Materials

HUD Formaldehyde Emissions in Mobile Homes

The U.S. Department of Housing and Urban Development (HUD) has set limits for formaldehyde emissions from plywood and particleboard used in mobile homes. HUD has several mandated responsibilities that directly involve indoor air quality, although its basic mission is to provide adequate housing, promote community and economic development of urban areas, and eliminate discrimination in housing markets. In 1984, HUD established formaldehyde emission standards for plywood and particleboard used in mobile homes: test chamber concentrations are not to exceed 0.2 ppm and 0.3 ppm, respectively, to maintain indoor air concentrations of formaldehyde in mobile homes below 0.4 ppm [24 CFR 3280.309(a)]. CFR Title 24 Section 3280.309 also states that each new manufactured home shall have a health notice on formaldehyde emissions prominently displayed in the kitchen. The HUD mobile home standards preempt any existing or future standards of state or local governments that apply to the same aspect of mobile home performance (Sec. 604 [d]).

ARB Composite Wood Control Measures

An air toxic control measure (ATCM) is under development by the ARB to reduce the release of formaldehyde from composite wood products such as particleboard, medium density fiberboard, hardwood plywood, and composite veneer. The adhesive binding systems used to manufacture these composite products contain urea-formaldehyde resins, which release formaldehyde to the

air. As discussed earlier, these resins can be a strong source of formaldehyde emissions (Kelly *et al.*, 1999; Hodgson *et al.*, 2002). It is estimated that formaldehyde emissions from composite building materials could exceed 400 tons per year in California (ARB, 2001c).

4.3.3.2 Government Guidelines for Building Materials

DHS Non-Binding Guidelines

Reducing Occupant Exposure to Volatile Organic Compounds from Office Building Construction Materials: Non-Binding Guidelines was developed by DHS (1996), in response to California legislation (Ch. 1229, Statutes of 1990). The guidelines present a simple technical approach for evaluating, selecting, and installing building materials in order to minimize occupant exposures to VOCs emitted from the materials in newly constructed or remodeled office buildings. The guidelines recommend a five-step approach to reducing exposure to VOCs:

1. Evaluate and select low-VOC impact building materials and products.
2. Pre-condition certain materials to minimize VOC emissions after installation.
3. Install building materials and products based on their VOC emission decay rates.
4. Ventilate a building during and after installation of new materials and products.
5. Delay occupancy until VOC concentrations have been reduced adequately.

The basic principles in these guidelines have been expanded more recently in manuals that describe "best practices" for assuring good indoor environmental quality, as described below

Sustainable Buildings and Section 01350

Several state agencies collaborate on task forces to develop guidelines for "green" buildings, also called sustainable buildings. Components of sustainable buildings include materials for interior surfaces and furnishings that are protective of indoor air quality and health, cost-effective, durable, recyclable, and contain recycled content. Under the direction of the Secretary of the State and Consumer Services Agency (SCSA), interagency task forces have been established for Sustainable Buildings (Executive Order D-16-00) and Environmentally Preferable Purchasing (AB 498, Chan, 2002; Public Contract Code Section 12400-12404), to develop and implement guidelines for construction, renovation, and operation of state buildings, and procurement of state purchases. Stakeholders include members from 40 governmental agencies including ARB, DHS, OEHHA, Integrated Waste Management Board (CIWMB), Department of General Services (DGS), Division of State Architect (DSA), and the California Energy Commission.

In 1999, the Legislature directed DGS to incorporate sustainable building measures into the design and construction of a $392 million State office building complex in Sacramento known as the Capitol Area East End Complex. A product of the effort is *Special Environmental Requirements* (Section 01350; State of California, 2002), which provided protocols for testing of emissions of VOCs from building materials and furnishings to protect human health in state buildings (http://ciwmb.ca.gov/GreenBuilding/Specs/Section01350/). Manufacturers can have their products tested at independent labs using the test protocol developed for use at the Capitol Area East End Complex. Under Section 01350, primary emissions from a single material or product are modeled to estimate a room concentration; secondary emissions resulting from chemical reactions are not considered. The calculated room concentration cannot exceed one half the health-based chronic REL (developed by OEHHA for toxic air contaminants). Formaldehyde is an exception; emissions from a single product cannot exceed one half the interim 8-hour REL for formaldehyde, as discussed elsewhere in this document. The RELs have

undergone extensive public comments and peer review by the State's Scientific Review Panel, so they are appropriate for use as an indoor guideline for this purpose.

Section 01350 has been incorporated into the DGS standard agreement for engineering and architectural services. It is also used in *Reference Specifications for Energy and Resource Efficiency* (CEC, 2004) and the *Collaborative for High Performance Schools (CHPS) Best Practices Manual* (CHPS, 2001). Section 01350 has been expanded to include additional details for broader applications such as schools, and products used in state buildings. Most recently, DHS has published their "Practice for Testing of VOCs from Building Materials Using Small Chambers" (http://www.dhs.ca.gov/ehlb/IAQ/VOCS/Practice.htm), which updates the indoor air quality portions of Section 01350 .

Collaborative For High Performance Schools

The Collaborative for High Performance Schools (CHPS) is a California consortium of public agencies and energy utilities working to facilitate the design and construction of "high performance" schools. These are school facilities that aim to be models of energy and resource efficiency, as well as healthy and comfortable settings supporting quality education. CHPS uses a whole-building design approach, as well as providing designers with specific guidance on component systems, that incorporates the best of current knowledge and technologies. CHPS developed their own grading criteria using a point system, similar to the U.S. Green Building Council's Leadership in Energy & Environmental Design (LEED™) rating system (U.S. Green Building Council, 2004).

The CHPS Criteria include prerequisites and optional measures for low- or no-emission building materials, furnishings, and cleaning practices. Construction practices to minimize indoor pollutant buildup or dispersion are also required. The building materials requirements are based on the Section 01350 specifications discussed above, with adaptations for classroom conditions.

Environmentally Preferable Products for School Construction

As required under SB 373 (Chapter 926, Statutes of 2001), the Division of the State Architect (DSA, 2004) is developing a first of its kind database of environmentally preferable products (EPP) for use in school construction. SB 373 defines an environmentally preferable product as a product that promotes healthy indoor environments for children, and demonstrates the use of environmentally preferable materials and systems. When compared to other similar products with similar functions, an environmentally preferable product has some, or all, of the following characteristics relative to those similar products serving similar functions:

- Less hazardous to public health, safety and the environment.
- Consumes less energy in their manufacture or use.
- Contains more, or any amount of, recycled or post-consumer material content in their manufacture.
- Results in less potential waste.
- Results in less harm to indoor air quality.
- Consumes less water.
- Includes features, or is manufactured from materials, that promotes recycling or reuse of the product.

Development of the database is anticipated to include the following basic phases: research, criteria development, product screening, and database publishing. Products will be screened against a comprehensive set of environmental, health, and performance criteria. The indoor air

quality criteria will be based on the requirements of Section 01350, discussed above. A research report looking at existing programs and resources pertinent to this project was completed in February, 2004 and posted on the project website (http://www.eppbuildingproducts.org/). Between April and July 2004, draft standards for composite panels, gypsum board, and fiber-based insulation were released for public comment and a series of public workshops was initiated. However, the Department of General Services has temporarily halted the project and is examining a potential change in the project approach, in order to ensure public participation. The criteria will be developed by working groups of state, federal, and local government, private and non-profit organizations and manufacturer representatives.

4.3.3.3 Professional and Industry Guidelines and Practices for Building Materials

Several building material industries have developed voluntary emission standards and programs to certify products that meet those standards. The most notable are the composite wood products industry and the carpet and rug industry. These programs have been successful in reducing emissions from their products over the last few decades.

The Composite Panel Association (CPA) is a professional organization for manufacturers of composite wood products. Composite wood products include pressed wood products such as plywood, particleboard, oriented strand board, and medium density fiberboard. The Association, founded in 1960, is dedicated to promoting the benefits of their products and producing products that meet a variety of rigid standards. Of particular note is the Grademark Certification Program that involves third-party certification of formaldehyde emission levels from wood panel products. This program uses the American National Standard Institute (ANSI) test procedures and limits for formaldehyde emissions from wood products.

The ANSI standard for particleboard and medium density fiberboard (MDF) establishes product specifications for dimensional criteria, physical and mechanical criteria, and formaldehyde emissions. In some cases, the industry formaldehyde emissions criteria are more stringent than those established by HUD for mobile homes. Standard test method ASTM E 1333-96, *Determining Formaldehyde Concentrations in Air and Emission Rates from Wood Products Using a Large Chamber,* is used to simulate and measure emissions during normal product use. Products from each manufacturer must pass this test to bear the CPA Formaldehyde Grademark Program stamp. The Hardwood Plywood and Veneer Association (HPVA) also uses the ASTM E 1333 Large Chamber test for product evaluation. For applications for which urea-formaldehyde resin compressed wood products are needed, ARB's guideline entitled *Formaldehyde in the Home* encourages the consumer to use composite wood products bearing the CPA or HPVA certification marks, because those marks confirm that the products meet relevant HUD and ANSI formaldehyde emission standards.

The Carpet and Rug Institute (CRI; http://www.carpet-rug.com/) initiated a voluntary testing program in 1994 as a result of the U.S. EPA's Carpet Policy Dialogue. Carpet samples are tested for chemical emissions by a third party laboratory according to ASTM D 5116 – *Guide for Small-Scale Environmental Chamber Determinations of Organic Emissions from Indoor Materials/Products.* For carpet, emissions limits were established at 0.5 mg/m^2 for TVOC; 0.05 mg/m^2 for 4-PC (4-phenylcyclohexene, the compound most associated with "new carpet odor"); 0.05 mg/m^2 for formaldehyde; and 0.4 mg/m^2 for styrene. Most, but not all, carpet manufacturers participate in the CRI test program. CRI also has emission standards and labeling programs for carpet cushion and adhesive. Products are tested quarterly to assure continued compliance with the test program requirements. Carpets that meet the emission test criteria bear a green and

white CRI Indoor Air Quality Carpet Test Program logo in carpet showrooms so that consumers can identify the lower emitting carpet. Despite this labeling program, new carpet may still have a substantial "new carpet" odor, and extensive ventilation is recommended during installation and immediately after installation.

During the summer of 2004, at DHS's urging, CRI further expanded their voluntary test program with the introduction of Green Label Plus (http://www.carpet-rug.com/News/040614_GLP.cfm), which includes some additional chemicals and emission limits from DHS's Section 01350 test protocols. After a carpet product has been certified to meet the Green Label Plus criteria, it will be tested annually for the emission levels of 13 chemicals, and quarterly for emissions of total volatile organic chemicals (TVOC). The addition of some Section 01350 criteria is a major step toward further reducing carpet assembly emissions and assuring healthy indoor environments in newly carpeted areas, and addresses at least in part concerns that had continued regarding carpet assembly emissions. It is commendable that CRI has taken this action, and it is hoped that CRI will adopt any additional criteria identified in the future as necessary to assure healthful carpet and rug products.

The Greenguard Environmental Institute (GEI) has established performance-based, field validated standards to define products and materials with low chemical and particle emissions for use indoors. The standards establish certification procedures, including test methods, allowable emissions levels, product sample collection and handling, testing type and frequency, and program application processes and acceptance. Currently, over 20,000 different products are listed as certified under the Greenguard low emitting product standards (http://www.greenguard.org). Greenguard certification is a referenced standard incorporated in the U.S. Green Building Council's LEED Program for commercial interiors, and is used by many municipalities and organizations across the U.S.

Greenguard requires products to be tested on an annual basis for over 2,000 individual chemicals including formaldehyde, measured carcinogens and reproductive toxins, as well as the sum of all measured chemicals. The modeled indoor concentratons of most individual VOCs detected must meet the concentration criteria of less than 1/10th of the threshold limit values (TLVs) established by the American Conference of Government Industrial Hygienists. However, as in the case of OSHA's PELs for worker health and safety, using TLV fractions is not protective for non-industrial workplaces and residential environments. In fact, public health groups and ASHRAE (1981, 2003b) have abandoned this approach based on TLVs.

4.4 BUILDING DESIGN AND CONSTRUCTION STANDARDS AND GUIDELINES

Unlike some states, California does not have specific building design and construction standards to address IAQ. Standards for the design and construction of building ventilation systems and appliances in California are located mainly within Title 24 of the State's building energy and ventilation regulations. These standards are promulgated by the California Energy Commission (Commission), and are enforced by local building officials, or in the case of public schools, by certified inspectors and the California Division of the State Architect.

For existing buildings, there are limited regulations or guidelines affecting indoor air quality. The Title 24 standards include requirements for substantial additions and alterations of buildings, including those for HVAC systems. Home weatherization programs operated by the State and some California utilities address the indoor emissions from combustion appliances and the building ventilation system. In addition, the Commission is beginning to consider energy efficiency programs for existing buildings.

Guidelines for IAQ in building design and construction are available for the major building types, usually as an integral part of "green", "healthy", and "sustainable" building programs. In recent years, ARB and other State agencies have worked together, through the State Sustainable Building Task Force and the Collaborative for High Performance Schools, to develop IAQ measures to be used in sustainable buildings for the State and public schools. However, these IAQ measures have not yet been fully incorporated into all state building requirements, and are not currently required in non-State buildings, in schools, or in homes.

4.4.1 State Regulation of Design and Construction

The general mandate of the Commission is to ensure a reliable energy supply in a manner consistent with protecting the State's environment and enhancing its economy. The Commission develops and periodically updates energy efficiency standards for the design and construction of new buildings, appliances, and insulation materials. The Commission does not directly regulate the operation and maintenance of buildings and appliances, factors that are among the most important determinants of IAQ. However, some aspects of the Title 24 standards deal with HVAC controls systems, building operation modes, and providing building operation, maintenance, and design ventilation rate information to building owners and building managers upon occupancy (Sec. 10-03[b]). In addition, the Title 24 standards provide the basis for HVAC operation and maintenance standards developed and enforced by Cal/OSHA.

The Commission is required to routinely address indoor air quality impacts of its energy efficiency standards, under the authority of AB 4655 (Tanner; PRC 25402.8) and the California Environmental Quality Act. The Commission (CEC, 1994) prepared a report to the Legislature that assessed the indoor air quality impacts of its energy efficiency standards (PRC 25402.8). The Commission's standards and other activities pertinent to indoor air quality and building design and construction standards are summarized below.

4.4.1.1 Design Ventilation Standards: Nonresidential, High-Rise Residential, And Hotel Buildings

The California Energy Commission sets minimum ventilation standards for the design and construction of new buildings and building additions in nonresidential buildings. The standards also apply to high-rise residential buildings and hotels. These standards are published under CCR, Title 24, Part 6, and are required to be cost-effective. Local building officials enforce these State standards, but for public school buildings, the California Division of the State Architect (DSA, 2003) reviews the building plans and certifies building inspectors, thereby replacing the local building official function.

Currently, the 2001 edition of the Title 24 standards for nonresidential buildings require that newly constructed nonresidential buildings, and new HVAC systems in additions, be capable of supplying specific minimum ventilation rates for outdoor air. These minimum ventilation rates are typically met by a mechanical ventilation system that includes duct systems, control systems, and air filters. The standards require air balancing of airflows in the HVAC system, or airflow measurements, before occupancy in order to document that minimum ventilation rates are supplied.

The minimum ventilation rate is calculated by two alternative criteria. New HVAC equipment must be designed and installed to be capable of providing no less than the <u>larger</u> of the following:

- The minimum ventilation rate based on the size of the building, expressed in cubic feet per minute (cfm) per square foot of conditioned building floor area. Rates required for different types of building use are shown in Table 4.2.
- The minimum ventilation rate based on the number of occupants: "15 cfm per person times the expected number of occupants." The expected number of occupants may be specified by the designer. However, the expected number of occupants may not be assumed to be less than one half the maximum occupant load for existing purposes in Chapter 10 of the California Building Code.

These Title 24 standards also address the control of the HVAC system, which can markedly affect IAQ. The system and its controls must be capable of: 1) supplying the minimum ventilation rate at all times the building is occupied, and 2) providing one-hour pre-occupancy flushing at full occupancy ventilation rates, or at 3 air changes per hour or more. For spaces with high occupant density and at least 3,000 cfm capacity, such as auditoriums and large meeting rooms, the standards require "demand controlled ventilation" using a CO_2 sensor to achieve at least 0.15 cfm per square foot and maintain indoor CO_2 levels at or below 800 ppm. Such control systems have not been widely used in California, and it is not clear how well this requirement has been implemented. For the upcoming 2005 nonresidential Title 24 standards, the Commission required "acceptance testing" of demand control ventilation systems before occupancy, to ensure that the system performs as designed.

The Title 24 nonresidential standards also include an option for natural ventilation rather than mechanical ventilation. Natural ventilation by windows or roof openings is allowed if the openings are operable, accessible to the occupants, within 20 feet of the room's outer edge, and sized to be greater than 5% of the conditioned floor area (this was recently increased to 25 feet for high-rise residential and hotel/motel guest rooms). Because there is no requirement for anyone to actually open the windows or to avoid nearby sources of outdoor pollutants or noise, the actual ventilation and IAQ provided under this option is highly variable, difficult to quantify, and difficult for the building owner or Cal/OSHA to verify.

4.4.1.2 Design Ventilation Standards: Low-Rise Residential Buildings

Unlike nonresidential buildings, most homes in California do not have outdoor air provided by mechanical ventilation – outdoor air is provided instead via open windows and doors and by unintentional air leakage in the building shell and the HVAC ductwork. In 1978, the Commission adopted residential building design standards that required extensive caulking, weather-stripping, and sealing to reduce air infiltration in new low-rise homes. To avoid IAQ problems for very tight homes in a few climate zones of California, heat recovery ventilators (air-to-air heat exchangers) were required to provide outdoor air ventilation for certain home designs (Maeda, 2004), but very few builders used this option and the requirement was subsequently dropped.

More recent Title 24 standards for low-rise residential buildings require additional reduction of air leakage through the sealing of HVAC ductwork. The sealing of duct work not only reduces natural air infiltration, but also unintentional infiltration of pollutant sources from spaces adjacent to the home, such as crawlspaces, garages, vehicle traffic, and local woodburning. Compliance credit for additional reductions in building air leakage reduction can be obtained by wrapping the building shell with an infiltration barrier, but this measure is optional.

Table 4.2. Minimum Design Ventilation Rates For Nonresidential, High-Rise Residential, and Hotel Buildings, 2001 Title 24 Standards[1]

Type of Use	Cubic Feet per Minute per Square Foot of Conditioned Floor Area
Auto repair workshops	1.50
Barber shops	0.40
Bars, cocktail lounges, and casinos	1.50
Beauty shops	0.40
Coin-operated dry cleaning	0.30
Commercial dry cleaning	0.45
High-rise residential (5 stories or more)	Per CBC Section 1203[2]
Hotel guest rooms (less than 500 sq. ft.)	30 cfm/guest room
Hotel guest rooms (500 sq. ft. or greater)	0.15
Retail stores	0.20
Smoking Lounges	1.50
All others	0.15

1. Adapted from CCR, Title 24, Part 6, Sec. 121(b) 2, http://www.energy.ca.gov/title24/standards/index.html.
2. Specifies minimum opening areas for operable windows, but does not require actual operation of window or maintenance of operability.

To help ensure adequate IAQ, the 2001 Title 24 standards require mechanical ventilation with outdoor air when the target infiltration is below a certain limit. Energy credits for "unusually tight" construction are not allowed unless: 1) air leakage is measured by an approved home energy rater using a blower door test method, 2) additional outdoor air is provided continuously by mechanical ventilation, and 3) combustion appliances such as furnaces and waters heaters that use indoor air for combustion are not installed. The mechanical system may be either an independent system with its own fan, filter, and ductwork, or it may be integrated with a larger central air system. This approach is entitled "Compliance Through Quality Construction" (http://www.energy.ca.gov/title24/residential_manual/res_manual_2001.PDF, p. 129).

A few other states have already required mechanical ventilation systems for new homes. For example, the State of Washington (2003) adopted the Ventilation and Indoor Air Quality Standard as part of its energy efficiency standards for new home design and construction. This standard requires continuous, whole-house ventilation, and exhaust ventilation by low-noise fans in the bathroom and kitchen. In addition, it requires that composite wood products be low-formaldehyde (exterior grade) and that a radon removal system be installed under the foundation. The State of Minnesota (MDC, 2004) has promulgated a state residential ventilation

standard that requires continuous, whole-house ventilation, and exhaust ventilation by low-noise fans in the bathroom and kitchen.

4.4.1.3 Design and Performance Standards for Existing Buildings

The Commission has begun to investigate strategies to achieve peak load reduction in existing buildings (mandated by AB 549, Longville). Such strategies may involve weatherization and duct sealing in existing buildings, which would affect the levels of indoor air pollutants produced by indoor pollutant sources such as combustion appliances and building materials and the levels of indoor moisture produced by human activities and other indoor sources. Strategies under consideration for non-residential buildings could affect and perhaps improve the operation of existing HVAC systems, e.g., retro-commissioning (performance testing) and the increased use of control systems to obtain optimum building system performance. The Commission's recommendations to the Legislature are expected in October 2005.

4.4.1.4 Standards for Home Weatherization Programs

The California Department of Community Services Development administers the federal Low-Income Weatherization Assistance Program through trained and certified contractors (DCSD, 2003; U.S. DOE, 2003a,b). This no-fee program installs insulation, weather-stripping, caulking, duct sealant, furnaces, and air conditioners as needed in low-income homes, both renter- and owner-occupied. To avoid IAQ and ventilation problems, the program contractors use a standard protocol across the state to:

- Limit the tightening of the building shell. This minimum leakage rate is based on modeling of the amount of leakage typically needed to provide 0.35 air changes per hour when averaged over the heating season.
- Inspect and test combustion appliances, venting systems, and ductwork, as discussed above under Section 4.3.2.1, Emissions Limits: Appliances. Testing requirements include measurements of CO emissions, vent draft pressure, and building depressurization using the Combustion Appliance Safety (CAS) test.

4.4.2 Federal Regulations for Building Design and Construction

Federal standards do not currently exist for building design, except for new federal buildings, manufactured housing (mobile homes), and federal public housing. New federal buildings such as office buildings and residential buildings must meet federal design standards for energy efficient design, but federal policy has been to comply with local building standards where they are more stringent. Although California energy efficiency standards have long been more stringent than federal standards, the state and federal building ventilation standards are similar, except that new federal standards for new single family homes include mechanical ventilation. Standards for manufactured housing and HUD-financed public housing are discussed below.

4.4.2.1 Manufactured Housing

Manufactured housing is regulated by HUD, which has developed requirements for low-formaldehyde plywood and particle board (but not medium density plywood), moisture control, exhaust venting, and fresh-air ventilation systems (HUD, 2002). These standards are enforced in California by the Manufactured Housing Section of the Department of Housing and Community Development. The formaldehyde emission requirements for wood products are discussed in Section 4.3.3.

Earlier versions of the HUD standards gave homebuyers the option of the fresh-air ventilation system, but very few buyers in California selected that option. The current standard requires such a whole-house ventilation system and exhaust ventilation for kitchens and bathrooms. The whole-house ventilation system must provide 0.10 air changes per hour (ach), plus an assumed natural infiltration rate of 0.25 ach, for a total of 0.35 ach. However, a large study of newer manufactured hones in the Pacific Northwest found that many homes received much less than 0.35 ach, and that occupants did not use the ventilation systems as they are designed to be used (Lubliner and Gordon, 1990). In addition, some homes with wood fireplaces that have loose operable access doors have been observed to "backdraft" smoke into the homes, due to excess negative pressures caused by the HVAC equipment (Boe, 1999). The U.S. Department of Energy is funding research to improve the energy and indoor air quality performance of manufactured housing (Lubliner and Gordon, 1990).

4.4.2.2 Public Housing

Federal regulations for public housing financed by HUD require that the site characteristics, including the neighborhood and surrounding properties, undergo environmental review. HUD (1996) has provided a guide for this review, which addresses site hazards such as lead, PCBs from transformers, fuels, and solvents that could contaminate the homes during and after construction. At least three public housing sites in the U.S. have been built on known hazardous waste sites. Other housing built by the public or private sector may undergo a similar review through the California Environmental Quality Assurance (CEQA) review of environmental impacts, but the extent of the review for CEQA appears to be much less.

4.4.3 Professional and Industry Guidelines for Design and Construction

Several private and public sector organizations develop recommendations that serve as "consensus standards" or guidelines for achieving good indoor air quality through building design and construction. Many of these guidelines go beyond current regulatory requirements, but some are eventually incorporated into the regulations of government agencies. Concern has sometimes been expressed regarding the validity and effectiveness of such standards in protecting public health and welfare. However, although they may not meet all recognized needs, consensus standards serve an important function by providing guidelines for professionals such as building designers, managers and consultants, often long before government rule-making procedures could provide similar guidance. Government agency staff often actively participate in the development of such consensus standards by serving on standards-development committees of the major standards organizations.

ASHRAE has adopted a number of standards related to IAQ. These standards include those for ventilation and IAQ (Standard 62), air filter performance, thermal comfort, air-to-air heat exchanger performance, and building commissioning. These ASHRAE standards, especially Standard 62, have provided part of the basis for some building standards adopted by agencies such as HUD, CEC, and local governments. Thus, ASHRAE standards and their revisions have played a significant role in the previous and current State regulations for ventilation system design and energy-efficient building design as they relate to indoor air quality. Key guidelines from the public and private sector that address indoor air quality through building design, construction, and commissioning are summarized below.

4.4.3.1 Guidelines for Nonresidential Buildings

ASHRAE Standard 62, Ventilation for Acceptable Indoor Air Quality, was first adopted in 1973. It was based on criteria for odor perception and indoor carbon dioxide concentrations, with a prerequisite that all outdoor and re-circulated air meet State or federal outdoor air quality standards. The Standard included minimum ventilation rates for various types of rooms in nonresidential buildings, and specified higher recommended ventilation rates for areas with smoking or other indoor sources of pollutants present. In 1981, ASHRAE Standard 62-1981 permitted lower ventilation rates in non-smoking areas, and included additional indoor air quality guidelines for several non-criteria pollutants such as formaldehyde and radon. The current version of Standard 62 for nonresidential buildings (ASHRAE, 2003a) specifies ventilation rates for outdoor air to adequately dilute pollutants emitted by occupants, occupant activities, and building materials, furnishings, and systems. For example, 15 cubic feet per minute (cfm) of outdoor air per person is required for most types of building occupants, and increased ventilation rates are specified for building areas with significant sources of indoor air pollution, such as bathrooms, printing and copying rooms, cooking areas, and garages. In addition, an appendix to the standard addresses the amount of ventilation required to reach recommended levels of specific indoor air pollutants. Standard 62-2001 is currently being modified under the continuous maintenance process; addenda to the standard are considered and undergo public review several times yearly.

4.4.3.2 Guidelines for School Buildings

Design guidelines for environmentally sustainable, healthy schools in California have been developed by the Collaborative for High Performance Schools (CHPS). CHPS Board Members include individuals from state agencies, utilities, and building professionals. CHPS (2001) has published the *Best Practices Manual* that describes such design features as selection of low-emitting materials, ventilation system specifications, and IAQ management plans. The *Manual* provides a self-rating certification system and the CHPS criteria for the indoor environment and related building topics. School designers have latitude to incorporate practices in the manner that best fits the district's application. CHPS also provides ongoing training for school facility staff, building designers, engineers, consultants, and manufacturers. At least ten school districts, including Los Angeles Unified School District and San Francisco Unified School District, have adopted CHPS Criteria for new school construction planning. A national version of CHPS has been adopted by a few other states recently. The U.S. EPA (2003b) has recently published "IAQ Design Tools for Schools," which is largely based on the CHPS *Best Practices Manual.*

CHPS recently added an Operations and Maintenance volume to the *Manual,* and plans further updates and additions in 2005. Recently, the U.S. Department of Energy adapted the CHPS *Best Practices Manual* Volume II (Design) for a national audience (U.S. DOE, 2003). Several school districts, notably Los Angeles Unified, have established policies to require all new facilities to meet the CHPS criteria, including those for emission testing of building materials. CHPS manuals are available free of charge on the Internet at http://www.chps.net.

4.4.3.3 Guidelines for Residential Buildings

For residences, ASHRAE Standard 62-73 and subsequent versions minimally addressed ventilation and IAQ. However, ASHRAE recently expanded Standard 62 to include ASHRAE Standard 62.2, which specifically addresses IAQ in low-rise residential buildings (Sherman, 2003a; ASHRAE 2003a). This standard now includes several IAQ features, such as whole-

house mechanical ventilation with outdoor air, "local exhaust" fans in the kitchen and bathrooms, backdraft testing, and carbon monoxide alarms (Sherman 2003b). Ventilation rate requirements are 7.5 cfm/person, based on the need to remove pollutants generated by occupants' indoor activities, plus 3 cfm/100 square feet of floor are, based on the need to remove pollutants generated by background or building-related sources. The standard also refers to Standard 62.1 for specific guidance in providing sufficient ventilation based on the pollutant emissions from the occupant activities and from the building materials and furnishings. This standard is for new home design, but can also be applied to existing homes. It is not a regulatory requirement in California, but it may help provide the basis for future State standards.

Professional home builder and public health groups in California have developed recommended practices for improving the performance of new and existing homes that take a "whole-building approach" to address building materials, ventilation, combustion safety, moisture control, thermal comfort, and energy efficiency. Examples of such groups are the Energy Efficient Building Association (EEBA, 2003), and the California Building Performance Contractors Association (CBPCA, 2003ab). In addition, the National Association of Home Builders (NAHB, 2004) is beginning to develop guidelines for "green" home building that consider the whole building and environmental concerns, and the Canadian Home Builders Association already has a green home building program (CHBA, 2004). The American Lung Association (ALA, 2004) has implemented the Health House Program, that includes builder guidelines, training, and educational information developed with a focus on preventing and reducing asthma and other indoor pollutant hazards. The HUD (1999) Healthy Neighborhood Program has funded several groups across the U.S. to use inspection and intervention to address asthma triggers, CO poisoning, lead poisoning, and other health and safety hazards for children in low-income populations. Numerous similar guidelines and programs have come into existence.

The recommendations from these groups generally include measures such as low-VOC building materials, whole house mechanical ventilation, exhaust fans for high-moisture areas such as bathrooms and kitchens, combustion appliance safety testing, and combustion appliances that are resistant to backdrafting and other venting problems. However, these approaches have not been widely used in California – only a few custom homes or demonstration projects have used these approaches in California so far.

4.4.3.4 Building Commissioning Guidelines

Building commissioning is the process of verifying and documenting that construction, operation, and maintenance of a building meet the design specifications of the project. Ideally, commissioning is conducted by a trained, third-party inspector, begins during the design phase of a project, and includes the development of operation and maintenance procedures and related training. Through commissioning, potential problems are detected early, HVAC systems are precisely tuned, change orders are reduced, building operators are trained, utility and other costs are reduced, and a healthful and comfortable workplace is produced (ODOE, 2004).

ASHRAE (1993, 1996) has published guidelines for commissioning HVAC and control systems in large buildings. The California Commissioning Collaborative (CCC, 2004), which includes government, utility and building services professionals, has been promoting building commissioning through incentives, training, research, and the development of model Requests for Proposals. Research funded by CCC members have found that in order to achieve building performance goals over the lifetime of the building, it is necessary to conduct enhanced training of building operators, to track building performance, and to conduct ongoing commissioning to meet current needs of building occupants (Friedman *et al.*, 2003).

Building commissioning has proven to be very cost-effective in achieving reduced use of energy and other resources, while providing health and safety benefits through improved indoor air quality. The Commission included basic commissioning requirements in earlier Title 24 standards – air balance and flow testing of HVAC systems in nonresidential buildings (residential buildings do not have such requirements). The State of California has recently become more active in the building commissioning area.

- With guidance from the State Sustainable Building Task Force, DGS has begun to incorporate building commissioning in new projects for large office buildings. DGS is also beginning to "re-commission" existing state buildings.
- The Commission included HVAC commissioning requirements for HVAC control systems and major components in the 2001 Title 24 standards.

However, despite these and some related activities, formal and complete building commissioning is still relatively rare in California's nonresidential buildings, and it is still in the methods development stage for residential applications (LBNL, 2003).

4.5 BUILDING OPERATION AND MAINTENANCE STANDARDS

4.5.1 State Standards

The primary building operation and maintenance regulations in California are those of Cal/OSHA, which apply to most industrial and non-industrial workplaces. Non-industrial workplaces include beauty salons, hospitals, dry cleaners, medical laboratories, retail shops, copy shops, and many other workplaces that can at times have elevated levels of air pollutants associated with specific activities. School buildings are also subject to Cal/OSHA regulations, although school maintenance staff are not always aware that these workplace regulations apply. Residential buildings such as nursing and rest homes, daycare centers, and public housing used for special purposes also are subject to Cal/OSHA regulations.

The Cal/OSHA regulations most applicable to indoor air quality are Sections 5142 and 5143 (CCR, Title 8, http://www.dir.ca.gov/samples/search/query.htm). These regulations address the proper operation and maintenance of mechanical ventilation systems. Cal/OSHA enforcement of these standards mainly occurs when a worker files a complaint, and is therefore very limited. Other sections of Title 8, such as those addressing asbestos and lead, take a pro-active approach by requiring worker training, building assessment, and occupant notification.

4.5.1.1 Ventilation Regulations for Workplaces

Employers are required to maintain and operate HVAC systems to provide at least the minimum quantity of outdoor air required by the State Building Code (Title 24) at the time the building permit was issued. Section 5142, *Mechanically Driven Heating, Ventilating and Air Conditioning (HVAC) Systems to Provide Minimum Building Ventilation*, was adopted in 1987 in response to a labor union petition. With limited exceptions, systems must be operated during working hours, unless the employer can document that the outdoor air requirements are being met by non-mechanical means. Employers must perform at least annual inspections, and correct problems found during those inspections. This section also includes record-keeping requirements; employees can obtain these records on request.

Section 5143, *General Requirements of Mechanical Ventilation Systems*, was adopted in 1976. This section requires annual performance measurement of ventilation systems that are used to

prevent harmful exposures, such as local exhaust ventilation systems and laboratory hoods. This section requires contaminant-free make-up air (supply air), regular filter maintenance, and pressure gauge installation to indicate when filters must be cleaned or replaced. This section also has record-keeping requirements.

4.5.1.2 Mold in Workplaces

Cal/OSHA requires that workplaces be maintained in a sanitary condition. Section 3362 in CCR Title 8, *Sanitation – General Requirements*, was amended in 2002 to include new subsection (g) that requires employers to prevent and correct water intrusion, leakage or other uncontrolled accumulation because of the potential to cause mold growth. Other subsections require the employer to maintain the workplace in a clean, orderly, and sanitary condition; to prevent the entrance of insects, rodents or other vermin; to store putrescible wastes and garbage in covered cans; and to remove wastes as frequently as necessary to prevent harmful exposure.

4.5.1.3 Construction-related Indoor Air Quality Investigations in Workplaces

Many complaints to Cal/OSHA result from remodeling or other construction activities conducted in or adjacent to areas in which employees are working. In these situations, employers are required under Section 3203 to inspect the new operations, to evaluate hazards found, and to take measures to correct them in a timely manner. Section 5194 further requires employers to inform employees about hazardous substances in their work area, including substances being applied by contractors. In addition, the following Cal/OSHA sections require employers to control specific construction related hazards, such as asbestos, lead, and dusts.

Section 5208, *Asbestos*, was filed in 1996 and amended subsequently. It requires building and facility owners to determine the presence, location, and quantity of installed asbestos-containing materials (ACM) or presumed asbestos-containing materials (PACM), and to inform employers or employees in that building or facility of those results. Employers are required to provide that information to their employees. This section further requires training for employees who perform janitorial or maintenance activities in areas containing ACM or PACM, and the posting of labels or signs on installed ACM or PACM.

Section 1529, *Asbestos*, requires employers engaged in construction activities involving ACM or PACM to use certain control measures to protect employees in adjacent areas. Subsection (d) requires that asbestos contractors inform employers of employees in adjacent areas of those control measures, and it requires those employers to take measures to protect their employees from asbestos hazards and take steps on a daily basis to ensure that the control measures are adequately protecting their employees.

Section 1532.1, *Lead*, requires employers engaged in construction activities involving lead to use specified control measures, to establish a regulated area where exposures exceed the PEL, and to inform adjacent contractors of those control measures.

4.5.1.4 Environmental Tobacco Smoke in Workplaces

Environmental tobacco smoke historically has been strongly correlated with poor indoor air quality. Since 1995, California regulations (Labor Code 6404.5, Section 5148) prohibit smoking in most enclosed workplaces (also discussed in Section 4.1.5). This statewide prohibition is primarily enforced at the local level. Cal/OSHA is not required to respond to complaints until the employer has been found guilty at a local level three times in the previous year

(http://www.dir.ca.gov/dosh/dosh_publications/smoking.html). On May 29, 2003, in a Decision After Reconsideration (Robert D. Schultz and James A. Noll [OSHAB 01-125]), the Appeals Board held that the Division does not have authority to take action to enforce the Labor Code in the absence of a regulation promulgated by the Standards Board. Subsequently, in February 2004, the Standards Board adopted a workplace regulation for enforcing the smoking ban.

4.5.1.5 Indoor Air Quality Enforcement Policy & Procedures for Workplaces

Policy and Procedure C-48 (http://www.dir.ca.gov/doshpol/p%26pc%2D48.htm) describes Cal/OSHA enforcement procedures for indoor air quality investigations. In general, it defines "serious indoor air quality complaints" as those in which there is a known or suspected building-related illness, or a cluster of cancer cases or adverse birth outcomes. Non-specific complaints related to building IAQ are also considered serious when their magnitude, frequency, and their severity are above certain levels. "Non-serious indoor air quality complaints" are defined as those involving particles, HVAC systems, thermal stress, or symptoms that are not specific to a particular disease or pollutant. Investigations of building related illness might involve the Cal/OSHA medical unit, as well as support from state and local health agencies. Interview, inspection, monitoring, and citation procedures are also outlined.

4.5.1.6 Standards for Nursing Homes and Public Housing

Nursing homes, group homes, and community care facilities in California are regulated by the DHS Licensing and Certification Program (HSC 1265 et seq., HSC 1520 et seq.; HSC 1725 et seq.). The licensing requirements include provisions to protect the health and safety of clients. State inspectors conduct annual inspections or "surveys" and investigate complaints regarding the condition of the building, food preparation, and health care. They also make follow-up visits to assure that problems have been identified and corrected. However, it is not clear that IAQ is a high priority item for inspections unless there are several complaints by clients or an outbreak of a building related illness.

Public and Indian housing in California are funded by federal and state agencies, often through local public housing authorities and redevelopment agencies. Also, HUD provides funding for 5% of U.S. homeowners and renters through programs for public, assisted, and insured housing as well as housing financed through HUD's formula for competitive grant programs (http://www.hud.gov/news/release.cfm?content=pr02-105.cfm). State agencies often provide additional funding. HUD has adopted regulations regarding the operation and maintenance of public and Indian housing that receives federal funds. Public housing authorities are required to annually set aside funds for building operation and maintenance. Some operation and maintenance activities are required to undergo environmental review (24CFR990.111). However, except for lead and asbestos contamination problems, public and Indian housing programs have not routinely addressed IAQ in their operation and maintenance programs.

4.5.2 Guidelines for Building Operation and Maintenance

Various guidelines for providing good indoor environmental quality through building operation and maintenance are available from the public and private sectors. Examples from California and national organizations are discussed below. Many other guidelines are available from other states and nations that have active programs to address indoor environmental quality.

4.5.2.1 IAQ Tools for Schools

The U.S. EPA's *IAQ (Indoor Air Quality) Tools for Schools* Program is a program developed to help schools identify and prevent indoor air quality problems, using a team approach to school indoor environmental quality management. The program provides educational materials and tools for evaluating the impact of school maintenance functions and occupants' daily activities on indoor air quality. U.S. EPA makes their *IAQ Tools for Schools* action kits available at no cost, and has funded numerous training workshops, including many in California. Implementation of this program by school districts in California and other states has not taken a significant amount of staff or funding, but it has yielded substantial health and economic benefits (see Section 3, Costs of Indoor Air Pollution). As a prerequisite for obtaining state funding for school operation and maintenance, the State of Minnesota has required schools to adopt an IAQ management plan based on the *IAQ Tools For Schools* program.

Despite the outreach efforts for the *IAQ Tools For Schools* program, awareness and use of the program among California schools are still low: 35% of schools recently reported that they were familiar with the program, but only 11% of California schools reported that they use all or part of the program (ARB/DHS, 2003). These low levels may be due to a misperception regarding the level of effort required, although the program is adaptable to any level of resources.

4.5.2.2 Large Building Air Quality Guidance

U.S. EPA (1991) and the National Institute of Occupational Safety and Health have published "Building Air Quality: A Guide for Building and Facility Managers." This document describes the common principles of good facility management, provides background information on topics related to IAQ, and recommends strategies for problem identification and resolution in large buildings such as office buildings. The guidance emphasizes changing how building managers operate and maintain their building, not increasing the amount of work or cost of maintaining the building. The companion document, the "Building Air Quality Action Plan", is an easy-to-understand, 8-step implementation path for building owners and managers (U.S. EPA, 1998). The U.S. EPA (1997b) has also published guidelines for occupants of office guidelines, entitled "An Office Building Occupant's Guide to Indoor Air Quality". Most recently, U.S. EPA is offering I-BEAM, an interactive software program that integrates IAQ, energy efficiency, and building economics into a powerful management tool.

DHS (1995) has published guidance for occupants of large buildings with HVAC systems. Entitled "A 'Do-It-Yourself' Inspection of a Ventilation System," this document provides background information and guidance to help occupants identify potential causes of IAQ problems in their building, and to help them ask the right questions of building managers and investigators.

5. METHODS TO PREVENT AND REDUCE INDOOR AIR POLLUTION

There are five basic approaches to indoor pollution prevention and reduction of indoor air pollution. The most effective and reliable approach is usually source control. Source control avoids, removes, or reduces the sources of indoor pollution by using building materials, consumer products, and appliances that emit little or no air pollution. Ventilation also is necessary for healthful indoor air quality and comfort. It dilutes and removes indoor air pollutants to help reduce exposures, but not as effectively or reliably as source reduction. Proper operation and regular maintenance of buildings and their ventilation systems are key to preventing indoor air quality problems, especially over the long term. Public and professional education are also key to pollution prevention; they provide the awareness and understanding necessary to promote informed choices about preventing and controlling indoor air quality problems. Finally, air cleaning devices (air filters and air cleaners) can also be helpful in certain situations when used along with source control and ventilation.

An effective strategy for maintaining good indoor air quality must integrate all of these approaches. Many actions can be taken to prevent and reduce indoor air pollution and, in some cases, completely eliminate emissions of indoor air pollutants. The best approach will depend on the specific problem and will vary accordingly.

5.1 SOURCE CONTROL

Source control is the prevention or reduction of emissions at the source. It is the most effective and reliable approach to reducing indoor pollution because it keeps pollutants from entering and spreading throughout a building. It is the most reliable approach because it does not rely on building maintenance, or other human actions (ARB, 1989; NRC, 1981). Source control can be accomplished through source substitution, source removal, and source modification.

Source substitution involves using an alternative product that emits little or no pollutants of concern. For example, alternative building materials that emit little or no formaldehyde are generally available and can be used in place of a building material that emits formaldehyde. Source removal involves eliminating the source from the building. For example, having family members smoke outdoors, and properly disposing of old cans of paint stored in a closet, removes these sources from the home. Source modification involves reducing the rate at which a pollutant is emitted into the indoor environment. Source modification could involve a change in design, formulation, or usage of a consumer product. For example, sealing all surfaces of a particleboard bookshelf can greatly reduce formaldehyde emissions (Kelly *et al.*, 1999; ATS, 1997; NRC, 1981). In addition, "green" cleaning products that have reduced levels of irritant and odorous compounds can reduce health and safety hazards for janitorial workers and occupants of buildings (OFEE, 2005; WRPPN, 2003).

> **Minimizing indoor emissions is generally more effective than removing them after emission has occurred.**
>
> *T.J. Kelly, Battelle, Indoor Air Quality Symposium: Risk Reduction in the 21st Century, Sacramento, May, 2000*

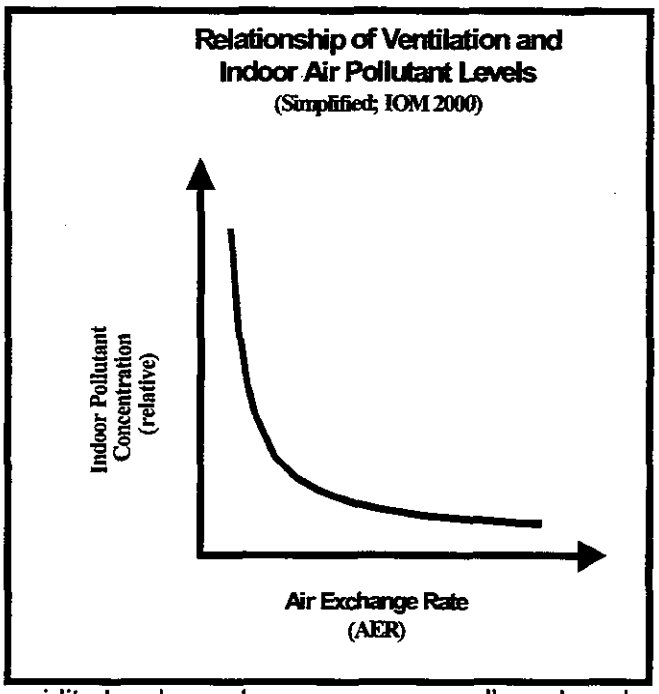
Source control typically can be accomplished at the manufacturing level without direct impact on people's activities and homes. Many consumer products, for example, have been reformulated by manufacturers to comply with ARB's consumer product regulations to control emissions of

reactive VOCs to the outdoors. Gas-fired furnaces, boilers, and water heaters have also been re-designed to comply with air quality management district limits for nitrogen oxides emitted to the outdoor air, but such regulations do not exist for emissions of nitrogen oxides to the indoor air, which can be substantial for unvented appliances such as gas stoves. Most such product changes have not been noticeable to consumers, or have had minimal impact on their choices and activities. Appliance standards could also be improved at the manufacturing stage to increase combustion efficiency, thereby lowering indoor and outdoor air emissions. Such standards might require reduced indoor nitrogen oxide emissions, automated local exhaust, and a clearly specified maintenance schedule.

Standard methods for testing emission rates are available for a number of indoor pollutant sources, such as consumer products, paints, carpets, and other building materials. Additional work is needed to develop test standards for other indoor pollutant sources, e.g., ozone emissions from air cleaners and office equipment, and combustion product emissions from gas stoves and fireplaces. More importantly, consumers, architects, building and interior designers, and building maintenance personnel need access to accurate and reliable information on the emissions of toxic and irritant compounds from products and appliances.

5.2 VENTILATION

Building ventilation is defined as air delivered to a space to dilute airborne contaminants. It serves many purposes. Ventilation removes and dilutes chemical contaminants and body effluents (such as carbon dioxide); maintains comfortable levels of temperature, humidity, and air movement; and helps remove excess moisture to prevent the growth of mold, bacteria, and dust mites. Thus, adequate and effective ventilation is necessary for acceptable indoor air quality, even when known air contaminants and sources are minimized. For instance, the air may have very low levels of measured air pollutants, but without ventilation, exhaled carbon dioxide, other body effluents, and non-measured air pollutants will build up, resulting in a stuffy, uncomfortable environment and potentially increased levels of toxic and irritant compounds. The figure illustrates the basic principle that indoor pollutants, body effluents, humidity levels, and so on are generally reduced as the number of air changes with outdoor air per hour (or air exchange rate) increases.



5.2.1 Types of Ventilation

Ventilation can be "natural" or mechanical. Natural ventilation is the passive air movement through open doors or windows, and unintentional air infiltration through the cracks and gaps of the building envelope. Mechanical ventilation is the active movement of air through the building using fans to pull outdoor air in (make-up air), mix and circulate the air, and exhaust the indoor air to the outdoors.

There are two types of mechanical ventilation – mixing and displacement. Mixing ventilation mixes "fresh" air and existing room air to uniformly dilute pollutants; this is the most common type of mechanical ventilation. Displacement ventilation is the introduction of "fresh" air so that the air around the occupants (in their breathing zone) is separated from the polluting sources. Displacement ventilation requires precision design and operation (Liddament, 2000). To operate efficiently and control air flows, mechanical ventilation systems require a relatively airtight building, hence, energy efficient builders commonly follow the principle of "build tight and ventilate right" (ESB, 1995).

Localized mechanical ventilation is often used to remove pollutants and increase ventilation in special use areas. For example, ducted exhaust fans are useful in bathrooms, laundry rooms, and kitchens to control odors, excess moisture, and potential mold growth. Local exhaust ventilation can be very effective when airflow is sufficient and exhaust fans are not too noisy. The use of exhaust hoods ducted to the outdoors is especially critical when using gas stoves or ovens, in order to remove the emissions from the appliances and those generated during cooking, which can be substantial.

For ventilating entire buildings, appliances such as heating, ventilation, and air conditioning (HVAC) systems are commonly used to provide mechanical ventilation, along with space heating and cooling. In California, these systems are used in most nonresidential buildings such as offices and schools, and usually include at least some minimal level of particle filtration for outdoor air. However, most residential buildings in California rely on natural ventilation for ventilating the whole building. California homes usually have a central air system, but rarely is it designed to bring in outdoor air or to run continuously when a building is occupied. Consequently, these residential systems mainly use recirculated air. They may have a small percentage of unfiltered outdoor air coming from unintentional leakage pathways from the garage, crawlspace, attic, or other areas.

Natural ventilation has limited effectiveness and reliability because it depends on building occupants to routinely open windows and doors. It also relies on indoor-outdoor temperature differences and wind speeds sufficient to force air through small cracks and other leakage points in a building, but these conditions are not consistently present in much of California. Natural ventilation does not allow for filtration of pollen and outdoor air pollutants, which can be substantially elevated in many parts of California. In addition, air flows from potentially contaminated areas such as garages and crawlspaces cannot be prevented by natural ventilation.

Despite its advantages, mechanical ventilation nonetheless has its limitations. It often does not provide adequate indoor air quality and comfort because of insufficient ventilation rates, poor air mixing within the building, uncontrolled air flows from contaminated areas, or poor quality of delivered air. These problems are usually due to improper design, operation, and maintenance of the HVAC system and local ventilation units (U.S. EPA, 1997b). For example, HVAC systems can produce excessive indoor noise, which lead occupants to frequently turn off the systems, as shown in the California Portable Classrooms Study (ARB/DHS, 2004). Building modifications may occur over the years without proper modifications to the HVAC system. Ventilation systems themselves also can be sources of indoor air pollutants, due to buildup of dust in ductwork and filters, and mold and bacterial growth in drain pans, cooling coils, filters, and ductwork. Inadequate training of building operators and insufficient maintenance funding also contribute to poor performance of HVAC systems.

5.2.2 Ventilation, Health, and Comfort

The quantity and quality of the outdoor supply air plays an important role in assuring acceptable air quality in non-industrial buildings. A multi-disciplinary scientific consensus group recently reviewed over 100 peer-reviewed papers from the U.S. and Europe (Wargocki *et al.*, 2002). This group concluded that increased ventilation is strongly associated with improved comfort and improved health (SBS symptoms, inflammation, infections, asthma, allergy, short-term sick leave). Increased ventilation was also associated with improved productivity of office workers. For residential buildings, the authors concluded that ventilation rates above 0.5 air changes per hour in Nordic homes were found to reduce infestation of dust mites. However, air conditioning systems in non-industrial buildings with mechanical systems have been associated with the increased prevalence of SBS symptoms, indicating the need for better design, maintenance, and training of building operators (Wargocki *et al.*, 2002; Seppanen and Fisk, 2002).

The conclusions above from literature reviews are relevant to buildings in the U.S. and California. In a study of 41 U.S. office buildings, lower ventilation rates per occupant (as indicated by increased indoor CO_2 levels over outdoor levels) were significantly associated with increased SBS symptoms, increased absences, and reduced productivity among workers (Apte *et al.*, 2000). These associations held after adjusting the statistical model for several indoor air quality and occupant characteristics of the buildings. This study included some California office buildings.

5.2.3 Adequacy of Ventilation in California Buildings

Information on the quantity and quality of ventilation in California's public and commercial buildings is very limited. Neither Cal/OSHA nor other agencies routinely inspect or track ventilation rates in buildings with mechanical ventilation systems. The results of a recent large statewide survey of K-12 classrooms in California indicated that ventilation rates were inadequate for about 40% of the school day, and were severely inadequate about 10% of the time (ARB/DHS, 2004). Results from a small study of California buildings found that average air exchange rates (in air changes per hours) were 2.24 in 14 school buildings, 1.35 in 22 office buildings, and 1.35 in 13 retail buildings (Grot, 1995), and ranged from about 0.2 to 4.7 or larger. A substantial percentage of these buildings had minimum ventilation rates that did not meet applicable building energy and workplace regulations.

Information on ventilation rates in California's residential buildings is also limited. A few studies measured air exchange rates in homes of various ages, but all of the seasons and major regions of California have not been well represented. ARB (1998) summarized the available information, estimating that average air exchange rates (in air changes per hour) for California homes are 0.5 to 0.9 in the winter, and 0.7 to 2.8 in the summer, with spring and fall rates falling between the summer and winter values. The range of reported air exchange rates was very wide, as indicated by the large standard deviations and the lowest values near 0.1 air change per hour. A national study of air exchange rates from a database maintained by Brookhaven National Lab prior to 1993 indicated an overall mean air exchange rate for California and Arizona homes of 1.03 air changes per hour, and a 5th – 95th percentile range of 0.22 –2.87 (Pandian *et al.*, 1993, 1998).

Newer California homes are likely to have lower air exchange rates, due to the increased "tightness" of new homes resulting from energy efficiency requirements such as the increased use of water vapor barriers on exterior walls, and the mandatory sealing of HVAC ductwork. As homes are made tighter, there is an increasing risk of degrading indoor air quality. To avoid this,

the California Energy Commission specifically considers IAQ when setting state energy efficiency standards for buildings and appliances, as discussed in Chapter 4. However, very little data on ventilation rates and occupant ventilation practices are available for new homes in California. To assure that air quality in new California homes is healthful, the ARB and the California Energy Commission are jointly conducting a study to examine the adequacy of residential air exchange rates in new homes. It is especially important to avoid very low ventilation rates, which have been associated with the most severe impacts on IAQ and human health in some studies. The incremental cost of energy to increase ventilation rates to acceptable levels has been found to be minimal, especially when considering the avoided costs of impacts on human health and productivity.

What is needed to improve ventilation in California buildings?

- Better commissioning and annual operational checks
- Quieter systems, especially for schools
- Control of building space pressures
- Use of higher efficiency air filters
- Better control of thermal comfort parameters (temperature and humidity)

F.J. Offermann III, Indoor Environmental Engineering, Indoor Air Quality Symposium: Risk Reduction in the 21st Century, Sacramento, May, 2000

To achieve the full benefits of building ventilation, additional work is need to design mechanical ventilation systems that are quiet and easier to clean and maintain. A convenient, inexpensive but accurate method for assessing ventilation rates is necessary to facilitate routine testing of ventilation systems. Improved awareness of, and compliance with, state regulations for operation and maintenance of mechanical ventilation systems in schools and other workplaces are also needed.

Although it is necessary for adequate indoor air quality and comfort, ventilation is not a complete solution to indoor pollution. Ventilation consumes energy, and does not completely remove some pollutants, such as formaldehyde from building materials, that require years to off-gas. Ventilation reduces but does not completely eliminate near-source exposures when people use individual products in a building. Thus, it is generally preferable to remove highly toxic compounds from these products all together (ARB, 2000b).

5.3 BUILDING OPERATION AND MAINTENANCE

A well-operated building and regular maintenance is fundamental to providing good indoor air and a comfortable and productive indoor environment (Spengler, 2001; ATS, 1997). Basic building elements interact to affect the indoor environment. The building shell shelters the occupants from the elements, and at the same time may both emit and trap contaminants. The HVAC system conditions the air for comfort, but if not properly maintained it can add to the discomfort of occupants and become a source and dispersion method for mold, bacteria, dust, and chemical contaminants in the building (Clausen, 2004; Liddament, 2000; U.S. EPA, 1991).

Proper building operation and maintenance (O&M) for indoor air quality are comprised of several key elements. First, a written plan and maintenance schedule are needed to operate and maintain the building as originally intended and as appropriate for the specific building design, and to ensure that health and safety standards are met. The plan should address not only the routine maintenance after occupancy, but also the control of dust, fibers, mold, and chemical contaminants during building construction, repairs, and renovation. Next, the building should be commissioned after initial construction, after major renovations, and periodically

throughout the life of the building. As discussed in Chapter 4, commissioning involves performance testing and inspection of the building to ensure that it performs as intended and that the various building systems work together properly. For example, commissioning helps verify the building's energy use, ventilation rates, thermal comfort levels, and use of specified building materials, air filters, and other environmental features. Commissioning also includes completion of the O&M plan and the initial training of O&M staff before the building is occupied. From the energy standpoint alone, commissioning by third party agents has been shown to be very cost-effective.

Another key element is training of O&M staff in the basic principles of indoor air quality and the proper methods to achieve good indoor air quality. Maintaining high quality indoor air requires a solid understanding of how buildings operate, the relationship between the HVAC equipment and indoor air quality, and how to achieve the design intent (Bearg, 1993). Regular cleaning with the proper equipment and methods can go far to eliminate some biological and dust contaminants. Effective cleaning, for example, involves using a vacuum cleaner with good filtration and efficiency, and allowing sufficient residence time of the vacuum head on the carpet or floor to assure good pick-up. Proper vacuuming can significantly reduce asthma and allergy triggers such as mold spores and animal dander in carpets, as well as pollutants that are often adsorbed onto floor dust, such as pesticides and PAHs. Similarly, cleaning properly with disinfectants and cleaning products can reduce bacteria and mold on surfaces and reduce exposure to potentially harmful bacteria. Training guidance is available from various sources mentioned throughout this report. Ongoing commissioning also is needed to assure that systems continue to perform as intended.

To implement a good O&M program, adequate funding is also essential. A reasonable funding level can be determined based on industry norms, experience in similar buildings, and on manufacturer recommendations for equipment repair and replacement. However, maintenance programs are often the first to be cut during a budget crisis, and the maintenance budget for California's public schools are especially strained (Patterson, 2000). Without adequate funding, maintenance programs fall further and further behind, thereby increasing the likelihood of major IAQ problem, structural damage, catastrophic failures, and litigation.

A good O&M program can not only help avoid worker productivity and health impacts from poor indoor air quality, but it can also help reduce the potential for litigation. Several lawsuits have been successfully litigated for mold cases in public buildings, commercial buildings, and homes. (Hirsch, 2005). Lawsuits over other IAQ problems such as CO poisoning, SBS, and pesticide poisoning have also been filed, but often end in out-of-court settlements. Litigation over IAQ has increased in recent years in the areas, especially in the area of mold damage (Facilities Network, 2005). This has led insurance companies to drop or limit their coverage for mold or water damage (Kats, 2003). It has also led to a proliferation of mold remediation companies that offer to inspect, test, and correct for mold problems. However, because there is no widely recognized licensing and training program for such companies, consumers and building owners are vulnerable to fraudulent practices and poor workmanship.

Litigation and liability risks do provide an incentive for better building design, operation, and maintenance, but they are not an efficient approach for society. Some IAQ cases are hard to prove because the toxic effects are chronic and delayed long after the initial exposures. In addition, litigation is very expensive, and individual homeowners and small businesses rarely have the resources to pursue litigation against a large company. Finally, increased litigation and liability may affect only business practices for certain IAQ problems such as mold, and may help the litigants but not the population in general.

Building O&M is effective in preventing and controlling indoor air pollution, but only to the extent that proper training and adequate funding are provided. O&M can help achieve good indoor air quality if the indoor pollutant sources are controlled and the HVAC system is properly designed and commissioned, but this is not always the case.

In order to assure good O&M for buildings, additional work is needed to improve compliance with Cal/OSHA regulations for maintaining mechanical ventilation systems and preventing moisture intrusion in nonresidential buildings. For residential buildings, guidance is needed to improve the maintenance of the HVAC system, combustion appliances, and the building shell. Training of personnel in local health departments and building departments is also necessary to help them recognize and prevent IAQ problems in residential and nonresidential buildings and help enforce building and worker health standards.

5.4 PUBLIC AND PROFESSIONAL EDUCATION

People's choices and behavior have a major impact on their exposures to air pollution. Human activities are one of the key factors in determining the condition of the indoor environment. Cooking, the use of various consumer products, cigarette smoking, and other activities can result in immediate, significant releases of pollutants indoors which are immediately inhaled (ARB, 1987). Also, whether people choose to clean and maintain homes and buildings properly can have a major impact.

People are often not aware of the risks associated with indoor pollution and what they can do to protect their health. Many people assume incorrectly that any indoor air quality problem can be solved with an air cleaning device in their home or office, as witnessed by the recent boom in the sales of air cleaners. Sometimes activity pattern changes are needed, e.g., people must not smoke or must select different building products or consumer products to reduce their exposure and risk. Similarly, timely building maintenance and repair is critical to prevent mold development and accumulation of particles on surfaces. Even some building professionals are not aware of the impact of their lack of action on the building environment. Thus, increased awareness is a key step for reducing exposures to indoor air pollution (NRC, 1981; ATS, 1997).

Increasing people's awareness can be accomplished through public and professional education. However, public education will never be a complete solution. Some groups of the population cannot respond appropriately to take needed action. For example, children cannot read or understand all written information that is provided, elderly people living in group settings cannot control the products used in the facility, and low-income families may not be able to afford safer alternatives, even when they are fully aware of them.

Additionally, public education will not always reach all members of society. The many non-English speaking ethnic groups that enrich the California population pose an increased communication challenge. The California population contains a large number of people who speak a language other than English at home, as well as a large number of foreign-born individuals. According to the U.S. Census Bureau, the year 2000 census reported about 18% of the U.S. population 5 years and older spoke a language other than English at home. In California, that number is higher: about 40% of Californians speak a language other than English at home (DOF, 2002b). Also, the proportion of the California population that reported being foreign-born has increased from about 21% in 1990 to over 26% in 2000 (Malone *et al.*, 2003).

Public education can change people's awareness and actions, but it does so slowly and requires a long-term commitment with adequate funding in order to reach the desired audiences and compete with commercial advertising. Because of these reasons, increased public education and information are not adequate as a sole approach to preventing indoor pollution.

The decisions of various persons and groups in the building, healthcare, and education professions can also help prevent and control indoor air pollution. Consumers and employers often rely on professionals to advise them on IAQ issues in their building, and perhaps to inspect and remediate their building. However, training for and awareness of IAQ is minimal in most professions involved with buildings and environmental health. Furthermore, there is no widely accepted training and certification program for IAQ professionals. Several groups offer certification as an indoor air quality expert, but the certification requirements are sometimes as minimal as completing a form or completing a one-day class. To change the awareness and actions of building owners and employers, existing training programs for building professions should be expanded, and adequate training on IAQ for professionals in the healthcare and education fields should be provided.

5.5 AIR CLEANING DEVICES

Air cleaning devices are available in a variety of types and sizes. Most air cleaners remove particles, a few remove gases, and some perform both removal processes. Air cleaners can remove particles from the air using a mechanical or physical barrier, or through an electronic device. Mechanical air cleaners draw air through a filter with different sized pores that trap the particles. The most common types use a flat or pleated fibrous filter that can range from low to high efficiency at trapping particles. Electronic air cleaners are available in three basic types: electrostatic precipitators (ESPs), ionizers, and mechanical-electronic hybrids. ESPs use a small electrical charge to charge particles, which are then collected on oppositely charged plates as air is drawn through the device. Ionizers, or negative ion generators, also charge particles, causing them to stick to surfaces near the ionizer, such as the carpet and walls. Hybrid devices use both mechanical and electronic devices for pollutant removal. Air cleaners that remove gases and odors are less common, and relatively more expensive to purchase and maintain. Gaseous pollutants are typically trapped or destroyed as the air is drawn through materials such as activated charcoal or alumina coated with potassium permanganate. However, the filter material can become quickly overloaded and may need to be replaced often.

Air cleaning devices usually come as portable, stand-alone appliances, or as filters or cleaners in a central air system. Portable air cleaners are sometimes practical for rooms in existing homes where addition of a central air cleaner is too costly. Proper size, installation, and maintenance are critical for portable air cleaners to be effective. Test standards for particle removal by air cleaning devices have been developed by trade and engineering groups. The effectiveness of some portable air cleaners in removing particles is rated in terms of pollutant removal efficiency or clean air delivery rate (CADR), measured in cubic feet per minute (cfm). The CADR equals the airflow (cfm) multiplied by the efficiency of particle removal; a larger CADR is better. The CADR ratings are given separately for the removal of dust, pollen, and environmental tobacco smoke. Standards for gas removal have not been developed.

Portable air cleaning devices have limited utility, are often not very effective (ARB, 2000c; ATS, 1997; Consumers Union, 2003a), and can incur high-energy costs relative to the benefit gained. However, air cleaners may be useful for controlling airborne particles for some individuals with special sensitivities, such as those with asthma or allergies who use them in their bedrooms at night. Additionally, recent research has shown that in-duct ESPs can effectively reduce

residential particles when the ESP is properly maintained (Howard-Reed *et al.*, 2003; Wallace *et al.*, 2004). Based on the limited scientific evidence that is currently available, the health benefits of air cleaners are not clear. Generally, it is more effective to prevent emissions rather than to try to remove them from the air once they are there.

For new homes or major remodels, "whole-house" or "fresh-air" ventilation systems that include some type of air cleaning device can be installed. Installed costs depend on the system size and the type of air cleaning device. Fresh-air ventilation systems are recommended in new, tightly built energy-efficient houses and for situations where the outdoor air is a major source of indoor pollution. To design and install any central system air cleaner, a company or contractor that is well experienced in designing and installing central filtration systems is important (ARB, 2005b).

Ozone Does Not Clean the Air!

Some air cleaning devices emit ozone, either purposely or as a by-product of the particle-removal technology used in the device. Those that generate ozone by design are often called 'ozone generators". ESPs, ionizers, and hybrid models often emit ozone as an unintentional by-product. While just a few studies have examined the health risk from the use of ionizers and electrostatic precipitators, research has shown that the use of ozone generators can result in harmful levels of indoor ozone.

Independent studies by the U.S. EPA, the Consumers Union, and others have shown that ozone generators can produce unsafe room levels of ozone up to several times higher than the state ambient air quality ozone standard of 90 ppb for one hour, reaching levels equal to a Stage One smog alert (200 ppb) (Mason *et al.*, 2000; Consumers Union 1992; Chen and Zhang, 2004). In a U.S. EPA study, ozone generators generated over 300 ppb in a test house within an hour or two of operation, and over 400 ppb in a chamber experiment (Mason *et al.*, 2000). In another study of a variety of air cleaner models tested in a chamber under realistic conditions, the ozone generators were the only models to exceed the OSHA 8-hour PEL for ozone of 100 ppb; levels again reached 300-400 ppb (Chen and Zhang, 2004). Personal air purifiers, which are typically small ozone-generating devices worn around the user's neck, can produce over 100 ppb in the user's breathing zone (Phillips *et al.*, 1999).

Ozone-generating Air Cleaners

"These machines are insidious. Marketed as a strong defense against indoor air pollution, they emit ozone, the same chemical that the ARB and USEPA (U.S. Environmental Protection Agency) have been trying to eliminate from our air for decades."

Barbara Riordan, interim ARB Chairperson. California Air Resources Board, Press Release 05-02, Sacramento, January 2005.

In addition to the potential harm posed by excessive ozone emissions, ozone generators also do not effectively destroy microbes, remove odor sources, or reduce indoor pollutants enough to provide any health benefits as manufacturers claim (Foarde *et al.*, 1997; Boeniger, 1995; Chen and Zhang, 2004). Ozone generators are often advertised as able to kill mold and bacteria even at "safe" ozone levels; however, ozone in the air must reach extremely hazardous levels (50-100 times the state standard) to effectively kill microbes (Foarde *et al.*, 1997). Ozone generators also are advertised as able to remove gaseous pollutants and odors, but ozone only reduces one group of VOCs, alkenes (a group that includes benzene). Additionally, ozone deadens the sense of smell. Most importantly, ozone reacts with other chemicals present indoors and leads

to significant increases in the indoor levels of formaldehyde and other aldehydes, other VOCs, and ultrafine PM (Boeniger, 1995; Weschler, 2004). Some of these reaction products are listed as California toxic air contaminants, which can irritate the mucous membranes and respiratory tract or cause other health impacts.

Current market trends indicate that the use of ozone generators in California is widespread and growing. In an aggressive marketing campaign for ozone-generating devices, companies have targeted the most vulnerable populations, such as the elderly and persons with asthma or other respiratory diseases. Often, the advertisements used in the marketing of these devices make unsubstantiated claims and use misleading terms such as "super-oxygenated," "activated oxygen," or "trivalent oxygen," when referring to the ozone that is emitted.

State agencies have recommended that ozone-generating air cleaners not be used in the home (DHS, 1997; ALA, 1997; ARB, 2005). Presently, no state or federal agency has the direct regulatory authority to control emissions from these devices, although the Consumer Product Safety Commission has some authority to address products through a ban, required labeling, or other approaches. Efforts to restrict these devices through lawsuits, establishment of voluntary emission standards, cease and desist orders, and other means have all failed to restrict the sales and use of ozone generators.

6. PRIORITIZATION OF SOURCES AND POLLUTANTS BASED ON EXPOSURE AND ADVERSE IMPACTS

Reduction of public exposure to the many indoor air pollutants is most effectively achieved by reducing pollution at the source. Because exposure is dependent on the presence and use of sources, pollutants are best prioritized for action by source categories. Mitigation approaches focused on source categories would be most effective for reducing pollutant exposure, and would generally be feasible to implement, although the feasibility would vary by specific source. Tables 6.1 and 6.2 suggest a prioritization scheme for implementation of mitigation measures, by source categories. The source categories have been ranked into two groups – high and medium priority – and are listed alphabetically within each ranking group.

The primary factors considered in prioritizing the source categories included the extent of the population's exposure to the sources and their emissions, the relative reduction in health impacts that could be achieved with further action beyond any already undertaken, ease of mitigation, trends in emissions from and use of source categories, and the extent of the gap in reducing exposure and risk from categories of indoor sources. A quantitative prioritization was not undertaken because such an effort is beyond the scope of this report. Such an effort would be an appropriate step prior to taking action under a comprehensive program to address indoor sources; a detailed prioritization based on quantified criteria would be needed. Additionally, the preliminary indoor air pollution cost estimates provided in this report were considered, but were not weighted heavily in the prioritization for several reasons. Most importantly, the cost estimates primarily reflect the availability of cost information and the length of time a given pollutant, such as ETS and radon, has been studied. Because of the lack of key cost data for most indoor pollutants, the cost estimates do not necessarily reflect the actual extent of exposure and risk in California, nor does it account for current information and trends in the scientific literature on exposure and risk. Thus, cost information was considered but was not a determining factor in the prioritization of sources.

Tables 6.1 and 6.2 also suggest potential approaches for mitigating the pollutants and sources listed. Emission reductions should be accomplished at the manufacturing, distribution, or construction stage. Alternatives or mitigation options are currently available for most of the sources listed. Emission limitations achieved at the manufacturing stage, such as reducing toxic contaminants in building materials, would be effectively invisible to the consumer and assure exposure reduction. Similarly, new appliance standards would reduce combustion emissions without reducing the range of product choices in the market place. With this approach, little or no change in individual behavior would be anticipated. As a result of public awareness and demand, there are an increasing number of "green" or low-emitting building materials, furnishings, and consumer products already available. Low-emitting carpets, no-formaldehyde furniture, and non-toxic cleaning products are currently available, and continue to grow in market share.

Finally, Tables 6.1 and 6.2 includes a column indicating whether direct authority exists at the state level to take the actions listed in column three. For most source categories, there is no state agency with clear, direct authority to take the mitigation actions indicated. In some categories, one or more agencies has limited authority to address a small portion of the sources included. For example, Cal/OSHA could impose product use restrictions or require other actions to reduce worker exposure to institutional cleaning product emissions. However, neither Cal/OSHA nor any other state agency has direct authority to restrict pollutant emissions from

Table 6.1. High Priority Source Categories for Mitigation: [1]

SOURCES OF POLLUTANTS[2] (listed alphabetically)	EXAMPLES OF POLLUTANTS[3] EMITTED	POTENTIAL APPROACH TO MITIGATION[4]	DIRECT STATE AUTHORITY TO TAKE IAQ MITIGATION ACTIONS
Air Cleaners (ozone-generating)	Ozone	Emission limitations	No
Biological Contaminants (mold, pollen, bacteria, viruses, house dust mites, cockroaches)	Particles, allergens, asthma triggers, toxins	Requirements for habitable spaces; require certification of mold assessors and mitigators	Limited
Building Materials & Furnishings (particle board, plywood, paneling, flooring, caulk, adhesives, new carpet assembly, furniture)	Formaldehyde, acetaldehyde, benzene derivatives, acrylates, naphthalene, phenol, some other VOCs	Emission limitations, product use restrictions, market incentives	Limited (some indirect)
Combustion Appliances (gas & propane stoves, ovens, furnaces, heaters; woodstoves and fireplaces)	Carbon monoxide, nitrogen oxides, particles, soot, polycyclic aromatic hydrocarbons	Emission limitations, active exhaust ventilation, safety devices, product use restrictions, product re-design, improved venting	No
Environmental Tobacco Smoke *(cigarettes, cigars)*	Particles, polycyclic aromatic hydrocarbons, benzene, carbon monoxide, some other VOCs	Focused parent education; reduce smoking in homes and vehicles	Yes, workplaces No, private homes and vehicles
Radon (soil, rock, ground water, building materials containing radium)	Radionuclides, radon gas	Screening measurements, building codes	Limited

1. Individual sources may be higher or lower than the source category ranking.
2. All of the examples of pollutant sources may not emit all of the pollutants listed in the corresponding box in column two.
3. Air pollutants may be identified as toxic air contaminants (TACs) by the California Air Resources Board, and/or identified as Proposition 65 chemicals; or, criteria (traditional) air pollutants.
4. Public education, economic incentives, and non-regulatory approaches should also be used where appropriate. The actual approach taken would be determined after extensive discussions with the relevant industries, in consideration of costs, feasibility, and effectiveness.

cleaning products for the purpose of reducing indoor air concentrations and exposures. Also, for most categories, any related authority is usually indirect – the authority is not focused on reducing indoor pollution to protect public health, but rather is a mitigation measure to reduce

impacts of actions taken under the primary authority, or is incidental to the primary regulatory activities. For example, one mission of the Energy Commission is to improve energy efficiency in California's buildings. The Commission has been attentive to indoor air quality impacts that might result from their regulatory actions, and includes provisions to prevent adverse impacts on indoor air that might result. However, the Commission does not have authority to develop a regulation to address indoor air quality outside of their energy-related missions. Finally, while the ARB has regulatory authority over some indoor sources such as consumer products, that authority is targeted toward actions to reduce the impacts on outdoor air quality. Some such actions taken by the ARB have had positive impacts on indoor air quality as well, but those impacts are either incidental to the actions taken for the purpose of reducing or preventing outdoor air pollution, or are mitigation measures designed to reduce adverse environmental impacts from ARB regulations.

The specific rationale for the ranking of each category is discussed below. Note that the prioritization is a ranking of the overall group of sources included in a given category: some individual sources within the group may have a higher or lower priority. A more detailed assessment would be needed to prioritize specific products within these larger categories.

6.1 HIGH RANKED SOURCE CATEGORIES

- **Air cleaning devices or "air purifiers" that generate ozone** should not be used in occupied places, and their ozone emissions should be limited. Some devices marketed as air cleaners purposely release ozone, which can directly harm sensitive occupants. These air cleaners should not be used in occupied spaces because they can produce potentially harmful levels of indoor ozone, up to several times the state outdoor standard level and even a Stage 1 smog alert level. Additionally, they are ineffective at safe levels. Safe, effective alternatives are available in the marketplace. Additionally, ionizers and electrostatic precipitators, two other types of indoor air cleaners, emit ozone to varying degrees as a by-product of their function. These devices are effective at removing particles, but poor maintenance and old age can result in increased ozone emissions. Ozone emissions from these devices should also be limited to assure that indoor concentrations remain well below concentrations that may harm sensitive individuals. Consumers often seek advice about the type of air cleaner they should buy to meet their specific needs, but there are few criteria available to guide consumers in their purchase. Emission limitations are needed for these devices, which are currently unregulated.

  A program to reduce the use of high-ozone-emitting devices and to educate the public should be undertaken. Such a program might include:
  a) Development of public and professional guidance materials for indoor ozone.
  b) A public and professional outreach program to alert them to the potential harm of ozone generators and safe alternatives. Such a program should target medical and dental professionals, building managers, school administrators and facility managers, senior citizen organizations, public health entities, environmental organizations and others.
  c) Development of test protocols for air cleaners, to include methods for testing ozone emissions as well as for effectiveness of cleaning the air.
  d) Meetings with manufacturers and their professional organizations to facilitate actions to address this issue.

- **Biological contaminants** are a group of both sources and pollutants. Some mold and bacteria can emit chemical toxins, but for the most part the organism or its parts actually

cause the effect. Biologicals are a high priority because of their ubiquitous presence and their widespread health and fiscal effects. Animal dander, pollen, house dust mites, and cockroaches cause millions of sensitive individuals to experience allergy symptoms and asthma attacks. Indoor mold has been an increasing problem in recent years, costing substantial sums of money for remediation and lawsuit settlements. Bacteria such as *Legionella* cause both serious illness (for example, Legionnaire's disease) and shorter-lived disease (for example, Pontiac Fever). While not emitted into the indoor environment per se, infectious disease transmission is increased in indoor environments with crowded or dirty conditions and insufficient outdoor air flow. Improved methods are needed to objectively and efficiently identify and quantify dampness and microbiological growth in building, especially within wall cavities or other areas that are difficult to access. Additional research is needed to develop such methods.

The mitigation approaches for biologicals are varied. Mitigation actions for mold and some other biologicals might include required annual inspections and remediation in public buildings, group homes, and rental units, and in private homes at the time of sale. Certification requirements for mold assessors and remediators would help assure the quality of inspections and remediation.

- **Building materials and furnishings** are a high priority for mitigation because they often emit multiple toxic air pollutants, especially when new, and have a high loading level in indoor environments, resulting in high exposure levels for occupants. Additionally, a substantial percent of the population is exposed to such emissions due to the continued high rate of new building construction in California and the increasing number of home renovations undertaken by homeowners. Emission limits for pollutants emitted from building materials and furnishings (formaldehyde being the most predominant) would benefit all indoor environments and has potential for significant health benefits due to reduced incidence of asthma exacerbation, cancer, and eye, nose and throat irritation.

  Low-emitting alternatives are available. Non-wood alternatives and composite wood products made with phenol-formaldehyde resin or methyl diisocyanate (MDI) have much lower formaldehyde emissions than composite wood products made with urea-formaldehyde resin, and could be substituted for some applications. In cabinets and furniture, all surfaces of these products can be coated or laminated to substantially reduce formaldehyde emissions. Building materials are currently available that meet Section 01350 emission requirements for formaldehyde and other chemicals of concern (Alevantis, 2003). Low-emitting modular office furniture was used in the Capitol Area East End Complex, and could be used in all future state and private offices. Numerous governmental groups focused on sustainable building and environmentally preferable purchasing are compiling lists of acceptable products that have a low impact on indoor environments. For example, a list of products for use in school construction projects that meet Section 01350 requirements is available at http://www.chps.net/manual/lem_overvw.htm. DSA is working on a similar list. These low- and no-emitting alternatives are available and should be required in public buildings, group homes, schools, and other buildings.

- **Combustion appliances** are also a high priority for mitigation. They emit carbon monoxide, nitrogen dioxide, polycyclic aromatic hydrocarbons, particles, and other pollutants, depending on the appliance fuel, status of adjustment and maintenance, and other factors. These pollutants can have severe acute health effects including respiratory effects and exacerbation of asthma, and contribute to cancer risk. Reduced exposure to pollutants from gas and propane appliances, whether it be through emission limitations, active exhaust

ventilation, or both, could have immediate widespread benefits for occupants in environments with such appliances. Precedence for mitigation of appliance emissions has been set in the low-income weatherization program (DCSD, 2003). Guidelines for carbon monoxide levels and venting requirements in this program can be expanded to statewide regulations.

Statewide measures to reduce emissions from woodstoves and fireplaces both indoors and outdoors also are highly desirable. Such measures could have a major impact on improving both community-wide indoor and outdoor air quality in many areas of the state. Emission limitations, product re-design, product use restrictions, and improved venting can be used for reducing this type of pollution. A number of local government entities have recently approved regulations restricting the use of woodstoves and fireplaces: in the San Francisco Bay area, 24 cities have ordinances that prohibit conventional fireplaces in new construction. The mountain town of Truckee has a more aggressive policy that requires that existing unapproved wood burning appliances be removed by July 15, 2006. The San Joaquin Valley (www.valleyair.org/Burn Programs) implemented a daily advisory for restrictions on residential fireplace or wood stove use on January 1, 2004. Woodsmoke especially impacts those with asthma and other respiratory disease.

- **Environmental tobacco smoke** has been greatly reduced in California, primarily due to legislation that bans smoking at the workplace. However, ETS remains the number one cause of lung cancer, and should be reduced to the greatest extent feasible. Children's exposures remain a special concern, because they can be highly exposed when smoking occurs in their home or in vehicles driven by family or friends who smoke. Actions to reduce children's exposure, such as an increased focus of public education on smoking parents, and reduction of smoking inside vehicles and homes with children, remain a high priority. Additionally, some outdoor and commercial building (casino) exposures continue to occur, with potential for exposure reduction.

- **Radon** gas and its radionuclide precursors enter indoor environments largely from uranium-bearing soil or rock under and near the building. Despite the preliminary high lung cancer risk from radon estimated in this report for California, the lung cancer risk from radon is essentially inseparable from that of smoking, making reduced exposure to tobacco smoke the most effective mitigation approach. Radon levels in California are relatively low, except in a few less populated areas such as the Sierra Nevada foothills and the Ventura Mountains, and recent measurements in the Sierras indicates that statewide radon levels may be lower than estimates derived from measurements obtained in the 1980s. Mitigation approaches usually include depressurizing the basement, crawl space, or sub-slab region to reduce infiltration. However, mitigation is not recommended until adequate testing has been conducted in each building, so that the expense of mitigation is avoided if not needed, or to assure that mitigation measures will be sufficient if they are needed. Levels in buildings very close to each other can vary widely. California exposure data for radon are from the late 1980s, thus the exposure assessment needs to be reevaluated which may affect the priority level. The California Department of Health Services (DHS) has established a list of certified providers of radon services, in three categories: testers, mitigators, and laboratories.

6.2 MEDIUM RANKED SOURCE CATEGORIES

The pollutant source categories included in Table 6.2 are lower in priority than those above, but nonetheless include sources that warrant mitigation.

- **Architectural coatings,** such as paints, and lacquers, are available in "low VOC" versions due to formulation changes targeted toward reducing outdoor ozone. However, they are not directly regulated by the state. ARB develops Suggested Control Measures and provides guidance and technical assistance to air quality management districts in the state, 22 of which have adopted rules to reduce VOC emissions from coatings. Like building materials, architectural coatings are widely used and have a high loading in indoor environments when used, due to the large surface areas they typically cover. Additionally, some components of coatings can be harmful, especially if breathed for extended times such as by painters in non-industrial work settings, but are not necessarily addressed through reactive VOC reductions. However, because reductions have been achieved in districts that cover 95% of the California population, this source category is ranked as a medium priority.

- **Consumer products and personal care products** have been regulated by ARB to reduce emissions of reactive VOCs in order to reduce outdoor smog formation. Reactive VOCs, and in some cases toxic air contaminants, have been reduced substantially through reformulation of a number of product categories. In reducing VOC content to comply with ARB regulations, manufacturers often use water-based technologies and use VOC exempt compounds such as acetone. To prevent increased use of TACs, ARB has also prohibited the use of perchloroethylene, methylene chloride, and trichloroethylene in 13 categories. Antiperspirants and deodorants are not allowed to contain any compounds identified as TACs. Also, the Board recently approved a rule to remove *para*-dichlorobenzene from solid air fresheners and toilet/urinal care products.

  Depending on the product, zero and ultra-low VOC products are not always commercially and technologically feasible. Despite the breadth of products addressed under ARB's consumer products regulations, not all types of consumer products are regulated. Products such as wallpaper and vinyl coverings or plastic products may emit chemicals, but are not addressed in ARB regulations. Additionally, due to the nature of some products (household cleansers, air fresheners, stain removers, etc.), the user is in close proximity to the release of chemicals during use, and experiences greater pollutant exposure than individuals who may be elsewhere in the room or building where the product is used. Thus, there is an apparent need to reduce emissions from consumer products to prevent high personal exposures and risks, and to address types of products not currently regulated under ARB's programs. Because of the ARB's progress to date with chemically formulated products, some of the highest emitters, this category is ranked medium rather than high.

  Chemical reformulations, emission limitations, content limits, and/or product use restrictions of consumer products are mitigation approaches that could result in further significant risk reductions, especially for product users. Such measures have already been taken for many products under ARB's consumer product authority.

- **Household appliances and office equipment** such as computers, copy machines, and vacuum cleaners can emit a variety of pollutants such as particles, ozone, various VOCs of concern, phthalates, and PBDEs. Emissions from each type of appliance could be addressed through emission limitations and/or local exhaust requirements. Examples of a specification for local exhaust would be the placement of a large copy machine under a strong exhaust ventilation hood, or specifying a level of exhaust ventilation for a room. In most cases, these pollutants are emitted directly into the living or working area, and thus are of concern. Additionally, more and more office equipment is being purchased for use in the home, increasing the number of people potentially exposed in the home environment. Because emissions information on many specific sources in this category is outdated or

lacking, mitigation efforts for these products are a medium priority. As further research is conducted on these sources, some products may become a high priority for emission reductions.

Table 6.2 Medium Priority Source Categories for Mitigation[1]

SOURCES OF POLLUTANTS[2] (listed alphabetically)	EXAMPLES OF POLLUTANTS[3] EMITTED	POTENTIAL APPROACH TO MITIGATION[4]	DIRECT STATE AUTHORITY TO TAKE IAQ MITIGATION ACTIONS
Architectural Coatings (e,g.,paints, sealants, lacquers, varnishes)	Formaldehyde, acetaldehyde, ethylene glycol, metals, others	Emission limitations, chemical reformulations, use restrictions to reduce TACs & nonreactive VOCs with health impacts;	No
Consumer Products (e,g.,household and institutional cleaners, furniture- and floor-care products, air fresheners, stain removers, detergents) **Personal Care Products** (e,g.,products used for hair and skin care)	Methylene chloride, para-dichlorobenzene, perchloroethylene, toluene, benzene, naphthalene, formaldehyde, acetaldehyde, metals, others	Emission limitations, chemical reformulations, and product use restrictions to reduce TACs and nonreactive VOCs with health impacts; labeling program	Limited (some indirect)
Household & Office Equipment and Appliances (computers, photocopy machines, vacuum cleaners)	Particles, styrene, some other VOCs, phthalates, ozone, PBDEs	Emission limitations, local exhaust requirements	No
Pesticides (insecticides, herbicides etc, used indoors and outdoors; track-in, drift.)	Chlorpyrifos, diazinon, permethrin, DDT, dieldrin	Formulation and application changes for indoor use	Limited

1. Individual sources may be higher or lower than the source category ranking.
2. All of the examples of pollutant sources may not emit all of the pollutants listed in the corresponding box in column two.
3. Air pollutants may be identified as toxica air contaminants (TACs) by the California Air Resources Board, and/or identified as Proposition 65 chemicals; or, criteria (traditional) air pollutants.
4. Public education, economic incentives, and non-regulatory approaches should also be used where appropriate. The actual approach taken would be determined after extensive discussions with the relevant industries, in consideration of costs, feasibility, and effectiveness.

- **Pesticides** are used indoors and around the perimeter of buildings to control household pests such as ants, spiders, and cockroaches. Pesticide residues may be more persistent indoors than outdoors due to the lack of natural degradation forces such as ultraviolet light, high temperatures, wind, and rain. Pesticides also can be tracked into the home from outdoor application and drift after outdoor spray application. In rural areas, indoor concentrations can be greater due to increased use of pesticides for agricultural purposes.

Levels of pesticides can be measured in both air samples and house dust samples. Children are often exposed through multiple pathways, via dermal exposure and ingestion from pesticides absorbed onto floor dust particles, in addition to inhalation exposure. Thus, indoor pesticide levels and exposures warrant further attention. Pesticides are ranked in the medium category because programs are available to address them. The U.S. EPA and the California Department of Pesticide Registration (DPR) govern the use of pesticides in California. The U.S. EPA banned the use of chlorpyrifos and diazinon in indoor environments in 2000 and 2001, respectively. Continued research and intervention by the U.S. EPA and DPR are needed to assure that the least toxic pesticides are registered for indoor use and that the formulations prevent excessive human exposure. Most importantly, the implementation of integrated pest management approaches should be expanded to reduce the need for pesticide appications.

7. OPTIONS TO MITIGATE INDOOR AIR POLLUTION

This report has shown that there are many sources of indoor air pollution that produce substantial adverse health effects, result in lost productivity, and require considerable expenditures for health care. Despite these facts, there is no systematic program to improve indoor air quality, there are relatively few regulations or standards to specifically address indoor air quality problems, and few resources focused on effectively addressing problems and promoting improvements. Current efforts to address indoor pollution are not commensurate with the scope of the risk to health it poses to Californians.

7.1 GENERAL MITIGATION OPTIONS

Ambient (outdoor) air quality is protected through a comprehensive system. In California and under federal law, ambient air quality standards are established for traditional (criteria) pollutants and must be attained. Under other state authority, pollutants identified as toxic air contaminants must be reduced to the maximum extent feasible. The approach used to reduce toxic air contaminants in ambient air, in which source emissions are reduced without setting enforceable air quality levels, seems most applicable to indoor air. Actions to reduce indoor emissions and exposures would assure reduction of exposure and risk from key sources, and should be a major component of a new effort to address indoor air. Other approaches including public education, product testing and labeling, and setting of maximum exposure guideline levels, should also be part of the mitigation program. The following elements of an indoor air pollution reduction program are recommended for consideration:

1. **Create a management system for indoor air quality** that establishes and assigns authority and responsibility for assessing indoor health problems, identifying the actions needed to reduce the most significant problems, and setting guidelines, emissions limits, or other requirements that will be effective in reducing the health impacts of indoor sources. As discussed in Sections 4 and 6 of this report, many agencies' actions affect indoor air quality, and a few have limited authority over some aspect of indoor air quality, but no state (or federal) agency has the authority or mandate to conduct a comprehensive indoor air pollution mitigation program. Such a program is needed, and should be fully coordinated with activities of other agencies whose actions affect indoor air.

2. **Establish emission limits,** when needed, for indoor pollutant sources that pose excessive risks due to their indoor emissions. These might include air cleaners, building materials, furnishings, combustion appliances, and others. While ventilation authority exists in the Energy Commission and Cal/OSHA, no state agency has a direct mandate to establish emission limits for indoor sources for the purpose of reducing indoor exposure and risk. Establishment of such limits would better protect public health, and may reduce (but not eliminate) the amount of ventilation needed under certain circumstances in some buildings, thus saving energy. Compliance could be accomplished by requiring emissions testing through an independent laboratory certified by the state, and submittal of the data to the lead agency.

3. **Require manufacturers to submit building materials, furnishings, combustion appliances, consumer products, and other significant sources for emissions testing** by an independent laboratory certified by the state, and to report those results to the state and to the public. Also, require results to be transmitted to the public via product labeling or accompanying materials in language consumers can understand.

4. Implementation of a required test program could prove to be an effective approach, at least for reducing indoor pollutant levels in new buildings. A prototype emissions testing program has already been developed for state sustainable building projects: Section 01350 (State of California, 2002) is a testing and assessment protocol developed for reducing VOCs from building materials and furnishings, and is designed to protect human health. A partial list of products that meet this specification is available at http://ciwmb.ca.gov/GreenBuilding/Specs/EastEnd/. However, there is currently no requirement for state agencies or others to use these guideline emission specifications, and only limited incentive for them to do so. Other national and international emissions test protocols that are widely used also are available. ARB also has a consumer products test program with test methods (for VOC content) that could be adapted for a broader array of products. Currently, only a few laboratories have the chamber facilities and expertise to conduct such emissions tests. Thus, testing requirements would need to be phased in. However, required testing would prompt other laboratories to obtain appropriate chamber equipment and participate.

5. **Make children's health in schools, homes, and care institutions the top priority.** Implement the recommendations for schools in Section 7.2 of this report. In schools and public daycare centers, require the use of building materials that are certified to be low-emitting. Require that school HVAC sytems be quiet (under 45 decibels) and well maintained. Increase efforts to reduce children's exposure to environmental tobacco smoke. Increased education and outreach efforts to smoking parents and caretakers are needed to inform them of the health dangers of second-hand smoke, and the actions they should be taking to protect children under their care from these dangers.

6. **Develop indoor air quality guidelines** for homes, schools, offices, and institutional living quarters. These would largely identify "Best Practices" for the design, construction, operation and maintenance of public, commercial, school, and institutional buildings. In some cases, they might include the identification of healthful levels or "bright lines" for some pollutants to be used as goals for mitigation activities and "best practices", but would not have an associated compliance program. They should also include valid certification requirements for professionals directly involved in indoor air quality-related occupations; performance measures for buildings and appliances; and others. Full commissioning should be required for all new public, commercial, and institutional residential buildings, to assure that they are constructed and operate as intended, and that they provide acceptable indoor air quality. ASHRAE Guidelines (1993, 1996) provide basic guidance for building commissioning, but state requirements are needed.

7. **Amend building codes to address indoor air quality, with a focus on assuring adequate ventilation under all circumstances.** For example, unvented cook stoves, ovens, and combustion appliances should not be allowed in residences. They should be vented to the outdoors, such as through direct venting or an automatic (but quiet) exhaust fan that is activated when the appliance is turned on. Similarly, building codes should be established and enforced to prevent mold problems, residential ventilation issues, and others.

8. **Fund an outreach and education program focused on professionals, including** health professionals, teachers, school facility managers, and others who must be able to identify and remedy indoor air quality problems. Such individuals have many obligations, yet play a key role through their occupation in initial identification, prevention, and mitigation of indoor air quality problems. Most need more in-depth information and training on indoor air quality

than they typically have had. Training and technical assistance should be provided for the private sector to develop the skills and services needed for high-quality building commissioning, operation, and mainenance.

10. **Conduct more research on indoor air quality**. Several high priority areas are specifically identified in this report for further research. Because of the known serious health impacts of ambient PM and recent studies showing high emissions of PM from indoor sources, research on the health effects of indoor PM are a high priority. The health effects of terpene-ozone reaction products and the extent of people's exposures to them, as well as other indoor chemical reaction products, also are key areas warranting focused research. There are many new chemicals introduced into the product mix each year, yet few have had full health and exposure studies completed. The effects of more recently identified indoor chemicals, such as PBDEs, warrants further investigation. Improved methods and protocols to detect indoor dampness and hidden mold growth are also needed. Synergistic and cumulative health effects are suspected for a number of indoor pollutants with similar structures or properties, yet little research has been conducted in this area. Finally, mitigation approaches assumed to be effective have sometimes been found to be much less effective than anticipated; the effectiveness of recommended or required mitigation measures should be confirmed through appropriately designed studies to assure that the necessary reductions in exposure and risk will be achieved.

11. **Fund an Innovative Clean Air Technology program (ICAT) for indoor air quality** to foster the development and commercialization of legitimate, cost effective technologies that can improve IAQ. For example, improved low-noise ventilation technologies, improved air monitors and assessment tools, and effective low-noise air cleaners are needed. ARB's current ICAT program, focused on improving outdoor air quality through improved technology, has been very successful in bringing new technologies to commercialization in California, adding new options for reducing air pollution while also bringing jobs and investment into the state. An indoor air quality ICAT program would be expected to do the same.

All of these suggested mitigation options are feasible if appropriate mandates and resources are provided. The feasibility of individual measures, such as emission limits for a specific type of product, cannot be determined without substantial additional information. As discussed in previous sections of this report, alternative products or formulations are already available for some of the indoor sources of current concern. However, prior to taking any regulatory action, a more detailed assessment of the specific remedies available, including technological and economic feasibility, would be needed. Additionally, like ARB's current regulatory programs, any emission limitations or other mitigation measures would be developed with continuous discussion and review by stakeholders, the public, and other state agencies.

7.2 SOLUTIONS FOR SCHOOLS

The ARB and DHS recently completed a report on a statewide study entitled *Environmental Health Conditions in California's Portable Classrooms* (ARB/DHS, 2004). The study was required by the State Legislature (AB 2872 Shelley; HSC § 39619.6) as a result of concerns regarding reports of mold contamination, inadequate ventilation, elevated levels of volatile chemicals, excessive use of some pesticides, and other problems. The study was funded to help identify the extent of these problems and to determine whether those problems warranted response by the state and/or schools and school districts.

The study included kindergarten through 12th grade public schools. A large, representative sample of both portable and traditional classrooms was studied throughout the state. The results of this comprehensive study have been condensed into a report to the Legislature, and provide important information for state and local decision-makers regarding the degree to which California classrooms provide a safe, healthful, and productive learning environment for children. The report summarizes serious conditions identified in the study that need to be addressed at the State and local levels, and discusses options for improving conditions in both portable and traditional classrooms. The key results and recommendations included in the report are summarized below. The recommendations were developed in consultation with relevant state agencies, industries, school officials, and other interested stakeholders.

7.2.1 Problems Identified

The report identifies and addresses a number of environmental problems that were frequently found in classrooms throughout California. These problems were found in both portable (relocatable) and traditional (site-built) classrooms; however, some of the problems were found more frequently in portable classrooms. Government standards and guidelines that are designed to protect children in classrooms and other buildings are essentially lacking. Thus, in this study, results were compared to the most relevant environmental health guidelines and standards available, primarily from professional societies (such as those for ventilation and lighting) and government agencies, such as the Office of Environmental Health Hazard Assessment (OEHHA) and the California Department of Industrial Relations (Cal/OSHA). The primary problems found include:

- Inadequate ventilation with outdoor air during 40% of class hours, and seriously deficient ventilation 10% of the time. This is due largely to teachers turning off HVAC (heating, ventilating, and air-conditioning) systems because of excessive noise and to other factors such as closed outdoor air dampers and inadequate HVAC capacity.

- Temperature and humidity levels outside of professional standards for thermal comfort in about one-fourth of the classrooms.

- Formaldehyde air concentrations above guidelines for preventing acute eye, nose, and throat irritation in about 4% of the classrooms; and, nearly all classrooms exceeded formaldehyde guidelines for preventing long-term health effects, including cancer. Elevated formaldehyde is due primarily to the use of formaldehyde-containing building materials and furnishings.

- Noise levels in all classrooms exceeded the national voluntary acoustic standard for unoccupied classrooms of 35 decibels, a somewhat controversial standard that has not been adopted by any California agency. About one-half of the classrooms also exceeded 55 decibels, the level used by many communities in the state for their outdoor nuisance regulations. Excess noise was due primarily to noisy HVAC systems. Noisy lighting and noise from nearby outdoor activities also contributed. Excess noise directly impacts indoor air quality in the classrooms: when teachers turn off the systems due to noise, classrooms become stuffy and indoor pollutant levels rise.

- Lead, arsenic, and numerous pesticide residues in classroom floor dust; these residues are a concern because they can be inhaled, ingested, or absorbed through the skin (pesticides) by children, especially very young children who sit on the floor and put their hands in their mouths.

- Obvious mold in about 3% of classrooms; water stains, excess wall moisture, and other potential mold indicators in about one-third of classrooms; musty odors reported by 69% of teachers. These conditions are often attributable to inadequate maintenance.

- Lighting was inadequate in about one-third of the classrooms.

7.2.2 Report Recommendations

To address the breadth of problems identified and the many actions needed at all levels to resolve them, a total of 16 recommendations are discussed in the report to the Legislature. These are presented in two groups in the report. Group 1 includes high priority, high benefit actions that can be achieved at relatively low cost and should be accomplished in the near term, while Group 2 recommendations, also a priority, will require a longer timeframe and/or more substantial resources to accomplish. The recommendations fall into four general approaches needed to remedy and prevent the problems seen. The state should:

- Direct and assist schools to comply with state regulations, especially workplace regulations (Cal/OSHA) related to building operation and maintenance.

- Develop and promote "Best Practices" for design, construction, operation, and maintenance of school facilities.

- Improve support (both funding and training) for school facilities and staff.

- Establish guidelines and standards for school environmental health that are protective of children.

The specific Group I recommendations are:

- Schools, districts, and the state should assure that all school buildings meet all relevant state regulations, especially the Cal/OSHA workplace regulations regarding ventilation, sanitation and water intrusion, and illness and injury prevention. Many schools do not meet Cal/OSHA occupational health requirements.

- Schools and school districts should conduct "self-assessments of basic health and safety conditions, similar to the Facility Self-inspection Program undertaken by the Los Angeles Unified School District (LAUSD). Checklists are available on the web.

- The state should require schools to develop indoor environmental quality management plans. The U.S. Environmental Protection Agency's *IAQ Tools for Schools Program* provides guidance and free kits to accomplish this. The kit is available free of charge at http://www.U.S. EPA.gov/iaq/schools/.

- The state should establish a policy to incorporate "Best Practices" into the design, construction, operation, and maintenance of California schools, especially the measures developed by the Collaborative for High Performance Schools (CHPS). The CHPS *Best Practices Manual* at http://www.chps.net/ provides guidance for measures that will improve schools while also saving energy and reducing long-term costs.

- State-level review by the Division of the State Architect of the designs for new schools should be expanded to include elements such as ventilation systems and building materials in addition to current elements such as fire and life-safety provisions.

- Portable classrooms (and traditional classrooms) should be sited correctly, away from busy roadways and with proper drainage.

- The state should implement an interim requirement for new classrooms of a maximum noise level of 45 decibels, unoccupied, until a specially-convened task force can determine an appropriate level for California schools (see below).

Group 2 recommendations specify that:

- The state and school districts should assure stable, long-term funding mechanisms and sources for both construction and preventive maintenance; currently, funding fluctuates from year to year, especially for the Deferred Maintenance Program.

- The state should develop and offer focused training programs for school facility managers, custodial staff, and teachers, in cooperation with interested organizations; those closest to the classrooms often are not aware of current "best practices" for operation and maintenance of classrooms. A concerted, ongoing training program could go far to improve conditions in classrooms.

- Integrated Pest Management Programs should be implemented at all schools.

- Older portable classrooms should be retired when they become unserviceable or do not provide an adequate learning environment for children.

- The state and school districts should develop and require full new building commissioning procedures.

- The state should improve its database of school facilities: currently, there is no complete database on the condition, location, or even number, of school buildings.

- The state should convene a task force of experts to develop a California indoor noise guideline or standard for K-12 schools.

- The state should develop chemical exposure guidelines or standards for classrooms that are protective of children and teachers.

- Portable classrooms should be re-designed from the ground up. Several groups are producing new prototypes that use an integrated "whole building" approach; these should be supported through the demonstration phase to evaluate design changes that provide substantive improvements over older portables.

Some actions have already been taken to begin to address these problems; however, they constitute only a first step toward realizing actual improvements in school conditions. Only a small percentage of schools and districts have actively pursued the many tools that are readily available to them to help improve the school environment. The CHPS' *Best Practices Manual*, U.S. EPA's *IAQ Tools for Schools Kits*, and the LAUSD's "Safe School Inspection Guidebook" are all available on the Internet free of charge, yet the number of California schools utilizing

each of these tools is small. A proactive effort to implement the recommendations of the report is needed.

The complete *Report to the Legislature on Environmental Health Conditions in California's Portable Classrooms* is available at ARB's website at http://www.arb.ca.gov/research/indoor/pcs/pcs.htm.

8. SUMMARY

Indoor air pollution poses a significant health risk to Californians, in addition to the known risks from outdoor pollution. Indoor pollutant sources emit gases and particles known to trigger asthma attacks and cause cancer, heart and lung disease, and immediate irritant and neurological effects such as eye and throat irritation and headache. Indoor pollution has repeatedly been ranked in the "High Risk" categories in both federal and state comparative risk projects. Some of the known risks in California include:

- Indoor air pollution includes many asthma triggers. It has been implicated by national scientists as a factor in the serious increase in asthma observed in recent decades.

- It is estimated that about 230 excess cancers per year may occur in California due to indoor carcinogens from residential and consumer sources, such as formaldehyde. This approaches the 260 estimated excess cancer cases per year from diesel exhaust and equals about two-thirds of the total outdoor cancer burden. Environmental tobacco smoke also adds significant cancer risk. Also, preliminary estimates show that radon gas from certain soils and rock may contribute to about 1500 excess lung cancers per year.

- Each year, accidental carbon monoxide poisoning from indoor sources causes about 20-26 accidental deaths, hundreds of avoidable emergency room visits, and hundreds to thousands of cases of avoidable illness.

- Many VOCs, especially formaldehyde, are typically found at elevated levels indoors, and those levels sometimes exceed health-based guideline levels, such as acceptable cancer risk levels.

- Indoor sources of PM may be partly responsible for the large numbers of premature deaths, hospitalizations, emergency room visits, and increased respiratory disease associated with PM exposures in California each year, and may contribute to these effects beyond the levels quantified in the outdoor epidemiology studies.

- Biological contaminants can cause communicable disease, hypersensitivity reactions, and even toxic responses, in addition to their widely recognized role as asthma and allergy triggers in indoor environments. Indoor mold problems have increased over the last decade.

Indoor pollutants significantly impact people's health because there are many indoor sources of pollutants, which often result in elevated indoor concentrations. Additionally, Californians, like others, spend about 87% of their time indoors, on average. Infants and young children spend the most time indoors, most of it in their homes. Several scientists have calculated that indoor emissions are about 1000 times more likely to be breathed than outdoor emissions.

Indoor pollution is estimated to cost California's economy $45 billion each year due to medical costs, lost worker productivity, loss of life, and related factors. This estimate is derived from only partial costs of cancer, respiratory disease, cardiovascular disease, and sick building symptoms. This is believed to be an underestimate; the total cost is likely much higher.

Despite the high health and economic costs of indoor pollution, no state or federal agency has explicit authority to regulate indoor sources of pollution to protect building occupants from

harmful exposures. Indoor pollution remains the only major environmental health problem that does not have the benefit of a focused risk reduction program.

Many actions could be taken to reduce indoor pollution at relatively low cost that would reap large health benefits. Options for mitigation include the development of emission limitations for building materials, furnishings, consumer products, and appliances; improvements in building codes; focused outreach and education programs; improved technologies; and adherence to current "Best Practices" and other guidelines. Substitute products are available for many products and materials that currently emit high levels of pollutants; thus, emission reductions should be achievable in many products with little impact on consumers and homeowners. This would go far to improve indoor air quality in schools and homes, which require a focused effort because little has been done to improve those indoor environments. Also, the most sensitive members of the population–children, the elderly, and the infirm–spend most of their time there. Focused public education, improved appliance standards, and modified building codes would also foster improved indoor air quality in homes and schools. Following current "Best Practices" in design, construction, operation and maintenance is a non-regulatory approach that could yield large gains in indoor health in all types of buildings. Increased effort to publicize and enforce existing Cal/OSHA regulations in schools and non-industrial workplaces would also go far to reduce indoor pollution in those environments; those regulations are in place, but are often not followed.

Perhaps most importantly, a comprehensive management system with responsibility for indoor air quality is needed to combine and coordinate the options indicated above into a cohesive, effective program. That program should include a more detailed prioritization of mitigation actions, careful assessment of the technical and economic feasibility of specific actions, and continued discussion with, and input from, interested stakeholders and members of the public. Such a program also should be closely coordinated with state agencies whose programs include some existing authority related to indoor air quality, especially Cal/OSHA and the Energy Commission, to assure consistency and avoid duplication of effort

9. REFERENCES

AB 13, 1995. Assembly Bill 13, author Terry Friedman, California Labor Code Section 6404.5.

Abt E, Suh HH, Catalano P, and Koutrakis P, 2000. Characterization of indoor particle sources: a study conducted in the metropolitan Boston area. *Environmental Health Perspectives* **108(1)**: 3579-3587.

Abt E, Suh HH, Allen G, and Koutrakis P, 2001. Relative contribution of outdoor and indoor particle sources to indoor concentrations. *Environ Science and Technol* **34**: 3579-3587.

ACGIH (American Conference of Governmental Industrial Hygienists), 1999. Developing a Sampling Plan. In Bioaerosols: Assessment and Control. Editor: J Macher. American Conference of Governmental Industrial Hygienists, Cincinnati, OH. pp

Adams W, 1993. Measurement of breathing rate and volume in routinely performed daily activities. Final Report, ARB Contract no. A033-205.

Addiss SS, Alderman NO, Brown DR, Each CN, and Wargo J, 1999. Pest control practices in Connecticut public schools. North Haven, CT: Environment and Human Health, Inc.

AGA (American Gas Association), 2003. Combustion Emissions from Gas Ranges. Available from: http://www.aga.org/Content/NavigationMenu/Operations_and_Engineering/Codes_and_Standards1/Fact_Sheets_and_Reports/twfacts9902.pdf.

AHAM (Association of Home Appliance Manufacturers), 2004. Clean Air Delivery Rate: Frequenty Asked Questions. http://www.cadr.org/consumer/faq.html.

AIHA (American Industrial Hygiene Association), 1996. Field Guide for the Determination of Biological Contaminants in Environmental Samples. Editors: HK Dillon, PA Heinsohn, JD Miller. American Industrial Hygiene Association, Fairfax, VA. pp 37-95, 119-130.

Akazawa, M, Sindelar, JL, Paltiel, .D, 2003. Economic costs of influenza-related work absenteeism, *Value Health* **6**:107-115.

Akland G, and Whitaker DA, 2000. Characterizing the sources of human exposure to proposition 65 substances. Research Triangle Institute, NC. Final report to California Public Health Foundation, Public Health Trust, Grant No. 543A-8803-G11299.

ALA (American Lung Association), 1997. Residential Air cleaning Devices: Types, Effectiveness, and Health Impact. Available from: http://www.lungusa.org/site/pp.asp?c=dvLUK9O0E&b=39289.

ALA, 2004. Health Home Program. Available from: http://www.healthhouse.org/.

Alevantis L, 2003. Building material emissions study. Final Report to California Integrated Waste Management Board. http://www.ciwmb.ca.gov/GreenBuilding/Specs/Section01350/METStudy.htm.

Allen RJ, Wadden RA, and Ross ED, 1978. Characterization of potential indoor sources of ozone. *J Am Industrial Hygiene Assoc* **39**: 466-471.

Anderson HR, Spix C, Medina S, Schouten JP, Castellsague J, Rossi G, Zmirou D, Touloumi G, Wojtyniak B, Ponka A, Bacharova L, Schwartz J, and Katsouyanni K, 1997. Air pollution and daily admissions for chronic obstructive pulmonary disease in 6 European cities: results from the APHEA project. *European Respiratory Journal* **10**: 1064-1071.

Apte MG and Daisey JM, 1999. VOCs and "sick building syndrome": application of a new statistical approach for SBS research to U.S. EPA BASE study data. *Indoor Air* **1**: 117.

Apte MG, Gundel LA, Dod RL, Russell ML, Singer BC, Sohn MD, Sullivan DP, Chang GM and Sextro RG. Indoor Measurements of Environmental Tobacco Smoke, Final Report to the Tobacco Related Disease Research Program. Lawrence Berkeley National Lab report LBNL-49148. Berkeley, CA. March 2004.

ARB (California Air Resources Board), 1986. Staff Report: Initial Statement of Reasons for Proposed Rulemaking. Public Hearing to Consider the Adoption of a Regulatory Amendment Identifying Asbestos as a Toxic Air Contaminant. California Air Resources Board, agenda item no. 86-4-1.

ARB, 1987. Indoor Air Quality and Personal Exposure: Briefing Paper. Research Division, California Air Resources Board, May 1987.

ARB, 1989. Reducing Exposures to Indoor Air Pollutants in California: Existing authorities and Recommended Actions. Final Report, v.1, and appendices, v. 2. Research Division, Sacramento, CA.

ARB, 1991. Formaldehyde in the Home, Indoor Air Quality Guideline no. 1. Research Division.

ARB, 1992. Final report on the identification of formaldehyde as a toxic air contaminant, Part B, Health Assessment, Stationary Source Division.

ARB, 1994a. Benzo(a)pyrene as a Toxic Air Contaminant, Part A. Exposure Assessment. Sacramento, CA. Executive Summary at: http://www.oehha.ca.gov/air/toxic_contaminants/html/benzo%5Ba%5Dpyrene.htm.

ARB, 1994b. Combustion Pollutants in the Home. Indoor Air Quality Guideline no. 2, and Supplement. Sacramento, CA. Available from: http://www.arb.ca.gov/research/indoor/guidelines1.htm.

ARB, 1997a. Toxic air contaminant identification list–summaries. Stationary Source Division, California Air Resources Board. http://www.arb.ca.gov/toxics/tac/toctbl.htm.

ARB, 1997b. Woodburning Handbook: How to Burn More Efficiently in Your Stove or Fireplace and Produce Less Air Pollution. Available from: http://www.arb.ca.gov/cap/handbooks/wood_burning/wood_burning_handbook.pdf.

ARB, 1998. Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant, Part A, Exposure Assessment, Appendix D, Californians' Indoor and Total Air Exposures t Diesel Exhaust Particles. April. Sacramento, CA.

ARB, 2000a. Risk reduction plan to reduce particulate matter emissions from diesel-fueled engines and vehicles. Stationary Source Division, California Air Resources Board, http://www.arb.ca.gov/diesel/documents/rrpFinal.pdf

ARB, 2000b. Indoor Air Quality Risk Reduction in the 21st Century, Symposium Proceedings, Sacramento, CA May 2000. http://www.arb.ca.gov/research/indoor/proceedings.PDF

ARB, 2000c. Air Cleaning Devices for the Home: Frequently Asked Questions. Fact Sheet. Research Division. http://www.arb.ca.gov/research/indoor/guidelines1.htm

ARB, 2001a. Policies and actions for environmental justice, approved December 13, 2001. California Air Resources Board. http://www.arb.ca.gov/ch/programs/ej/ej.htm.

ARB, 2001b. Chlorinated Chemicals in Your Home, Indoor Air Quality Guideline no. 3. Research Division. http://www.arb.ca.gov/research/indoor/guidelines1.htm

ARB, 2001c. Composite Wood Products, Fact Sheet. Available from: http://www.arb.ca.gov/ch/factsheets/formaldehyde.pdf.

ARB, 2003. List of the Current Rules in Each of the 35 Air Districts. Available from: http://www.arb.ca.gov/drdb/drdbltxt.htm.

ARB, 2004. Formaldehyde in the Home (Updated). Indoor Air Quality Guideline no. 1. http://www.arb.ca.gov/research/indoor/guidelines1.htm.

ARB, 2005a. News release, January 20, 2005, "ARB Warns, Danger from Popular "Air Purifying" Machines." http://www.arb.ca.gov/newsrel/nr012005.htm.

ARB, 2005b. Air Cleaning Devices For the Home: Frequently Asked Questions. Fact sheet, updated February 4, 2005. http://www.arb.ca.gov/research/indoor/acdsumm.pdf.

ARB/DHS, 2003. Report to the Legislature: Environmental Health Conditions in California's Portable Classrooms, Nov. 2003. http://www.arb.ca.gov/research/indoor/pcs/pcs.htm.

ARB/OEHHA, 1994. Benzo(a)pyrene as a toxic air contaminant. Approved by the Scientific Review Panel.

ARB/OEHHA, 2000. Staff Report, Adequacy of California Ambient Air Quality Standards: Children's Environmental Health Protection Act. December.

ARB/OEHHA, 2002. Staff Report: Public Hearing to Consider Amendments to the Ambient Air Quality Standards for Particulate Matter and Sulfates. Air Resources Board and Office of Environmental health Hazard Assessment, Sacramento, CA.

ARB/OEHHA, 2003. Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant. (Draft Report, Parts A and B). Available from: http://www.arb.ca.gov/toxics/ets/dreport/dreport.htm.

ARB/OEHHA, 2004. Draft Report on the Ozone Ambient Air Quality Standard. http://www.arb.ca.gov/research/aaqs/ozone-rs/ozone-draft-rpt.htm.

ARB/OEHHA, 2004/2005. Revised Draft for SRP Review, Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant, Parts A and B.

Arbes SJ Jr, Cohn RD, Yin M, Muilenberg ML, Burge HA, Friedman W, Zeldin DC, 2003. House dust mite allergen in US beds: results from the First National Survey or Lead and Allergens in Housing. *J Allergy Clin Immunol* **111(2)**: 408-414.

Arbes SJ Jr, Cohn RD, Yin M, Muilenberg ML, Friedman W, Zeldin D, 2004. Dog allergen (Can f 1) and cat allergen (Fel d 1) in US homes: results from the National Survey of Lead and Allergens in Housing. *J Allergy Clin Immunol* **114(1)**: 111-117.

Aris RM, Christian D, Hearne PQ, Kerr K, Finkbeiner WE, and Balmes JR, 1993. Ozone-induced airway inflammation in human subjects as determined by airway lavage and biopsy. *American Review of Respiratory Diseases* **148**: 1363-1372.

ASHRAE, 1981. Ventilation for Acceptable Indoor Air Quality. ASHRAE Standard 62-1981. American Society of Heating Refrigerating and Air conditioning Engineers, Atlanta, GA.

ASHRAE, 1993. Guideline 4-1993 -- Preparation of Operating and Maintenance Documentation for Building Systems. Atlanta, GA.

ASHRAE, 1996. Guideline 1-1996. The HVAC Commissioning Process. Atlanta, GA.

ASHRAE, 2003a. Standard 62.2-2003, Ventilation and Acceptable Indoor Air Quality in Low-Rise Residential Buildings. Atlanta, GA. http://www.ashrae.org.

ASHRAE, 2003b. Standard 62-2001, "Ventilation for Acceptable Indoor Air Quality", plus Addenda approved by ASHRAE's Board of Directors since the 2001 adoption. Atlanta, GA.

ASHRAE, 2004. Standard 55-2004, "Thermal Environmental Conditions for Human Occupancy", supersedes ASHRAE Standard 55-1992. Atlanta, GA.

Atkinson R, and Arey J 2003. Gas-phase tropospheric chemistry of biogenic volatile organic compounds: A review. *Atmospheric Environment* **37 (supplement 2)**, 197-219

ATS (American Thoracic Society), 1997. Achieving Healthy Indoor Air. *American J Respiratory and Critical Care Medicine* **156**: s33-s64.

ATS (American Thoracic Society), 2003a. American Thoracic Society Statement: Occupational Contribution to the Burden of Airway Disease. *American J Respiratory and Critical Care Medicine* **167**: 787-797.

ATS (American Thoracic Society), 2003b. Workshop on Lung Disease and the Environment: Where Do We Go from Here? *American J Respiratory and Critical Care Medicine* **168**: 250-254.

ATSDR, 1999a. Toxicological profile for lead. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Available from: http://www.atsdr.cdc.gov/toxprofiles/tp13.html.

ATSDR, 1999b. Toxicological profile for mercury. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Available from: http://www.atsdr.cdc.gov/toxprofiles/tp46.html.
ATSDR, 2001. Toxicological profile for asbestos. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Available from: http://www.atsdr.cdc.gov/toxprofiles/tp61.html.

ATSDR, 2002. Health Consultation: Mercury Exposures from 3M Tartan Brand Floors. Available from: http://www.atsdr.cdc.gov/HAC/PHA/westerville/wes_p1.html.

Atkinson R, and Arey J, 1989. Atmospheric lifetime and fate of benzo(a)pyrene. In: Lifetimes and Fates of Toxic Air Contaminants in California's Atmosphere, Final Report, Contract No. A732-107, California Air Resources Board.

Atkinson RW, Anderson HR, Sunyer J, Ayres J, Baccini M, Vonk JM, Boumghar A, Forastiere F, Forsberg B, Touloumi G, Schwartz J, and Katsouyanni K, 2003a. Acute Effects of Particulate Air Pollution on Respiratory Admissions, In: Health Effects Institute Special Report: Revised Analyses of Time-Series Studies of Air Pollution and Health.

Atkinson R, and Arey J, 2003. Gas-phase tropospheric chemistry of biogenic volatile organic compounds: a review. *Atmospheric Environment* **37 Supplement No. 2**: S197-S1219.

Auinger P, Lanphear BP, Kalkwarf HJ, and Mansour ME, 2003. Trends in otitis media among children in the United States. *Pediatrics* **112**(3 Pt 1): 514-20.

AVMA (American Veterinary Medical Association), 1997. Results of the AVMA survey of US pet-owning households on companion animal ownership. American Veterinary Medical Association, Schaumberg, IL.

Avol E, Colome S, Estes M, Navidi W, Rappaport E, Lurmann F, Hering S, 1996. Residential Microenvironmental and Personal Sampling Project for Exposure Classification, Final Report for ARB Contract no. 92-317, University of Southern California, July 1996.

Avol EL, Navidi WC, and Colome SD, 1998. Modeling ozone levels in and around Southern California homes. *Environ Sci Technol* **32**: 463-468.

Balmes JR, Chen LL, Sannell C, Tager I, Christian D, Hearne PQ, Kelly T, and Aris RM, 1996. Ozone-induced decrements in FEV and FVC do not correlate with measures of inflammation. *American J Respiratory and Critical Care Medicine* **153**: 904-909.

Barck C, Sandstrom T, Lundahl J, Hallden G, Svartengren M, Strand V, Rak S, and Bylin G, 2002. Ambient level of NO_2 augments the inflammatory response to inhaled allergen in asthmatics. *Respiratory Medicine* **96**: 907-917.

Bearer C, 1995. How are children different from adults? *Environmental Health Perspectives* **103** (Supplement 6): 7-12.

Bearg DW, 1993. Indoor Air Quality and HVAC Systems. Lewis Publishers, Boca Raton. Chapter 1.

Becker S, and Soukup JM, 1999. Effect of nitrogen dioxide on respiratory viral infection in airway epithelial cells. *Environmental Research* **81**: 159-166.

Belanger K, Beckett W, Triche E, Bracken MB, Holford T, Ren P, McSharry JE, Gold DR, Platts-Mills TA, Leaderer BP, 2003. Symptoms of wheeze and persistent cough in the first year of life: associations with indoor allergens, air contaminants and maternal history of asthma. *American Journal of Epidemiology* **158(3)**:195-202.

Bennet DH, McKone TE, Evans JS, Nazaroff WW, Margni MD, Jolliet O, and Smith KR, 2002. Defining intake fraction. *Environmental Science and Technology* **May 1, 2002**: 207A-211A.

Blomberg A, Krishna MT, Helleday R, Soderberg M, Ledin M, Kelly FJ, Frew AJ, Holgate ST, and Sandstrom T, 1999. Persistent airway inflammation but accomodated antioxidant and lung function responses after repeated daily exposure to nitrogen dioxide. *American J Respiratory and Critical Care Medicine* **159**: 536-543.

Bobak M, 2000. Outdoor air pollution, low birth weight, and prematurity. *Environmental Health Perspectives* **108**(2): 173-176.

Bobak M, and Leon DA, 1998. Air pollution and infant mortality: the effects are specific for respiratory causes in the post-neonatal period. *Epidemiology* **9**: S58.

Boe AB, 1999. Field observations from SGC/NC homes in Oregon. Oregon State University Energy Extension Program. 1997-99. As cited in Lubliner and Gordon, 1990.

Boeniger MF, 1995. Use of ozone generating devices to improve indoor air quality. *J American Industrial Hygiene Association* **56**: 590-598.

Bollinger ME, Eggleston PA, Flanagan E, Wood RA, 1996. Cat antigen in homes with and without cats may induce allergic symptoms. *Journal of Allergy and Clinical Immunology* **97(4)**: 907-914.

Bonita R, Duncan J, Truelsen T, Jackson RT, and Beaglehole R, 1999. Passive smoking as well as active smoking increases the risk of acute stroke. *Tobacco Control* **8(2)**: 156-160.

Bornehag CG, Blomquist G, Gyntelberg F, Järvhom B, Malmberg P, Nordvall L, Nielsen A, Pershagen G, Sundell J, 2001. Dampness in buildings and health. Nordic interdisciplinary review of the scientific evidence on associations to "dampness" in guildings and health effects (NORDDAMP). *Indoor Air* **11**:72-86.

Bornehag, CG, Sundell J, Bonini S, Custovic A, Malmber P, Skerfving S, Sigsgaard T, and Verhoeff A, 2004. Dampness in buildings as a risk factor for health effects, EUROEXPO: a multidisciplinary review of the literature (1998-2000) on dampness and mite exposure in buildings and health effects. *Indoor Air* **14(4)**: 243-257.

Boudreau DR, Spadafora MP, Wolf LR, and Siegel E, 1994. Carbon monoxide levels during indoor sporting events-Cincinnati, 1992-1993. MMWR available at http://www.cdc.gov/mmwr/preview/mmwrhtml/00023631.htm.

Boutin-Forzano S, Adel N, Gratecos L, Jullian H, Garnier JM, Ramadour M, Lanteaume A, Hamon M, Lafay V, and Charpin D, 2004. Visits to the emergency room for asthma attacks and short-term variations in air pollution. *Respiration* **71**: 134-137.

Bradman MA, Harnly ME, Draper W, Seidel S, Teran S, Wakeham D, and Neutra R, 1997. Pesticide exposures to children from California's Central Valley: results of a pilot study. *J Exposure Analysis and Environmental Epidemiology.* **7(2)**: 217-234.

Bramley TJ, Lerner D, and Sarnes M, 2002. Productivity losses related to the common cold. *J Occup Environ Med* **44**:822-829.

Brauer M, Blair J, and Vedal S, 1996. Effect of ambient ozone exposure on lung function in farm workers. *J Respiratory and Critical Care Medicine* **154**: 981-987.

Brauer M, Hirtle R, Lang B, and Ott W, 2000. Assessment of indoor fine aerosol contributions from environmental tobacco smoke and cooking with a portable nephelometer. *J Exposure Analysis and Environmental Epidemiology* **10**: 136-144.

Bridges, B, 2002. Fragrance: emerging health and environmental concerns. *Flavour and Fragrance Journal* **17**: 361-371.

BRFSS, 2001. Behavior Related Factors Survey. California Department of Health Services, Cancer Surveillance Section.

Brown SK, 1999. Chamber assessment of formaldehyde and voc emissions from wood-based panels. *Indoor Air* **9**: 209-215.

Brown, SK, 1999b. Assessment of pollutant emissions from dry-process photocopiers. *Indoor Air* **(9)**: 259-267.

Brown SK, Sim MR, Abramson MJ, and Gray CN, 1994. Concentrations of volatile organic compounds in indoor air-a review. *Indoor Air* **4**: 123-134.

Brugge D, Vallarino J, Ascolillo L, Osgood N-D, Steinbach S, and Spengler J, 2003. Comparison of multiple environmental factors for asthmatic children in public housing. *Indoor Air* **13**: 18-27.

Brunekreef B, Hoek G, Breugelmans O, and Leentvaar M, 1994. Respiratory effects of low-level photochemical air pollution in amateur cyclists. *J Respiratory and Critical Care Medicine* **150**: 962-966.

BSC, 2003. BSC Building America Performance Criteria. Westford, MA. Available from: http://www.buildingscience.com/buildingamerica/targets.htm.

Burnett RT, Brook JR, Yung WT, Dales RE, and Krewski D, 1997. Association between ozone and hospitalization for respiratory diseases in 16 Canadian cities. *Environmental Research* **72**: 24-31.

Burnett RT, and Goldberg MS, 2003. Size-fractionated particulate mass and daily mortality in eight Canadian cities in Health Effects Institute Special Report: Revised Analyses of Time-Series Studies of Air Pollution and Health.

Burton L, 2000. Airborne particulate matter within 100 randomly selected office buildings in the United States (BASE). Proceedings of Healthy Building 2000, 157-163.

Cal/EPA, 1994. A guide for reviewing environmental policy studies, Moss S, McCann R, Feldman M. A Handbook for the California Environmental Protection Agency.

Carslaw N, 2003. New directions: where next with indoor air measurements? *Atmospheric Environment* **37**: 5645-5646.

Carpi A, and Chen YF, 2001. Gaseous Elemental Mercury as an Indoor Air Pollutant. *Environmental Science and Technology* **35**: 4170-4173.

CBPCA (California Building Performance Contractor Association), 2003a. California Home Performance UPDATE, September/October. San Francisco, CA. Available from: http://www.calhomeperformance.org/pdfs/newslettersep-oct03.pdf.

CBPCA, 2003b. PY 2002 - 2003 CPUC Energy Efficiency Programs Quarterly Report Narrative. Available from: http://www.cbpca.org/reports/CBPCA_172-02_Q3_2003.doc.

CCC (California Commissioning Collaborative), 2004. Mission Statement. Sacramento, CA. Available from: http://www.cacx.org/.

CCRP, 1994. Toward the 21st Century: Planning for the Protection of California's Environment. Berkeley, CA: Office of Environmental Health Hazard Assessment (OEHHA). Available from: http://www.oehha.ca.gov/multimedia/comprisk.html.

CDC, 1982. Perspectives in Disease Prevention and Health Promotion Carbon Monoxide Intoxication – A Preventable Environmental Health Hazard. *MMWR* **31(39)**: 529-531.

CDC, 1995. Mercury Exposure in a Residential Community—Florida, 1994. *MMWR* **44**(23): 436-437, 443. Available from: http://www.cdc.gov/mmwr/preview/mmwrhtml/00037313.htm.

CDC, 1997. Screening young children for lead poisoning: guidance for state and local public health officials. Atlanta: US Department of Health and Human Services, CDC Childhood Lead Poisoning Prevention Program. Available from URL: http://www.cdc.gov/nceh/lead/guide/guide97.htm.

CDC, 2000. Pulmonary Hemorrhage/Hemosiderosis Among Infants—Cleveland, Ohio, 1993-1996. *MMWR* **49**: 180-184.

CDC, 2003. Self-reported asthma prevalence and control among adults – United States, 2001. *MMWR* **52**: 381-384.

CDC, 2004. Basic information about SARS. Fact Sheet. http;//www.cdc.gov/ncidod/sars/factsheet.htm.

CDC, 2004. Adult Cigarette Smoking in the United States: Current Estimates. Fact Sheet. May 2004. Office on Smoking and Health National Center for Chronic Disease Prevention and Health Promotion. Atlanta, GA. Available at: http://www.cdc.gov/tobacco/factsheets/AdultCigaretteSmoking_FactSheet.htm.

CEC, 1994. California's Energy Efficiency Standards and Indoor Air Quality. Publication P400-94-003. Sacramento, CA.

CEC, 2002. Acceptance Requirements for Nonresidential Buildings: Nonresidential Quality Assurance Project. (P400-02-010). Available from: http://www.energy.ca.gov/2005_standards/documents/2002-04-22_workshop/2002-04-11_ACCEPTANCE.PDF.

CEC, 2003a. 2005 Building Energy Efficiency Standards: Express Terms – 15 Day Language Standards for Residential and Nonresidential Buildings. (P400-03-001ET15). Available from: http://www.energy.ca.gov/2005_standards/rulemaking/documents/15-day_language/2003-10-21_400-03-001-ET15F.PDF.

CEC, 2003b. AB 549 Proceeding. Assembly Bill 549 (Statutes of 2001, Chapter 905, Longville). Sacramento, CA. Available from: http://www.energy.ca.gov/ab549/index.html.

CEC, 2004. Reference Specifications for Energy and Resource Efficiency, Contract Number 500-98-027. San Francisco, CA: Eley Associates. Available from: http://www.energy.ca.gov/reports/2004-05-24_500-04-015.PDF.

Chalupa DC, Morrow PE, Oberdorster G, Utell MJ, and Frampton MW, 2004. Ultrafine particle desposition in subjects with asthma. *Environmental Health Perspectives* **112(8):** 879-882.

Chang JCS, Fortmann R, Roache N, and Lao H, 1999. Evaluation of low-VOC latex paints. *Indoor Air* **9**: 253-258.

Chauhan AJ, Inskip HM, Linaker CH, Smith S, Schreiber J, Lohnston SL, and Holgate ST, 2003. Personal exposure to nitrogen dioxide (NO_2) and the severity of virus-induced asthma in children. *Lancet* **361:** 1939-1944.

CHBA (Canadian Home Builders Association), 2004. The R-2000 Program. http://r2000.chba.ca/What_is_R2000/R2000_program.php.

CHIS, 2003. Asthma in California: Findings from the 2001 California Health Interview Survey. Available from: http://www.healthpolicy.ucla.edu.

CHPS, 2001. Best Practices Manual. Available from: www.chps.net.

Chuang JC, Mack GA, Stockrahm JW, Hannan SW, Bridges C and Kuhlman MR, 1988. Field evaluation of sampling and analysis for organic pollutants in indoor air. Prepared by Batelle Columbus Laboratories for the U.S. EPA, EPA Report No. 600/4-88/028.

Chuang JC, Callahan PJ, Lyu CW, and Wilson NK, 1999. Polycyclic aromatic hydrocarbon exposures of children in low-income families. *J Exposure Analysis and Environmental Epidemiology* **2**: 85-98.

Churchill R, 1997. Radon mapping – Santa Barbara and Ventura Counties. *California Geology* **50(6)**: 167-177.

Churchill RK, and Youngs LG, 1993. The Department of Health Services Elementary School Radon Survey: Relationship between geology, soil and indoor radon. California Department of Conservation.

City of Hayward, 2002. General Plan, Appendix O: Opportunities for Energy Conservation in Residential Development. Available from: http://www.ci.hayward.ca.us/about/generalplan/xAppendix-O.pdf.

Clausen G. 2004. Ventilation filters and indoor air quality: a review of research from the International Centre for Indoor Environment and Energy. *Indoor Air* **14**: Suppl 7:202-7.

Clayton AC, Pellizzari ED, Whitmore RW, Quackenboss JJ, Adgate J, and Sefton K, 2003. Distributions, associations, and partial aggregate exposure of pesticides and polynuclear aromatic hydrocarbons in the Minnesota Children's Pesticide Exposure Study (MNCPES). *J Exposure Analysis and Environmental Epidemiology* **13(2)**: 100-111.

Clayton JC, Perritt RL, Pellizzari ED, Thomas KM, Whitmore RW, Wallace LA, Ozkaynak H, and Spengler JD, 1993. Particle total exposure assessment methodology (PTEAM) Study: distribution of aerosol and elemental concentrations in person, indoor, and outdoor air samples in a Southern California community. *J Expos Anal and Environ Epidem* **3(2)**: 227-250.

Cohen MA, Ryan PB, Yanagisawa Y, Spengler JD, Ozkaynak H, and Epstein PS, 1989. Indoor/outdoor measurements of volatile organic compounds in the Kanawha Valley of West Virginia. *J Air Pollution Control Association* **39**: 1086-1093.

Cohen AJ, Anderson HR, Ostro B, Pandey KD, Krzyzanowski M, Kuenzli N, Gutschmidt K, Pope CA, Romieu I, Samet JM, and Smith KR, 2004. *Mortality Impacts of Urban Air Pollution, in* Ezzati M, Rodgers AD, Lopez AD, Murray CJL (eds) Comparative Quantification of Health Risks: Global and Regional Burden of Disease due to Selected Major Risk Factors. Geneva: World Health Organization, Vol 2 of 3, pp.1353-1433.

Conner TL, Norris GA, Landis MS, and Williams RW, 2001. Individual particle analysis of indoor, outdoor, and community samples from the 1998 Baltimore particulate matter study. *Atmospheric Environment* **35**: 3935-3946.

Consumers Union, 2003a. Air cleaners: behind the hype. *Consumer Reports*, October, pp.26-29.

Consumers Union, 2003b. Vacuum cleaners: when more isn't better. *Consumer Reports,* November.

Consumers Union, 2004. Cannister vacuum cleaners. *Consumer Reports*. May.

Cook M, Simon PA, and Hoffman RE, 1995. Unintentional Carbon Monoxide Poisoning in Colorado, 1986 through 1991. *Amer J Public Health* **85(7)**: 988-990.

Cooper SD, Raymer JH, Pellizzari ED, and Thomas KW, 1995. The identification of polar organic compounds found in consumer products and their toxicological properties. *Journal of Exposure and Environmental Epidemiology* **5(1)**: 57.

Corn M, Crump K, Farrar DB, Lee RJ, and McFee DR, 1991. Airborne concentrations of asbestos in 71 school buildings. *Regulatory Toxicology and Pharmacology* **13(1)**: 99-114.

Cothern CR, 1987. Environmental Radon. *Environmental Science Research Series* **35**: 1-28.

Cox CS, 1995. Stability of airborne microbes and allergens. Bioaerosols Handbook, Eds., Cox and Wathes, Lewis Publishers, Boca Raton, FL.

Cox DH, 2002. Heart Disease Deaths in California, 1999. Data Summary No. DS02-

CPSC, 1987a. Statement of enforcement policy concerning household products containing methylene chloride. *Fed Register* 52: 34698.

CPSC, 1987b. Commercial Practices, Code of Federal Regulations, 16CFR1500.17A2, Washington DC, 1987.

CPSC, 1996. Report on Lead in Vinyl Miniblinds. Available from: http://www.cpsc.gov/LIBRARY/FOIA/FOIA97/OS/BP971.PDF.

CPSC (U.S. Consumer Product Safety Commission), 1997. Estimates of Non-fire Carbon Monoxide Poisoning Deaths and Injuries. Memorandum from Kimberly Ault to Elizabeth Leland, December 17. Epidemiology and Health Sciences. Washington, D.C. http://www.cpsc.gov/library/foia/foia98/brief/3512c1f.pdf.

CPSC, 2003. Non-fire carbon monoxide deaths associated with the use of consumer products: 1999 and 2000 annual estimates. Available from: http://www.cpsc.gov/LIBRARY/co03.pdf.

CPUC (California Public Utilities Commission), 2003. Draft Decision of ALJ Gottstein. Interim Opinion: Phase 4 Standardization Results for Low-Income Energy Efficiency Measure Cost-Effectiveness and Natural Gas Appliance Testing. November 11. San Francisco, CA. Available at: http://www.cpuc.ca.gov/Published/Agenda_decision/31319.htm.

Crump KS, and Farrar DB, 1989. Statistical analysis of data on airborne asbestos levels collected in an EPA survey of public buildings. *Regulatory Toxicology and Pharmacology* **10**: 51-62.

Custovic A, Fletcher A, Pickering CA, Francis HC, Green R, Smith A, Chapman M, Woodcock A, 1998. Domestic allergens in public places. III: house dust mite, cat, dog and cockroach allergens in British hospitals. *Clinical and Experimental Allergy* **28(1)**: 53-59.

Daisey JM, Mahanama KRR, and Hodgson AT, 1998. Toxic volatile organic compounds in simulated environmental tobacco smoke: emission factors for exposure assessment. *Journal of Exposure Analysis and Environmental Epidemiology* **8**: 313-334.

Dales RE, Zwanenburg H, Burnett R, and Franklin CA, 1991a. Respiratory health effects of home dampness and molds among Canadian children. *American Journal of Epidemiology* **134(2)**: 196-203.

Dales RE, Burnett R, and Zwaneburg H, 1991b. Adverse health effects among adults exposed to home dampness and molds. *American Review of Respiratory Disease* **143(3)**: 505-509.

Darnerud PO, Eriksen GS, Hohannesson T, Larsen PB, and Viluksela M, 2001. Polybrominated Diphenyl Ethers: Occurrence, Dietary Exposure and Toxicology. *Environmental Health Perspectives* **109(s1)**: ___.

Day JH, Ellis AK, 2001. Allergenic microorganisms and hypersensitivity. In: Microorganisms in Home and Indoor Work Environments. Editors: B Flannigan, RA Samson, JD Miller. Taylor & Francis. New York, NY.

DCSD (California Department of Community Services Development), 2003. California Low-Income Home Energy Assistance Program (CAL LIHEAP). Sacramento, CA. Available from: http://www.csd.ca.gov/calliheap.html.

Dejmek J, Selevan SG, Benes I, Solansky I, and Sram RJ, 1999. Fetal growth and maternal exposure to particulate matter during pregnancy. *Environmental Health Perspectives* **107(6)**: 475-480.

Delfino RJ, Coate BD, Zeiger RS, Seltzer JM, Street DH, and Koutrakis P, 1996. Daily asthma severity in relation to personal ozone exposure and outdoor fungal spores. *American Journal of Respiratory and Critical Care Medicine* **154**: 633-641.

Delfino RJ, Murphy-Moulton AM, Burnett RT, Brook JR, and Becklake MR, 1997. Effects of air pollution on emergency room visits for respiratory illnesses in Montreal, Quebec. *American Journal of Respiratory and Critical Care Medicine* **155**: 568-576.

Delfino RJ, Murphy-Moulton AM, and Becklake MR, 1998. Emergency room visits for respiratory illnesses among the elderly in Montreal: association with low level ozone exposure. *Environmental Research* **76**: 67-77.

Delfino RJ, 2002. Epidemiological evidence for asthma and exposure to air toxics: linkages between occupational, indoor, and community air pollution research. *Asthma Occurrence* **110 (s4)**: 573-589.

Delfino RJ, Gong H, Jr., Linn WS, Pellizzari ED, and Hu Y, 2003. Asthma symptoms in Hispanic children and daily ambient exposures to toxic and criteria air pollutants. *Environmental Health Perspectives* **111**: 647-656.

Delfino RJ, Gong H, Linn WS, Hu Y, and Pellizzari ED, 2003b. Respiratory symptoms and peak expiratory flow in children with asthma in relation to volatile organic compounds in exhaled breath and ambient air. *Journal of Exposure Analysis and Environmental Epidemiology* **13(5)**: 348-363.

Delfino RH, Quintana PJE, Flora J, Gastanaga VM, Samimi BS, Kleinman MT, Liu S, Bualino C, Wu CF, McLaren, CE., 2004. Association of FEV_1 in asthmatic children with personal and microenvironmental exposure to airborne particulate matter. *Environmental Health Perspectives* **112(8)**: 932-941.

Dennekamp M, Howarth S, Dick CAJ, Cherrie JW, Donaldson K, and Seaton A, 2001. Ultrafine particles and nitrogen oxides generated by gas and electric cooking. *Occupational and Environmental Medicine* **58**: 511-516.

Devlin RB, Horstman DP, Gerrity TR, Becker S, and Madden MC, 1999. Inflammatory response in humans exposed to 2.0 ppm nitrogen dioxide. *Inhalation Toxicology* **11**: 89-109.

Devlin RB, McDonnell WF, Mann R, Becker S, House DE, Schreinemachers D, and Koren HS, 1991. Exposure of humans to ambient levels of ozone for 6.6 hours causes cellular and biochemical changes in the lung. *American Journal of Respiratory Cell and Molecular Biology* **4**: 72-81.

Devlin RB, McDonnell WF, Mann R, Becker S, Madden MC, McGee MP, Perez R, Hatch G, House DE, and Koren HS, 1996. Time-dependent changes of inflammation mediators in the lungs of humans exposed to 0.4 ppm ozone for 2 hr: A comparison of mediators found I bronchoalveolar lavage fluid 1 and 18 hr after exposure. *Toxicology and Applied Pharmacology* **138**: 176-185.

Devos M, Patte F, Rouault J *et al.*, 1990. Standardized Human Olfactory Thresholds. New York, Oxford University Press.

DHS (California Department of Health Services), 1995. A "Do -it-yourself" Inspection of a Ventilation System. Air and Industrial Hygiene Laboratory, Berkeley CA.

DHS, 1996. Reducing Occupant Exposure to Volatile Organic compounds from office building construction materals: Non-binding guidelines. Available from: www.cal-iaq.org/VOC/.

DHS, 1997. State Warns Against Ozone Air Cleaners. News releases (April 1997) available at http://www.dhs.cahwnet.gov/opahome/prssrels/pressrel.htm

DHS, 1998. Lead Hazards in California's Public Elementary Schools and Child Care Facilities: Report to the California State Legislature. Available from: http://www.dhs.cahwnet.gov/childlead/schools/opening.htm.

DHS, 2000. IAQ Info Sheet: Asbestos in the Home and Workplace. Available at: http://www.cal-iaq.org/asb00-03.htm.

DHS, 2002a. California Tobacco Control Update. Tobacco Control Section, Sacramento, CA. Available at: http://www.dhs.ca.gov/tobacco/documents/TCSupdate.PDF.

DHS, 2002b. Indoor and Outdoor Secondhand Smoke Exposure. Tobacco Control Section. Sacramento, CA. http://www.dhs.ca.gov/ps/cdic/ccb/tcs/documents/secondhandsmoke.pdf, http://www.dhs.ca.gov/tobacco.

DHS, 2003. Occupational illness among flight attendants due to aircraft disinsection. Full report by Occupational Health Branch, October 23, 2003. www.afanet.org/afa/aefiles/disinsection.pdf.

DHS, 2004. California Asthma Facts, August 2004: Work-Related Asthma. Prepared by the Environmental Health Investigations Branch, available at www.californiabreathing.org.

DHS, 2004b. California Asthma Facts, Volume 2, Issue 2. California Dept. of Health Services, Environmental Health Investigations Branch. http://www.ehib.org/cma/papers/California_Asthma_Facts_WRA.pdf.

DHS, 2004c. Cleaning Products Can Trigger Work-Related Asthma. *Occupational Health Watch*, Summer 2004. California Dept. of Health Services, Occupational Health Branch. http://www.dhs.ca.gov/ohb/ohw3.pdf.

Diamond M, 2001. IAQ and the Law. *In*: Spengler JD, Samet JM, and McCarthy JF, eds. *Indoor Air Quality Handbook*. San Francisco, CA: McGraw-Hill, pp.____.

DIR, 1997. Interim Tuberculosis Control Enforcement Guidelines. Policy & Procedures Manual, P&P C-47. California Dept. of Industrial Relations, Division of Occupational Safety and Health. http://www.dir.ca.gov/DOSHPol/P&PC-47.HTM

Dockery DW, Pope C, Xu X, Spengler JD, Ware JH, Fay M, Ferris BG, and Speizer FE, 1993. An association between air pollution and mortality in six U.S. cities. *The New England J Medicine* **329(24)**: 25-74.

Dockery DW, and Pope C, 1994. Acute respiratory effects of particulate air pollution. *Annual Review of Public Health* **15**: 107-132.

DOF, 2002a. E-4 Revised Historical City, County and State Population Estimates, 1991-2000, with 1990 and 2000 Census Counts. Available from: http://www.dof.ca.gov/HTML/DEMOGRAP/E-4text2.htm.

DOF, 2002b. Census 2000 California Profile. Available from: http://www.dof.ca.gov/HTML/DEMOGRAP/Census_2000_CA_Profile.htm.

Dominici F, Samet JM, and Zeger SL, 2003. Combining evidence on air pollution and daily mortality from the 20 largest U.S. cities: a hierarchical modeling strategy. *J Royal Statistical Society* **163(3)**: 263-302.

DSA, 2003. The DSA Project Submittal and Plan Review Process. Sacramento, CA: Department of General Services. Available from: http://www.dsa.dgs.ca.gov/ProjectSubmittalProcess/default.htm.

DSA, 2004. EPP Database Project. Sacramento, CA. http://www.eppbuildingproducts.org/.

Dubowski SD, Wallace LA, and Buckley TJ, 1999. The contribution of traffic to indoor concentrations of polycyclic aromatic hydrocarbons. *J Exposure Analysis and Environmental Epidemiology* **9**: 312-321.

Dufresne A, Rivard CI, Malo JL, and Gautrin D, 2002. Exposure to formaldehyde among animal health students. *American Industrial Hygiene Association Journal* **63**: 647-650.

Duhme H, Weiland SK, and Keil U, 1998. Epidemiological analyses of the relationship between environmental pollution and asthma. *Toxicology Letters* **102-103**: 307-316.

EEBA, 2003. EEBA Criteria. Bloomington, MN. Available from: http://www.eeba.org/technology/criteria.htm.

Eggleston PA, 2000. Environmental causes of asthma in inner city children; the National Cooperative Inner City Asthma Study. *Clinical Reviews in Allergy & Immunology* **18(3)**:311-324.

Eichholz GG, 1987. Environmental Radon. *Environmental Science Research Series* **35**: 157-172.

Eriksson P, Jokobsson E, and Fredriksson A, 1998. Developmental neurotoxicity of brominated flame-retardants polybrominated diphenyl ethers and tetrabromo-bisphenol A. *Organohalogen Compounds* **35**: 375-377.

Eriksson P, Viberg H, Jakobsson E, Orn U, and Fredriksson A, 1999. PBDE, 2,2,4,4,5-pentabromodiphenyl ether, causes permanent neurotixic effects during defined period of neurotoxic effects. *Organohalogen Compounds* **40**: 330-335.

ESB (Energy Source Builder), 1995. Build Tight, Ventilate Right. April 1995, # 37. http://oikos.com/esb/index.html#37.

Eskenaze B, Gold EB, Lasley BL, Samuels S, Hammond SK, Wight S, Rasor MON, Hines CJ, and Schenker MB, 1995. Prospective monitoring of early fetal loss and clinical spontaneous abortion among female semiconductor workers. *Amer J Industrial Medicine* **28**:833-846.

Eskenaze B, Gold EB, Samuels SJ, Wight S, Lasley BL, Hammond SK, Rasor MO, and Schenker MB, 1995. Prospective assessment of fecundability of female semiconductor workers. *American Journal of Industrial Medicine* **28**: 817-831.

Eskenaze B, Bradman A, and Castorina R, 1999. Exposures to children to organophosphate pesticides and their potential adverse health effects. *Environmental Health Perspectives*, **107(S3)**: 409-419. Available from: http://ehp.niehs.nih.gov/members/1999/suppl-3/409-419eskenazi/eskenazi-full.html.

Evans GF, Highsmith RV, Sheldon LS, Suggs JC, Williams RW, Zweidinger RB, Creason JP, Walsh D, Rodes CE, and Lawless PA, 2000. The 1999 Fresno particulate matter exposure studies: comparison of community, outdoor, and residential PM mass measurements. *Journal of Air and Waste Management Association* **50**: 1887-1896.

Evans GW, Kantrowitz E, 2002. Socioeconomic status and health: the potential role of environmental risk exposure. *Annual Review of Public Health* **23**:303-331.

Facilities Net, 2005. Risks, Lawsuits Linked to Mold Are Growing. http://www.facilitiesnet.com/news/Jan05news2.shtml.

Fairley D, 2003. Mortality and air pollution for Santa Clara County, California, 1989-1996 in Health Effects Institute Special Report: Revised Analyses of Time-Series Studies of Air Pollution and Health.

Fan Z, Lioy P, Weschler C, Fiedler N, Kipen H, and Zhang J, 2003. Ozone-initiated reactions with mixtures of volatile organic compounds under simulated indoor conditions. *Environmental Science and Technology* **37**: 1811-1827.

Febo A, and Perrino C, 1991. Prediction and experimental evidence for high air concentration of nitrous acid in indoor environments. *Atmospheric Environment* **25A (5/6)**: 1055-1061.

Fisk WJ, 2000. Health and productivity gains from better indoor environments and their relationship with building energy efficiency. *Ann Rev. Energy Environ* **25**: 537-566.

Fisk, WJ, Faulkner D, Palone J, and Seppanen O (2002). Performance and cost of particle air filtration technologies. Indoor Air 12(4):223-234. LBNL-47833. http://eetd.lbl.gov/IED/viaq/pubs/LBNL-47833.pdf.

Flynn E, Matz P, Woolf A and Wright R, 2000. Indoor Air Pollutants Affecting Child Health. Alan Woolf, editor. A project of the American College of Medical Toxicology funded by the U.S. Agency for Toxic Substances and Disease Registry.

Foarde K., van Osdell D, and Steiber R.1997. Investigation of Gas-Phase Ozone as a Potential Biocide. *Applied Occupational Environmental Hygiene*. **12(8):** 535-542.

Folinsbee LJ, Horvath SM, Raven PB, Bedi JF, Morton AR, Drinkwater BL, Bolduan NW, and Gliner JA, 1977. Influence of exercise and heat stress pulmonary function during ozone exposure. *J Applied Physiology* **43(3)**: 409-413.

Folinsbee LJ, Silverman F, and Shephard RJ, 1977. Decrease of maximum work performance following ozone exposure. *J Applied Physiology* **42(4)**: 531-536.

Fortmann, R, Kariher, P, and Clayton, R, 2001. Indoor Air Quality: Residential Cooking Exposures. Final Report. ARCADIS Geraghty & Miller, Inc., Research Triangle Park, NC. Prepared for ARB, Research Division, Sacramento, CA. Contract Number 97-330. http://www.arb.ca.gov/research/indoor/cooking/cooking.htm.

Foster WM, 1999. Deposition and clearance of inhaled particles. In: Holgate ST, Samet JM, Koren HS, Maynard RL, eds. Air pollution and health. San Diego: Academic Press, 295-324.

Friedman H., Potter, A, Haasl T., Claridge D, and Cho S, 2003. Guide: Strategies for Improving the Persistence of Building Performance. Portland Energy Conservation, Inc., and Texas A&M University Prepared for the California Commissioning Collaborative (CCC), 2004. Mission Statement. Sacramento, CA. http://es57055.easystreet.com/news/NewsBriefDetails.aspx?id=32.

Frischer T, Studnicka M, Gartner C, Tauber E, Horak F, Veiter A, Spengler J, Kuhr J, and Urbanek R, 1999. Lung function growth and ambient ozone. American Journal of Respiratory and Critical Care Medicine **160**: 390-396.

Gates GA, 1996. Cost –effectiveness considerations in otitis media treatment. *Otolaryngol Head Neck Surg* **14**: 525-30.

Gauderman WJ, McConnell R, Gilliland F, London S, Thomas D, Avol E, Vora H, Berhane K, Rappaport EB, Lurmann F, and Margolis HG, 2000. Association between air pollution and lung function growth in Southern California children. *Am J Respir Crit Care Med* **162**: 1383-1390.

Geyh AS, Xue J, Ozkaynak H, and Spengler JD, 2000. The Harvard Southern California Chronic Ozone Exposure Study: assessing ozone exposure of grade-school-age children in two southern California communities. *Environ Health Perspect* **108(3)**: 265-70.

Gilliland FD, Berhane K, Rappaport EB, Thomas DC, Avol E, Gauderman WJ, London SJ, Margolis HG, McConnell R, Islam KT, Peters JM. 2001. The effects of ambient air pollution on school absenteeism due to respiratory illnesses. Epidemiology 12:43-54.

Gilliland FD, Berhane K, Rappaport EB, Thomas DC, Avol E, Gauderman WJ, London SJ, Margolis HG, McConnell R, Islam KT, and Peters JM, 2001. The effects of ambient air pollution on school absenteeism due to respiratory illnesses. *Epidemiology* **12(1)**: 43-54.

Gilpin E, Emery S, Farkas A, Distefan J, White M, and Pierce J, 2001. Cancer prevention and control program, University of California, San Diego. The California Tobacco Control Program; a decade of progress, results from the California tobacco survey, 1990-1999. California Dept. Health Services.

Girman JR, Chang Y-L., Hayward SB, and Liu K-S, 1998. Causes of Unintentional Deaths from Carbon Monoxide Poisonings in California. *West J Med* **168(3)**: 158-165.

Girman JR, Hadwen GE, Burton LE, Womble SE, and McCarthy JF, 1999. Individual volatile organic compound prevalence and concentrations in 56 buildings of the building assessment survey and evaluation (BASE) study. *Indoor Air* **2**: 460-465.

Glasgow RE, Foster LS, Lee ME, Hammond SK, Lichtenstein E, and Andrews JA, 1998. Developing a brief measure of smoking in the home: description and preliminary evaluation. *Addict Behav* **23**: 567-571.

Gold, 2005. Personal communication, Deborah Gold, Cal/OSHA, to Peggy Jenkins, 2005.

Goldberg MS, and Burnett RT, 2003. Revised analysis of the Montreal time-series study in Health Effects Institute Special Report: Revised Analyses of Time-Series Studies of Air Pollution and Health.

Goldmann DA, 2001. Epidemiology and prevention of pediatric viral respiratory infections in health-care institutions. *Emerging Infectious Diseases.* **April 7(2):** 249-253.

Gordon SM, Callahan PJ, Nishioka MG, Brinkman MC, O'Rourke MK, Lebowitz MD, and Moschandreas DJ, 1999. Residential environmental measurements in the National Human Exposure Assessment Survey (NHEXAS) pilot study in Arizona: preliminary results for pesticides and VOCs. *J Expo Anal Environ Epidemiol* **9**: 456-470.

Graves CG, Ginevan ME, Jenkins RA, and Tardiff RG, 2000. Doses and lung burdens of environmental tobacco smoke constituents in nonsmoking workplaces. *J Expo Anal Environ Epidemiol* **10**: 365-377.

Grot RA, 1995. Air Change Rates in Non-residential Buildings in California. Lagus Applied Technology, Escondido, CA. Prepared for the California Energy Commission, Report P400-91-034BCN. Sacramento, CA.

Guerin MR, Jenkins RA, and Tomkins BA, 1992. The chemistry of environmental tobacco smoke: Composition and measurement. Lewis Publishers, Boca Raton.

Guo Z, Chang JCS, Sparks LE, and Fortman RC, 1999. *Atmos Environ* **33**: 1205-1215.

Guo Z, Mosley R, McBrian J, and Fortmann R, 2000. Fine particulate matter emissions from candles. *Engineering Solutions to Indoor Air Quality Problems* **VIP-98**: 211-225.

Gurunathan S, Robson M, Freeman N, Buckley B, Roy A, Meyer R, Bukowski J, and Lioy PJ, 1998. Accumulation of chlorpyrifos on residential surfaces and toys accessible to children. *Environ Health Perspect* **106**: 9-16.

Gustavsson H, Lundgren B, 1997. Off-gassing from building materials: a survey of case studies. In: Brune D, Gerhardsson G, Crockford GW, Dáuria D, editors: The Workplace, volume 1. Fundamentals of health, safety and welfare. International Labor Office, Geneva, 533-555.

Hale RC, La Guardia MJ, Harvey EP, Mainor TM, Duff WH, and Gaylor MO, 2001. Polybrominated Diphenyl Ether Flame Retardants in Virginia Freshwater Fishes (USA). *Environ Sci Technol* **35(23)**: 4585-4591.

Hale RC, La Guardia MJ, Harvey E, and Mainor TM, 2002. Potential role of fire retardant-treated polyurethane form as a source of brominated diphenyl ethers to the US environment. *Chemosphere* **46**: 729-735.

Hammond SK, Hines CJ, Hallock MF, Woskie SR, Kenyon EM, and Schenker MB, 1996. Exposures to glycol ethers in the semiconductor industry. *Occupational Hygiene* **(2)**: 355-366.

Hammond SK, 1999. Exposure of U.S. workers to environmental tobacco smoke. *Environ Health Perspect* **107(Supplement 2)**: 329-340.

Hasselblad V, Eddy DM, and Kotchmar D, 1992. Synthesis of environmental evidence: nitrogen dioxide epidemiology studies. *J. Air Waste Manage. Assoc.* **42**: 622-671.

Hayden S, 2004. Natural Resources Canada, CANMET Advanced Combustion Technologies Group. . Ottawa, Ontario, Canada. Personal communication.

Healthy Schools Campaign, 2003. Apparently Size Doesn't Matter: Two Illinois School Districts Show Successful IAQ Management. *School Health Watch:The Newsletter of the Illinois Healthy Schools Campaign*, 2(2). Available from: http://www.healthyschoolscampaign.org/school%20health%20watch_summer-2003.pdf.

Hedge A, 1995. In defense of "the sick building syndrome." *Indoor Environment* **4**: 251-253.

HEI, 2003. Revised analyses of time-series studies of air pollution and health. HEI Special report.

Henderson KA, Matthews IP, Adisesh A and Hutchings AD, 2003. Occupational exposure of midwives to nitrous oxide on delivery suites. *Occup Environ Med* **60**: 958-961.

Heschong, 2003. Windows and Classrooms: A Study of Student Performance and the Indoor Environment. (P500-03-082-A-7). California Energy Commission. Available from: http://h-m-g.com/Daylighting/A-7_Windows_Classrooms_2.4.10.pdf.

HESIS, 2004. Site investigation of student health complaints at Federico's beauty College. Bakersfield, California. letter dated December 22, 2004.

Hiipakka D, and Samimi B, 1987. Exposure of acrylic fingernail sculptors to organic vapors and methacrylate dusts. *Am. Ind. Hyg. Assoc. J.* **48(3)**: 230-237.

Hoddinott KB, and Lee AP, 2000. The use of environmental risk assessment methodologies for an indoor air quality investigation. *Chemosphere* **41**: 77-84.

Hodgson AT, 1999. Common Indoor Sources of Volatile Organic Compounds: Emission Rates and Techniques for Reducing Consumer Exposures. Final report under contract no. 95-302. Sacramento, CA, California Environmental Protection Agency, Air Resources Board.

Hodgson, M, Levin, H, and Wolkoff,P, 1994. Volatile organic compounds in indoor air. *Journal of Allergy and Clinical Immunology* **94**, 296-303.

Hodgson AT, Rudd AF, Beal D, and Chandra S, 2000. Volatile organic compound concentrations and emission rates in new manufactured and site-built houses. *Indoor Air* **10**: 178-192.

Hodgson AT, Beal D, and McIlvaine JER, 2002. Sources of formaldehyde, other aldehydes and terpenes in a new manufactured house. *Indoor Air* **12**: 235-242.

Hodgson AT and Levin H, 2003a. Classification of measured indoor volatile organic compounds based on noncancer health and comfort considerations. Lawrence Berkeley National Laboratory Report – 53308, September 2003.

Hodgson AT and Levin H, 2003b. Volatile organic compounds in indoor air: a review of concentrations measured in North America since 1990. Lawrence Berkeley National Laboratory Report – 51715, April 21, 2003.

Hodgson M, 1995. The sick-building syndrome. *Occup Med* **10(1)**: 167-75. Review.

Horak F, Studnicka M, Gartner C, Spengler JD, Tauber E, Urbanek R, Veiter A, and Frischer T, 2002. Particulate matter and lung function growth in children: a 3-yr follow-up study in Austrian schoolchildren. *Eur Respir J* **19**: 838-845.

Hosler DL, 1998. Personal communication, Southern California Gas Company, Los Angeles, CA. As cited in Traynor (1999).

HUD, 1996. Environmental Assessment Guide For Public Housing. Washington, DC. Prepared by Steven Winter Associates, Inc., Norwalk, CT. October 30. http://www.huduser.org/publications/pubasst/enviro.html.

HUD, 1999. The Healthy Homes Initiative: A Preliminary Plan (Full Report), Office of Lead Hazard Control, Washington, DC. April. http://www.hud.gov/offices/lead/reports/HHIFull.pdf.

HUD, 2002. Manufactured Home Construction and Safety Standards, 24 CFR 3280. http://www.hud.gov/offices/hsg/sfh/mhs/mhshome.cfm.

Husman H, 1996. Health effects of indoor-air microorganisms. *Scandinavian Journal of Work, Environment and Health* **22**:5-13.

IARC (International Agency for Research on Cancer), 1983. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: Certain Polycyclic Aromatic Hydrocarbons and Heterocyclic Compounds, Volume 32. Available from: http://193.51.164.11/htdocs/monographs/vol32/benzo%5Ba%5Dpyrene.html.

IARC (International Agency for Research on Cancer), 2002. IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans: tobacco smoke and involuntary smoking **83**.

IOM (Institute of Medicine), 1993. *Indoor Allergens: Assessing and Controlling Adverse Health Effects*. Washington D.C: National Academy Press. http://books.nap.edu/books/0309048311/html/

IOM (Institute of Medicine), 1999. *Toward environmental justice research, education, and health policy needs.* Washington D.C: National Academy Press. http://books.nap.edu/books/0309064074/html/.

IOM (Institute of Medicine), 2000. Clearing the air: asthma and indoor air exposures. National Academy of Sciences, Washington DC: National Academy Press. http://www.nap.edu/books/0309064961/html/

IOM (Institute of Medicine), 2004. Damp indoor spaces and health. National Academy of Sciences. Washington D.C: National Academy Press. http://www.nap.edu/books/0309091934/html/.

Ito K, 2003. Associations of particulate matter components with daily mortality and morbidity in Detroit, Michigan in Health Effects Institute Special Report: Revised Analyses of Time-Series Studies of Air Pollution and Health.

Jarvis D, Chinn S. Luczynska C, and Burney P, 1996. Association of respiratory symptoms and lung function in young adults with use of domestic gas appliances. *Lancet* **347:** 426-431.

Jefferis G, 2004. Maintenance and Operations, Everett Unified School District, Everett, WA. Personal communication, January 7. See also: http://www.enterprisenewspapers.com/archive/2003/8/8/2003861629365.cfm.

Jenkins PL, Phillips TJ, Mulberg EJ, and Hui SP, 1992a. Activity patterns of Californians: use of and proximity to indoor pollutant sources. *Atmos Environ* **26A(12)**: 2141-2148.

Jenkins PL, Hui SP, Phillips TJ, and Lum SB, 1992b. Toxic Air Pollutants in California Residences. Presentation. *Current Issues in Air Toxics*, Proceedings of the Third Annual West Coast Regional Air and Waste Management Association Conference, November 1992, Sacramento, CA.

Jetter JJ, Guo Z, McBrian JA, and Flynn MR, 2002. Characterization of emissions from burning incense. *Sci Total Environ* **295(1-3)**: 51-67.

Jo WK, and Kim SH, 2001. Worker exposure to aromatic volatile organic compounds in dry cleaning stores. *American Industrial Hygiene Association Journal* **62, 4**: 466-471.

Johanning E, Biagini R, Hull D, Morey P, Jarvis B, Landsbergis P, 1996. Health and immunology study following exposure to toxigenic fungi (*Stachybotrys chartarum*) in a water-damaged office environment. *In Arch Occup Environ Health* **68**:207-218.

Kamens R, Lee C, Wiener R, and Leith D, 1991. A study to characterize indoor particles in three non-smoking homes. *Atmos Environ* **25(5/6)**: 939-948.

Kanarek M, Quackenboss JJ, Kaarakka P, Duffy C, and Rohrer KM, 1985. Energy conservation through weatherization and indoor air quality. Final Report May 16, 1985. Performed by the University of Wisconsin for Wisconsin Power and Light Co.

Kaplan J, Marquardt S, and Barber W, 1998. Failing Health: Pesticide Use in California Schools. San Francisco, CA: CALPIRG Charitable Trust.

Karol MH, 2002. Respiratory allergy: what are the uncertainties? *Toxicology* **181-182**: 305-310.

Kats G, 2003. The Costs and Financial Benefits of Green Buildings. A Report to California's Sustainable Building Task Force. P. 66. http://WWW.CIWMB.CA.GOV/GreenBuilding/Design/CostBenefit/Report.pdf.

Keeler GJ, Dvonch T, Yip FY, Parker EA, Isreal BA, Marsik FJ, Morishita M, Barres JA, Robins TG, Brakefield-Caldwell W, and Sam M, 2002. Assessment of personal and community-level exposures to particulate matter among children with asthma in Detroit, Michigan, as part of Community Action Against Asthma (CAAA). *Environ Health Perspect* **110 Suppl 2**:173-81.

Kelly TJ, Smith DL, and Satola J, 1999. Emission rates of formaldehyde from materials and consumer products found in California homes. *Environ Sci Technol* **33**: 81-88.

Kelly TJ, Battelle, May 2000, Indoor Air Quality Symposium: Risk Reduction in the 21st Century, Sacramento, CA.

Kerger BD, Schmidt CE, and Paustenbach DJ, 2000. Assessment of airborne exposure to trihalomethanes from tap water in residential showers and baths. *Risk Anal* **20(5)**: 637-651.

Kirkpatrick JN, 1987. Occult carbon monoxide poisoning. *West J Med* **146(1)**: 52-56.

Kissel JC, 1993. Potential impact of deliberately introduced ozone on indoor air quality. *J Expo Anal Environ Epidemiol* **3(2)**: 155-64.

Kleno JG, Clausen PA, Weschler CJ, and Wolkoff P, 2001. Determination of ozone removal rates by selected building products using the FLEC emission cell. *Environ Sci Technol* **35**: 2548-2553.

Krewski, D.; Burnett, R.; Goldberg, M.; Hoover, K.; Siemiatycki, J.; Jerrett, M.; Abrahamovicz, M.; White, W. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality; Health Effects Institute: Cambridge, MA, 2000

Krieger J, and Higgins DL, 2002. Housing and health: time again for public health action. *Am J Public Health* **92(5)**: 758-768.

Krieger JW, Takaro TK, Song L, and Weaver M, 2005. The Seattle-King County Healthy Homes Project: A Randomized, Controlled Trial of a Community Health Worker Intervention to Decrease Exposure to Indoor Asthma Triggers. In Press, American Journal of Public Health.

Kulle TJ, Sauder LR, Hebel JR, and Chatham MD, 1985. Ozone response relationships in healthy nonsmokers. *Am Rev Respir Dis* **132**: 36-41.

Labreche F, Forest J, Trottier M, Lalonde M and Simard R, 2003. Characterization of chemical exposures in hair dressing salons. *Applied Occupational and Environmental Hygiene* **18:** 1014-1021.

Lai ACK, Thatcher TL, and Nazaroff WW, 2000. Inhalation transfer factors for air pollution health risk assessment. *Journal of the Air and Waste Management Association* **50**: 1688-1699.

Landrigan PJ, Claudio L, Markowitz SB, Berkowitz GS, Brenner BL, Romero H, Wetmur JG, Matte TD, Gore AC, Godbold JH, and Wolff MS, 1999. Pesticides and Inner-City Children: Exposures, Risks, and Prevention. *Environ Health Perspect* **107(S3)**: 431-437. Available from: http://ehp.niehs.nih.gov/members/1999/suppl-3/431-437landrigan/landrigan-full.html.

Laslo-Baker D, Barrera M, Knittel-Keren D, Kozer E, Wolpin J, Khattak S, Hackman R, Rovet J, and Koren G, 2004. Child neurodevelopmental outcome and maternal occupational exposure to solvents. *Arch Pediatr Adolesc Med* **158**: 956-961.

LBNL, 2003. Residential Commissioning Project. Indoor Environment Department, Environmental Energy Technology Division. Berkeley, CA. http://epb1.lbl.gov/EPB/commissioning/index.html.

LBNL (Lawrence Berkeley National Laboratory), 2004. Sick Building Syndrome. Indoor Environmental Program. Berkeley, CA. http://eetd.lbl.gov/IED/viaq/v_syndrome_1.html.

Lee K, Xue J, Geyh AS, Ozkaynak H, Leaderer BP, Weschler CJ, and Spengler JD, 2002. Nitrous acid, nitrogen dioxide, and ozone concentrations in residential environments. *Environ Health Perspect* **110(2)**: 145-50.

Lee RJ, Van Orden DR, Corn M, and Crump KS, 1992. Exposure to airborne asbestos in buildings. *Regul Toxicol Pharmacol* **16**: 93-107.

Lee SC, Lam S, and Fai HK, 2001. Characterization of VOCs, ozone, and PM10 emissions from office equipment in an environmental chamber. *Building and Environment* **36**: 837-842.

Leech JA, Raizenne M, and Gusdorf J, 2004. Health in occupants of energy efficient new homes. *Indoor Air* **3**: 169-173.

Leikauf GD, 2002. Hazardous air pollutants and asthma. *Environ Health Perspect* **110(Supplement 4)**: 505-526.

Leino T, Kahkonen E, Saarinen L, Eckerman MLH, and Paakkulainen H, 1999. Working conditions and health in hairdressing salons. *Applied Occupational and Environmental Hygiene* **14:** 26-33.

Leovic, KW, Sheldon, LS, Whitaker, DA, Hetes, RG, Calcagni, JA, and Baskir, JN, 1996. Measurement of indoor air emissions from dry-process photocopy machines. *Journal of Air & Waste Management Association* **46**: 821-829.

Levesque B, Allaire S, Prud'homme H, Rhainds M, Lebel G, Bellemarre D, and Dupuis K, 1997. Indoor motocross competitions: air quality evaluation. *American Industrial Hygiene Association Journal* **58:** 286-290.

Levesque B, Allaire S, Prud'homme H, Dupuis K and Bellemare D, 2000. Air quality monitoring during indoor monster truck and car demolition shows. *Journal of Exposure Analysis and Environmental Epidemiology* **10**: 58-65.

Levy JI et al., 1998. Impact of residential nitrogen dioxide exposure on personal exposure: an international study. J. *Air & Waste Manage. Assoc.* **48:** 553-560.

Lewis R, Fortmann R, and Camann D, 1994. Evaluation of methods for monitoring the potential exposure of small children to pesticides in the residential environment. *Arch Environ Contam Toxicol* **26**: 37-46.

Lewis R, Fortune C, Willis R, Camann D, and Antley J, 1999. Distribution of pesticides and polycyclic aromatic hydrocarbons in house dust as a function of particle size. *Environ Health Perspect* **107**: 721-726.

Liddament MW, 2000. A Review of Ventilation and the Quality of Ventilation Air. *Indoor Air* **10**: 193-199.

Lilienfeld AM, and Lilienfeld DE, 1980. Foundations of Epidemiology (2nd Edition). New York: Oxford University Press.

Lillquist DR, Lee JS, Ramsay JR, Boucher KM, Walton ZL, and Lyon JI, 1998. A comparison of indoor/outdoor PM10 concentrations measured at three hospitals and a centrally located monitor in Utah. *Appl Occup Environ Hyg* **13(6)**: 409-415.

Linn WS, Gong, Jr H, Clark KW, and Anderson KR, 1999. Day-to-day particulate exposure and health changes in Los Angeles area residents with severe lung disease. *J Air Waste Manag Assoc* **49**: 108-115.

Linn WS, Szlachcic Y, Gong H, Kinney PL, and Berhane KT, 2000. Air pollution and daily hospital admissions in metropolitan Los Angeles. *Environ Health Perspect* **108**: 427-434.

Lioy PJ, and Greenberg A, 1990. Factors associated with human exposures to polycyclic aromatic hydrocarbons. *Toxicol Ind Health* **6(2)**: 209-223.

Lippmann M *et al.*, 2000. Association of particulate matter components with daily mortality and morbidity in urban populations. *Research Report / Health Effects Institute* **56(95)**: p. 5-72, discussion 73-82.

Lipsett M, Hurley S, and Ostro B, 1997. Air pollution and emergency room visits for asthma in Santa Clara County, California. *Environ Health Perspect* **105(2)**: 216-222.

Liu DL, and Nazaroff WW, 2001. Modeling pollutant penetration across building envelopes. *Atmospheric Environment* **35:** 4451-4462.

Liu KS, Hayward SB, Girman JR, Moed BA, and Huang FY, 1991. Annual average radon concentrations in California residences. *J Air Waste Manag Assoc* **41(9)**: 1207-1212.

Liu KS, Girman JR, Hayward SB, Shusterman D, and Chang YL, 1993a. Unintentional carbon monoxide deaths in California from charcoal grills and hibachis. *J Expo Anal Environ Epidemiol* **3(Suppl 1)**: 143-51.

Liu LJ, Koutrakis P, Suh HH, Mulik JD, and Burton RM, 1993b. Use of personal measurements for ozone exposure assessment: a pilot study. *Environ Health Perspect* **101**(4): 318-24.

Liu LJ, Koutrakis P, Leech J, and Broder I, 1995. Assessment of ozone exposures in the greater metropolitan Toronto area. *J Air Waste Manag Assoc* **45**(4): 223-34.

Liu KS, Paz MK, Flessel P, Waldman J, and Girman J, 2000. Unintentional carbon monoxide deaths in California from residential and other nonvehicular sources. *Arch Environ Health* **55(6)**: 375-381.

Liu LJS, Box M, Kalman D, Kaufman J, Koenig, Larson T, Lumley T, Sheppard L, and Wallace L, 2003. Exposure assessment of particulate matter for susceptible populations in Seattle. *Environ Health Perspect* **222(7)**: 908-918.

Lofroth G, Stensman C, Brandhorst-Satzkorn M, 1991. Indoor sources of mutagenic aerosol particulate matter: smoking, cooking and incense burning. *Mutat Res* **261(1)**: 21-28.

Long CM, Suh HH, and Koutrakis P, 2000. Characterization of indoor particle sources using continuous mass and size monitors. *J Air Waste Manag Assoc* **50**: 1236-1250.

Long CM, Suh HH, Kobzik L, Catalano PJ, Ning YY, and Koutrakis P, 2001. A pilot investigation of the relative toxicity of indoor and outdoor fine particles: in vitro effects of endotoxin and other particulate properties. *Environ Health Perspect* **109(10)**: 1019-26.

Lubliner M, and Gordon A, 1990. Ventilation in US Manufactured Homes: Requirements, Issues and Recommendations. Presented at the 21st Annual Air Infiltration and Ventilation Centre Conference, The Hague, Netherlands, September 26 –29. Washington State University Energy Extension Program. http://www.fsec.ucf.edu/Bldg/baihp/pubs/wsu-aivc/index.htm.

Lynch RM, and Kipen H, 1998. Building-related illness and employee lost time following application of hot asphalt roof: A call for prevention. *Toxicology and Industrial Health*, **14(6):** 857-868.

Magnani C, Dalmasso P, Biggeri A, Ivaldi C, Mirabelli D, Terracini B, and 2001. Increased risk of malignant mesothelioma of the pleura after residential or domestic exposure to asbestos: a case-control study in Casale Monferrato, Italy. *Environ Health Perspect* **109**:915–919.

Mah JC, 2000. Non-Fire Carbon Monoxide Deaths and Injuries Associated with the Use of Consumer Products: Annual Estimates. U.S. Consumer Product Safety Commission, Directorate for Epidemiology, Division of Hazard Analysis, Bethesda, MD 20814, October 2000. http://www.cpsc.gov/library/co00.pdf.

Maisonet M, Bush TJ, Correa A, and Jaakkola JJK, 2001. Relation between ambient air pollution and low birth weight in the Northeastern United States. *Environl Health Perspect* **109(3)**: 351-356.

Malone N, Baluja KF, Costanzo JM, and Davis CJ, 2003. The Foreign-Born Population: 2000, Census 2000 Brief, US Census Bureau, Washington, DC.

Mannino DM, Homa DM, Pertowski CA, Ashizawa A, Nixon LL, Johnson CA, Ball LB, Elizabeth J, and Kang DS, 1998. Surveillance for asthma: United States, 1960-1995. *MMWR* **47**: 1-28.

Mannino DM, Homa DM, Akinbami LJ, Moorman JE, Gwynn C, and Redd SC, 2002. Surveillance for asthma – United States, 1980-1999. *MMWR* **51(SS01)**: 1-13.

Marr LC, Morrison GC, Nazaroff WW, and Harley RA, 1998. Reducing the risk of accidental death due to vehicle-related carbon monoxide poisoning. *Journal of the Air and Waste Management Association* **48**, 899-906.

Mar TF *et al.*, 2000. Associations between Air Pollution and Mortality in Phoenix, 1995-1997. *Environmental Health Perspectives* **108(4)**: 347-353.

Marcinowski F, Lucas RM, and Yeager WM, 1994. National and regional distributions of airborne radon concentrations in U.S. homes. *Health Phys* **66(6)**: 699-706.

Mason MA, Sparks LE, Moore SA, Dolgov I, and Perry RB, 2000. Characterization of ozone emissions from air cleaners equipped with ozone generators and sensor and feedback control circuitry.

Mathieu-Nolf M, 2002. Poisons in the air: a cause of chronic disease in children. *J Toxicol Clin Toxicol* **40**: 483-489.

Mattison DR, Damus K, Fiore E, Petrini J, Alter C, 2001. Preterm delivery:a public health perspective. Pediatric Perinat Epidemiol. Suppl 2:7-16. http://www.marchofdimes.com/files/Preterm_delivery.pdf.

Mazdai A, Dodder NG, Abernathy MP, and Hites RA, 2003. Polybrominated diphenyl ethers in maternal and fetal blood samples. *Environ Health Perspect* **111**: 1249-1252.

McBride DE, Koenig JQ, Luchtel DL, Williams PV, and Henderson WR, 1994. Inflammatory effects of ozone in the upper airways of subjects with asthma. *Am J Respir Crit Care Med* **149**: 1192-1197.

McConnell R, Berhane K, Gilliland F, London SJ, Vora H, Avol E, Gauderman WJ, Margolis HG, Lurmann F, Thomas DC, and Peters JM, 1999. Air pollution and bronchitic symptoms in Southern California children with asthma. *Environ Health Perspect* **107(9)**:757-60.

McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, Avol E, Margolis SG, and Peters JM, 2002. Asthma in exercising children exposed to ozone: a cohort study. *Lancet* **359**:386-91.

McConnell R, Berhane K, Gilliland F, Molitor J, Thomas D, Lurmann F, Avol E, Gauderman WJ, and Peters JM, 2003. Prospective study of air pollution and bronchitic symptoms in children with asthma. *Am J Respir Crit Care Med* **168**: 790-797.

McDonald JC, Harris J, and Armstrong B, 2004. Mortality in a cohort of vermiculite miners exposed to fibrous amphibole in Libby, Montana. *Occup Environ Med* **61**: 363-366

McDonald TA, 2002. A perspective on the potential health risks of PBDEs. *Chemosphere* **46**: 745-755.

McDonnell WF, Stewart PW, Smith MV, Pan WK, and Pan J, 1999. Ozone-induced respiratory symptoms: exposure-response models and association with lung function. *Eur Respir J* **14**: 845-853.

MDC (Minnesota Department of Commerce), 2004. Minnesota Energy Code, Chapter 7672 Detached Single-Family and Two-Family Dwellings. Effective April 15, 2000. Available at: http://www.state.mn.us/cgi-bin/portal/mn/jsp/content.do?subchannel=-536881494&programid=536894293&sc2=-536882175&id=-536881350&agency=Commerce&sp2=y&sp3=y.

Mendell MJ, 1993. Non-specific symptoms in office workers: a review and summary of the epidemiologic literature. *Indoor Air* **3**: 227-236.

Mendell MJ, Fisk WJ, Deddens JA *et al.* 1996. Elevated symptom prevalence associated with ventilation type in office buildings. *Epidemiology* **7**: 583-589.

Mendell MJ, Fisk WJ, Kreiss K, Levin H, Alexander D, Cain WS, Girman JR, Hines CJ, Jensen PA, Milton DK, Rexroat LP, and Wallingford KM, 2002. Improving the Health of Workers in Indoor Environments: Priority Research Needs for a National Occupational Research Agenda. *Am J Public Health* **92** 1430–1440.

Mendez D, Warner KE, and Courant PN, 1998. Effects of Radon Mitigatoin vs. Smoking Cessation in Reducing Radon-Related Risk of Lung Cancer. *Amer J Public Health* **88(5)**: 811-812.

Meng YY, Babey SH, Malcolm E, Brown ER, and Chawla N, 2003. Asthma in California: findings from the 2001 California health interview survey. November 2003, UCLA Center for Health Policy Research, funded by the California Endowment.

Meng Y, Babey SH, Malcolm E, Brown ER, Chawla N, 2003. Asthma in California: Findings from the 2001 California Health Interview Survey. UCLA Center for Health Policy Research, University of California, Los Angeles, CA. http://www.healthpolicy.ucla.edu/pubs/publication.asp?publD=83.

Menzies D *et al.*, 1994. The "sick building" – a misleading term that should be abandoned. *Indoor Air Quality* 37-48.

Menzies D, and Bourbeau J, 1997. Building-related illnesses. *N Engl J Med* **337**: 1524-1531.

Miguel AG, Cass GR, Glovsky MM, and Weiss J, 1998. Allergens in paved road dust and airborne particles, Final Report to the California Air Resources Board contract No. 95-312, August 1998.

Miller JD, 2001. Mycological investigations of indoor environments. in Microorganisms in Home and Indoor Work Environments. Editors: B Flannigan, RA Samson, JD Miller. Taylor & Francis. New York, NY.

Mills JN, Corneli A, Young JC, Garrison LE, Khan AS, and Ksiazek TG, 2002. Hantavirus pulmonary syndrome--United States: updated recommendations for risk reduction. Centers for Disease Control and Prevention. *MMWR* Recomm Rep. 2002 Jul **26;51(RR-9)**:1-12.

Milton D, Glencross P, and Walters M, 2000. Risk of sick leave associated with outdoor air supply rate, humidification, and occupants' complaints. *Indoor Air* **10**, 212-221.

MOD (March of Dimes Birth Defects Foundation), 2003. PeriStats: Born Too Soon and Too Small in the United States. White Plains, NY. http://www.marchofdimes.com/peristats/prematurity.aspx?reg=99.

Molhave L, 1991. Indoor Climate, Air Pollution, and Human Comfort. *J Expo Anal Environ Epidemiol* **1(1)**: 63-81.

Molhave L., 1991b. Volatile organic compounds, indoor air quality and health. *Indoor Air* **(4)**: 357-376.

Molhave L, 2003. Organic compounds as indicators of air pollution. *Indoor Air* **13(Suppl. 6)**: 12-19.

Moolgavkar SH, 2003. Air pollution and daily deaths and hospital admissions in Los Angeles and Cook Counties in Health Effects Institute Special Report: Revised Analyses of Time-Series Studies of Air Pollution and Health.

Moriske HJ, Ebert G, Konieczny L, Menk G, and Schondube M, 1998. Concentrations and decay rates of ozone in indoor air in dependence on building and surface materials. *Toxicol Lett* **96-97**: 319-323.

Morrison GC, and Nazaroff WW, 2002. Ozone interactions with carpet: secondary emissions of aldehydes. *Environmental Science and Technology* **36**: 2185-2192.

Mortimer KM, Neas LM, Dockery DW, Redline S, and Tager IB, 2002. The effect of air pollution on inner-city children with asthma. *Eur Respir J* **19(4)**: 699-705.

Mott JA, Wolfe MI, Alverson C et al., 2002. National vehicle emissions policies and practices and declining US carbon monoxide-related mortality. *JAMA* **288**, 988-995.

Mukerjee S, Ellenson WD, Lewis RG, Stevens RK, Somerville MC, and Shadwick DS, 1997a. An environmental scoping study in the Lower Rio Grande Valley of Texas – I. Comparative assessement of air sampling methods. *Environ Int* **23(5)**: 611-628.

Mukerjee S, Ellenson WD, Lewis RG, Stevens RK, Somerville MC, Shadwick DS, and Willis RD. 1997b. An environmental scoping study in the Lower Rio Grande Valley of Texas – III. Residential microenvironmental monitoring for air, house dust, and soil. *Environ Int* **23(5)**: 657-673.

Mullins J, 2001. Microorganisms in outdoor air. In: Microorganisms in Home and Indoor Work Environments. Editors: B Flannigan, RA Samson, JD Miller. Taylor & Francis. New York, NY.

Myatt TA, Johnston SL, Zuo Z, Wand M, Kebadze T, Rudnick S, and Milton DK, 2004. Detection of airborne rhinovirus and its relation to outdoor air supply in office environments. *Am J Respir Crit Care Med.* **Jun 1;169(11):**1187-90.

Nagda NL, and Rector HE, 2003. A critical review of reported air concentrations of organic compounds in aircraft cabins. *Indoor Air* **13**: 292-301.

National Academy of Sciences, 1999. Health Effects of Exposure to Radon: BEIR VI. Committee on Health Risks of Exposure to Radon, National Research Council. http://www.epa.gov/radon/beirvi.html.

NAHB (National Association of Home Builders), 2004. Model Green Home Building Guidelines Initiative Launched At the 2004 International Builders' Show. Upper Marlboro, MD. http://www.nahb.org/news_details.aspx?newsID=724, http://www.nahbrc.org/green3.asp?CategoryID=1891.

NRCan (Natural Resources Canada), 2004a. All About Gas Fireplaces. Ottawa, Ontario, Canada. http://oee.nrcan.gc.ca/Publications/infosource/Pub/home/all_about_gas_fireplaces.pdf.

NRCcan, 2004b. Breathe Easier With Healthy Ventilation and Fewer Pollutants. Ottawa, Ontario, Canada. http://oee.nrcan.gc.ca/Publications/infosource/pub/r2000/BreatheFactEng.cfm. October 20.

Naumova YY, Eisenreich SJ, Turpin BJ, Weisel CP, Morandi MT, Colome SD, Totten LA, Stock TH, Winer AM, Alimokhtari S, Kwon J, Shendell D, Jones J, Maberti S, and Wall SJ, 2002. Polycyclic aromatic hydrocarbons in the indoor and outdoor air of three cities in the U.S. *Environ Sci Technol* **36**: 2552-2559.

Nazaroff WW, and Weschler CJ, 2004a. Cleaning products and air fresheners: exposure to primary and secondary air pollutants. *Atmos Environ* **38**: 2841–2865.

Nazaroff WW, and Singer BC, 2004b. Inhalation of hazardous air pollutants from environmental tobacco smoke in US residences. *Journal of Exposure Analysis and Environmental Epidemiology* **14**: S71-S77.

Nazaroff WW, and Klepeis NE, 2004c. Environmental tobacco smoke particles. *Indoor Environment: Airborne Particles and Settled Dust*, Morawska L, and Salthammer T (Eds.), Wiley-VCH, Weinheim, Germany, 2004, pp. 245-274.

NCI (National Cancer Institute), 1999. Health effects of exposure to environmental tobacco smoke: The report of the California Environmental Protection Agency. Smoking and Tobacco Control Monograph no. 10. Bethesda, MD. U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute, NIH Pub. No. 99-4645.

Neas LM, Dockery DW, Ware JH, Spengler JD, Speizer FE, and Ferris BG, Jr., 1991. Association of indoor nitrogen dioxide with respiratory symptoms and pulmonary function in children. *American Journal of Epidemiology* **134 (2):** 204-218.

Needleman HL, Gunnoe C, Leviton A, Reed R, Peresie H, Maher C, and Barrett P, 1979. Deficits in psychologic and classroom performance of children with elevated dentine levels. *N Engl J Med* **100**: 689-695.

New Jersey Mercury Task Force, 2002. *Volume II: Impacts of Mercury in New Jersey*. Available from: http://www.state.nj.us/dep/dsr/mercury_task_force.htm.

Nguyen TTL, Pentikäinen T, Rissanen P, Vahteristo M, Husman T, Nevalainen A, 1998. Health related costs of moisture and mold in dwellings. Publication of the National Public Health Institute, B13, Kuopio University Printing Office, Finland.

NIOSH, 1992. NIOSH Health Hazard Evaluation, Chapple Hair Styling Salon. HETA 89-138-2215. NIOSH HC 28.

NIOSH Pocket Guide to Chemical Hazards, February 2004, printed edition NIOSH Publication Number 97-140, available at http://www.cdc.gov/niosh/npg/npgd0293.html

Noren K, and Meironyte D, 1998. Contaminants in Swedish human milk. Decreasing levels of organochlorine and increasing levels of organobromine compounds. *Organohalogen Compounds* **40**: 1111-1123.

NRC (National Research Council), 1981. Indoor Pollutants. National Academy Press, Washington, D.C.

NRC (National Research Council) 1988. Health Risks of Radon and Other Internally Deposited Alpha-Emitters, Committee on the Biological Effects of Ionizing Radiations (BEIR IV). National Academy Press, Washington, D.C.

NRC (National Research Council), 1990. Health Effects of Exposure to Low Levels of Ionizing Radiation. Committee on the Biological Effects of Ionizing Radiations (BEIR V). National Academy Press, Washington, D.C.

NRC (National Research Council), 1999a. Biological effects of ionizing radiation (BEIR) VI report: The health effects of exposure to indoor radon. Committee on Health Risks of Exposure to Radon. National Academy Press: Washington DC.

NRC (National Research Council), 1999b. Risk assessment of radon in drinking water. Committee on risk assessment of exposure to radon in drinking water. National Academy Press: Washington DC.

NTP (National Toxicology Program), December 2002. Report on Carcinogens, Tenth Edition. Available from: http://ehp.niehs.nih.gov/roc/toc10.html.
NYDOH 2003. Fact Sheet: Tetrachloroethylene (PERC) in Indoor Air and Outdoor Air. New York State Department of Health. May 2003.

NYS (New York State), 1999. Assisting Communities to Improve Public Health. (Annual Report, 1999-2000). Albany, NY: Department of Health. Available from: http://www.health.state.ny.us/nysdoh/commish/annual_report/1999/asstcomm.htm.

ODOE (Oregon Department of Energy), 2004. Commissioning for Better Buildings in Oregon. Salem, OR. http://www.energy.state.or.us/bus/comm/bldgcx.htm.

OEHHA (Office of Environmental Health Hazard Assessment), 1997. *Health Effects of Exposure to Environmental Tobacco Smoke.* Final Report. Available from: http://www.oehha.org/air/environmental_tobacco/finalets.html.

OEHHA (Office of Environmental Health Hazard Assessment), 2000a. Acute reference exposure levels (RELs), averaging times, and toxicologic endpoints. Available from: http://www.oehha.ca.gov/air/acute_rels/allAcRELs.html.

OEHHA (Office of Environmental Health Hazard Assessment), 2000b. Asbestos fact sheet available from http://www.oehha.ca.gov/public_info/facts/index.html.

OEHHA (Office of Environmental Health Hazard Assessment), 2000c. Air Toxics Hot Spots Program Risk Assessment Guidelines Part III Technical Support document for the Determination of noncancer Chronic Reference exposure levels. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, April 2000.

OEHHA (Office of Environmental Health Hazard Assessment), 2001. Prioritization of toxic air contaminants under the children's environmental health protection act. OEHHA, Cal/U.S. EPA. October 2001. Available from: http://www.oehha.ca.gov.

OEHHA (Office of Environmental Health Hazard Assessment), 2002. Air toxics hot spots program risk assessment guidelines part two: technical support document for describing available cancer potency factors. OEHHA, Cal/U.S. EPA. April, 1999. Available from: http://www.oehha.ca.gov.

OEHHA, 2003a. All Chronic Reference Exposure Levels Adopted by OEHHA as of August 2003. Available from: http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html.

OEHHA, 2003b. Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant, Part B: Health Effects. November 2003. Public Review Draft. See also: Errata Page, March 3, 2004. http://www.arb.ca.gov/toxics/ets/dreport/dreport.htm.

OEHHA, 2004. Proposition 65 status report safe harbor levels: no significant risk levels for carcinogens and maximum allowable dose levels for chemicals causing reproductive toxicity. Available from: http://oehha.ca.gov/prop65/prop65_list/Newlist.html

OFEE (Office of the Federal Environmental Executive), 2005. Green Janitorial Products and Services. Washington, DC. http://www.ofee.gov/gp/greenjanitorial.html.

Offermann FJ, Colfer R, Radzinski P, and Robertson J, 2002. Exposure to environmental tobacco smoke in an automobile. *Proc. Indoor Air.* 506-511.

Offermann FJ, May 2000, Indoor Air Quality Symposium: Risk Reduction in the 21st Century, Sacramento, CA.

Ostro BD, 1987. Air pollution and morbidity revisited: a specification test. *J of Environmental Economics and Management* **14**: 87-98.

Ostro BD, 1993. The association of air pollution and mortality: examining the case for inference. *Arch Environ Health* **48(5)**: 336-342.

Ostro BD, and Rothschild S, 1989. Air pollution and acute respiratory morbidity: an observational study of multiple pollutants. *Environ Res* **50**: 238-247.

Ostro B, Lipsett M, Mann J, Braxton-Owens H, and White M, 2001. Air pollution and exacerbation of asthma in African-American children in Los Angeles. *Epidemiol* **12(2)**:200-8.

Ostro BD, Broadwin R, and Lipsett MJ, 2000. Coarse and fine particles and daily mortality in the Coachella Valley, California: a follow-up study. *J Expos Analysis and Environ Epidemiology*, **10**: 412-419.

Ott W, Switzer P, and Robinson J, 1996. Particle concentrations inside a tavern before and after prohibition of smoking: Evaluating the performance of an indoor air quality model. *J Air Waste Manag Assoc* **46**: 1120-1134.

Ott WR, and Roberts JW, 1998. Everyday Exposure to Toxic Pollutants. *Sci Am* February 1998: 86-91.

Ott W, Wallace L, and Mage D, 2000. Predicting particulate (PM_{10}) personal exposure distributions using a random component superposition statistical model. *JAir & Waste Management Assoc* **50**: 1390-1406

Ott WR, Klepeis NE, and Switzer P, 2003. Analytical solutions to compartmental indoor air quality models with application to environmental tobacco smoke concentrations measured in a house. *J Air Waste Manag Assoc* **53(8)**: 918-36.

Otten JA, Burge HA, 1999. Viruses. Bioaerosols: Assessment and Control, ed. J. Macher, American Conference of Governmental Industrial Hygienists, Cincinnati, OH.

Ozkaynak H, Xue J, Spengler J, Wallace L, Pellizzari E, Jenkins P, 1996a. Personal exposure to airborne particles and metals: results from the Particle TEAM study in Riverside, California. *J Expo Anal Environ Epidemiol* **6**: 57-77.

Ozkaynak H, Xue J, Weker R, Butler D, Koutrakis P, and Spengler J, 1996b. The Particle TEAM (PTEAM) study: analysis of the data. Report to the U.S. EPA, Volume III of Final Report, 1996a.

Pandian MD, Behar JV, Ott WR, Wallace LA, Wilson AL, Colome SD, Koontz M, 1993. Correcting errors in the nationwide database of residential air change rates. *J Expo Anal Environ Epidemiol* **(8)4**: 577-586.

Pandian MD, Ott WR, Behar JV, 1993. Residential air change rates for use in indoor air and exposure modeling studies. *J Expo Anal Environ Epidemiol* **3(4)**: 407-16

Park J, Spengler JD, Yoon D, Dumyahn T, Lee K, and Ozkaynak H, 1998. Measurement of air exchange rate of stationary vehicles and estimation of in-vehicle exposure. *J Expo Anal Environ Epidemiol* **8**: 1-9; 65-78.

Pasanen A-L, Korpi J-P, Pasanen P, 1998. Critical aspects on the significance of microbial volatile metabolites as indoor air pollutants. *Environment International* **24**: 703-712.

Payne-Sturges DC, Burke TA, Breysse P, Diener-West M, and Buckley TJ, 2004. Personal exposure meets risk assessment: a comparison of measured and modeled exposures and risks in an urban community. *Environmental Health Perspectives* **112(5)**: 589-598.

Peat JK, Dickerson J, Li J, 1998. Effects of damp and mould in the home on respiratory health: a review of the literature. *Allergy* **53**:120-128.

Peipins LA, Lewin M, Campolucci S, Lybarger JA, Miller A, Middleton D, Weis C, Spence M, Black B, and Kapil V, 2003. Radiographic abnormalities and exposure to asbestos-contaminated vermiculite in the community of Libby, Montana, USA. *Environ Health Perspect* **111(14)**: 1753-9.

Pelham TW, Holt LE, and Moss MA, 2001. Exposure to carbon monoxide and nitrogen dioxide in enclosed ice areanas. *Occup Environ Med* **59**: 224-233.

Pelham TW, Holt LE, and Moss MA, 2002. Exposure to carbon monoxide and nitrogen dioxide in enclosed ice arenas. *Occup Environ Med* **59**: 224-310.

Pellizzari ED, Michael LC, Perritt K., Smith DJ, Hartwell TD, and Sebastik J, 1989. Development and Implementation of Exposure Assessment Procedures for Toxic Air Pollutants in Several Los Angeles County, CA Communities. Final report, ARB Contract No. A5-174-33.

Penna MLF, and Duchiade MP, 1991. Air pollution and infant mortality from pneumonia in the Rio de Janeiro metropolitan area. *Bulletin of PAHO* **25**: 47-54.

Peters JM, Avol E, Navidi W, London SJ, Gauderman WJ, Lurmann F, Linn WS, Margolis H, Rappaport E, Gong H, and Thomas DC, 1999a. A study of twelve Southern California communities with differing levels and types of air pollution. I. Prevalence of respiratory morbidity. *Am J Respir Crit Care Med* **159(3)**:760-7.

Peters JM, Avol E, Gauderman WJ, Linn WS, Navidi W, London SJ, Margolis H, Rappaport E, Vora H, Gong H Jr, and Thomas DC, 1999b. A study of twelve Southern California communities with differing levels and types of air pollution. II. Effects on pulmonary function. *Am J Respir Crit Care Med* **159(3)**: 768-75.

Petreas M, She J, Brown FR, Winkler J, Windham G, Rogers E, Zhao G, Bhatia R, and Charles MJ, 2003. High body burdens of 2,2',4,4'-tetrabromodiphenyl ether (BDE-47) in California women. *Environ Health Perspect* **111(9)**: 1175-9.

Phillips TJ, Bloudoff DP, Jenkins PL, and Stroud KR, 1999. Ozone emissions from a "personal air purifier". *J Expo Anal Environ Epidemiol* **9(6)**: 594-601.

Phillips TJ, Mulberg EJ, and Jenkins PL, 1990. Activity Patterns of California adults and adolescents: Appliance use, ventilation practices, and building occupancy. Proceedings, ACEEE 1990 Summer Study of Energy Efficiency in Buildings, Environment **4**: 4.187-4.196. American Council for an Energy-Efficient Economy, Washington, DC.

Phillips TJ, Jenkins PL, and Mulberg EJ, 1991. Children in California: activity patterns and presence of pollutant sources. Proceedings of the 84th Annual Meeting and Exhibition, Air & Waste Management Association, (**17**) Paper no. 91-172.5, June 1991.

Pickrell J, Mokler B, Griffis L, Hobbs C, and Bathija A, 1983. Formaldehyde Release Rate Coefficients from Selected Consumer Products. *Environ Sci Technol* **17(12)**: 753-757.

Pilotto LS, Nitschke M, Smith BJ, Pisaniello D, Ruffin RE, EcElroy HJ, Martin J, and Hiller JE, 2003. Randomized controlled trial of unflued gas heater replacement on respiratory health of asthmatic school children. *International Journal of Epidemiology.* **33**: 208-214.

Pitts JN, Wallington TJ, Biermann HW, and Winer AM, 1985. Identification and measurement of nitrous acid in an indoor environment. *Atmospheric Environment* **19(5)** 763-767.

Pitts JN, Biermann HW, Tuazon EC, Green M, Long WE, and Winer AM, 1989. Time-resolved identification and measurement of indoor air pollutants by spectroscopic techniques: gaseous nitrous acid, methanol, formaldehyde and formic acid. *JAPCA* **39**: 1344-1347.

Platts-Mills TAE, and Carter MC, 1997. Asthma and indoor exposure to allergens. *N Engl J Med* **336**: 1382-1384.

Plopper CG, and Thurlbeck WM, 1994. Growth, Aging, and Adaptation. Textbook of Respiratory Medicine, 2nd Edition. Eds., Murray and Nadel. W.B. Saunders, Philadelphia.

Ponka A, Savela M, and VirtanenM, 1998. Mortality and air pollution in Helsinki. *Arch Environ Health* **53(4)**: 281-286.

Pope CA, Dockery DW, Spengler JD, and Raizenne ME, 1991. Respiratory health and PM_{10} pollution. *Am Rev Respir Dis* **144**: 668-674.

Pope CA, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, and Heath CW, 1995. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *Am J Respir Crit Care Med* **151**: 669-674.

Poytress J, 2003. Maintenance and Operations, Clovis Unified School District, Clovis, CA. Personal communications. November 14.

Ransom MR, Pope CA III, 1992. Elementary school absences and PM10 pollution in Utah Valley. *Environ Res* **58**: 204-219.

Raub JA, Mathieu-Nolf M, Hampson NB, and Thom SR, 2000. Carbon monoxide poisoning – a public health perspective. *Toxicology* **145**: 1-14.

Reiss R, Ryan PB, Koutrakis P, and Tibbetts SJ, 1995a. Ozone reactive chemistry on interior latex paint. *Environ Sci Technol* **29(8)**: 1906-12.

Reiss R, Ryan PB, Tibbetts SJ, and Koutrakis P, 1995b. Measurement of organic acids, aldehydes, and ketones in residential environments and their relation to ozone. *J Air Waste Manag Assoc* **45(10)**: 811-22.

Repace JL, 2003. An air quality survey of respirable particles and particulate carcinogens in Delaware hospitality venues before and after a smoking ban. Repace Associates, Inc., Bowie MD.

Repair Clinic, 2004. Answers To Common Questions About Range Vent Hoods. http://www.repairclinic.com/0088_7_4.asp.

Rhodes L, Bailey CM, and Moorman JE, 2002. Asthma prevalence and control characteristics by race/ethnicity. *MMWR* **53(07)**: 145-148. http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5307a1.htm.

Riley WJ, McKone TE, Lai ACK, and Nazaroff WW, 2002. Indoor particulate matter of outdoor origin: Importance of size-dependent removal mechanisms. *Environ Sci & Technol* **36**: 200-207. [See errata on p. 1868.]

Ritz B, Yu F, Chapa G, and Fruin S, 2000. Effect of air pollution on preterm birth among children born in Southern California between 1989 and 1993. *Epidemiology* **11(5)**: 502-511.

Ritz, B., Yu, F., Fruin, S., Chapa, G., Shaw, G., Harris, J. (2002). Ambient air pollution and risk of birth defects in Southern California. Am. J. of Epidemiology. 1:17-25.

Roberts JW, and Dickey P, 1995. Exposure of children to pollutants in house dust and indoor air. *Rev Environ Contam Toxicol* **143**: 60-77.

Rodes C, Sheldon L, Whitaker D, Clayton A, Fitzgerald K, Flanagan J, DiGenova F, Hering S, and Frazier C, 1998. Measuring concentrations of selected air pollutants inside California vehicles. Final Report to the California Air Resources Board, Contract no. 95-339, Sacramento CA.

Rodes CE, Lawless PA, Evans GF, Sheldon LS, Williams RW, Vette AF, Creason JP, and Walsh D, 2001. The relationships between personal PM exposures for elderly populations and indoor and outdoor concentrations for three retirement center scenarios. *J Expo Anal Environ Epidemiol* **11(2)**: 103-115.

Roinestad KS, Louis JB, and Rosen JD, 1993. Determination of pesticides in indoor air and dust. *J AOAC Int* **76**: 1121-1126.

Rojas-Bracho L, Suh HH, and Koutrakis P, 2000. Relationships among personal, indoor, and outdoor fine and coarse particle concentrations for individuals with COPD. *J Expo Anal Environ Epidemiol* **10**: 294-306.

Rosenfeld AH, 1999. The Art of Energy Efficiency: Protecting the Environment with Better Technology. *Ann Rev Energy Environ* **24**: 33-82.

Rosenman KD, Reilly MJ, Schill DP, Valiante D, Flattery J, Harrison R, Reinisch F, Pechter E, Davis L, Tumpowsky CM, and Filios M, 2003. Cleaning products and work-related asthma. *J Occup Environ Med* **45**: 556-563.

Rosenstreich DL, Eggleston P, Kattan M, Baker D, Slavin RG, Gergen P, Mitchell H, McNiff-Mortimer K, Lynn H, Ownby D, Malveaux F, 1997. The role of cockroach allergy and exposure to cockroach allergen in causing morbidity among inner-city children with asthma. *N Engl J Med* **336**: 1356-13634.

Rothenberg S, Nagy P, Pickrell J, and Hobbs C, 1989. Surface area, adsorption, and desorption studies on indoor dust samples. *Am Ind Hyg Assoc J* **50(1)**: 15-23.

Rudel RA, Camann DE, Spengler JD, Korn LE, and Brody JG, 2003. Phthalates, alkylphenols, pesticides, polybrominated diphenyl ethers, and other endocrine-disrupting compounds in indoor air and dust. *Environ Sci Technol* **37(20)**: 4543-4553.

Ryan TJ, Hart EM, and Kappler LL, 2002. VOC exposures in a mixed-use university art building. *American Industrial Hygiene Association Journal* **63**: 703-708.

Rylander R, Megevand T, 2000. Environmental risk factors for respiratory infections. *Archives of Environ Health.* **55(5)**:300-303.

Salam MT, Li Y-F, Langholz B, Gilliland FD, 2003. Early-life environmental risk factors for asthma: findings from the Children's Health Study. *Environ Health Perspect* **112(6)**: 760-765.

Samet JM, Dominici F, Curriero FC, Coursac MS, and Zeger SL, 2000. Fine particulate air pollution and mortality in 20 U.S. cities 1987-1994. *N Engl J Med* **343(24)**: 1742-1749.

Samat JA. Koutrakis P. and Suh HH. 2000. Assessing the relationship between personal particulate and gaseous exposures of senior citizens living in Baltimore, MD. *Journal of the Air & Waste Management Association.* **50(7)**:1184-1198.

Sarpong SB, Wood RA, and Eggleston PA, 1996. Short-term effects of extermination and cleaning on cockroach allergen Bla g 2 in settled dust. *Ann Allergy Asthma Immunol.* **Mar76(3)**: 257-60.

Sarwar G, Olson DA, Corsi RL, and Weschler CJ, 2004. Indoor fine particles: the role of terpene emissions from consumer products. *Journal of Air and Waste Management Association* **54**: 367-377.

SCAQMD (South Coast Air Quality Management District), 1999. Rule 1121 - Control Of Nitrogen Oxides From Residential Type, Natural Gas-Fired Water Heaters. http://www.arb.ca.gov/DRDB/SC/CURHTML/R1121.HTM. Diamond Bar, CA.

Schecter A, Pavuk M, PapkeO, Ryan JJ, Birnbaum L, and Rosen R, 2003. Polybrominated Diphenyl Ethers (PBDEs) in U.S. Mothers' Milk. *Environ Health Perspect* **111**(14): 1723-1729.

Schelegle ES, and Adams WC, 1986. Reduced exercise time in competitive simulations consequent to low level ozone exposure. *Med Sci Sports Exerc* **18(4)**: 408-414.

Schneider M, 2002. Public School Facilities and Teaching: Washington, DC and Chicago. November 2002. A Report Prepared for the Neighborhood Capital Budget Group (NCBG). Available from: http://www.ncbg.org/press/press111302.htm.

Schneider T, Sundell J, Bischof W, Bohgard M, Cherrie JW, Clausen PA, Dreborg S, Kildese J, Kjaergaard SK, Lovik M, Pasanen P, and Skyberg K, 2003. Europart. Airborne particles in the indoor environment. A European interdisciplinary review of scientific evidence on associations between exposure to particles in buildings and health effects. *Indoor Air* 13 (1): 38-48.

Schwartz J, 1994. Air pollution and daily mortality: a review and meta analysis. *Environ Res* **64**: 36-52.

Schwartz J, 1995. Air pollution and hospital admissions for cardiovascular disease in Detroit, Michigan. *Am J Epidemiol* **142(1)**:23-35.

SchwartzJ, 2003. Daily deaths associated with air pollution in six US cities and short-term mortality displacement in Boston in Health Effects Institute Special Report: Revised Analyses of Time-Series Studies of Air Pollution and Health.

Schwartz J, and Neas M, 2000. Fine particles are more strongly associated than coarse particles with acute respiratory health effects in schoolchildren. *Epidemiology* **11(1)**: 6-10.

Seal E, McDonnell WF, House DE, Salaam SA, Dewitt PJ, Butler SO, Green J, and Raggio L, 1993. The pulmonary response of white and black adults to six concentrations of ozone. *Am Rev Respir Dis* **147**: 804-810.

Selway MD, Allen RJ, and Wadden RA, 1980. Ozone production from photocopying machines. *Am Ind Hyg Assoc J* **41(6)**: 455-9.

Seppanen O, and Fisk WJ. 2002. Association of system with SBS symptoms in office workers. *Indoor Air.* **12(2)**: 98-112.

Sexton K, Adgate SJ, Ramachandran G, Pratt GC, Mongin SJ, Stock TH, and Morandi MT, 2004. Comparison of personal, indoor, and outdoor exposures to hazardous air pollutants in three urban communities. *Environ Sci Technol* **38**: 423-430.

Sexton K, Petreas MX, Liu KS, and Kulasingam GC, 1985. Formaldehyde concentrations measured in California mobile homes. For Presentation at the 78th Annual Meeting of the Air Pollution Control Association.

Sexton K, Liu KS, and Petreas MX, 1986. Formaldehyde concentrations inside private residences: a mail-out approach to indoor air monitoring. *Journal of the Air Pollution Control Association* **36(6)**: 698-704.

Sheldon LS, Handy RW, Hartwell TD, Whitmore RW, Zelon HS, and Pellizzari ED, 1988. Indoor air quality in public buildings: volume 1. U.S. EPA, Washington DC; U.S. EPA 600/6-88/009a. NTIS PB89-102503/AS.

Sheldon L, Clayton A, Jones B, Keever J, Perritt R, Smith D, Whitaker D, and Whitmore R, 1992a. Indoor Pollutant Concentrations and Exposures. Final report to ARB Contract No. A833-156.

Sheldon LS, Clayton CA, Keever J, Perritt RL, and Whitaker DA, 1992b. PTEAM: Monitoring of Phthalates and PAHs in Indoor and Outdoor Air Samples in Riverside, California - Volume 2. Final report to ARB, Contract No. A933-144.

Sheldon LS, Clayton CA, Keever J, Perritt RL, and Whitaker DA, 1993. Indoor concentrations of polycyclic aromatic hydrocarbons in California Residences, ARB Contract No. A033-132.

Shelton BG, Kirkland KH, Flanders WD, Morris GK, 2002. Profiles of airborne fungi in buildings and outdoor environments in the United States. *Applied and Environmental Microbiology* **68(4)**:1743-1753.

Shendell DG, Barnett C, and Boese, 2004. Science-based recommendations to prevent or reduce potential exposures to biological, chemical, and physical agents in schools. Journal of School Health, in press (likely December 2004 issue). Also available as extended complete final report in PDF at: http://www.healthyschools.org/documents/HPSchlsWhtPpr.pdf

Shepherd JL, Corsi RL, and Kemp J, 1996. Chloroform in Indoor Air and Wastewater: The Role of Residential Washing Machines. *J Air Waste Manage Assoc* **46(_)**:____.

Sheppard L, Levy D, Norris G, Larson TV, and Koenig JQ, 1999. Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington. *Epidemiology* **10(1)**: 23-30.

Sheppard L, 2003. Ambient air pollution and non-elderly asthma hospital admissions in Seattle, Washington, 1987-1994 in Health Effects Institute Special Report: Revised Analyses of Time-Series Studies of Air Pollution and Health.

Sherman, M, 2003a. ASHRAE & Residential Ventilation. LBNL Report Number 53776, October. Energy Performance of Buildings Group, Indoor Environment Department, Environmental Energy Technology Division, Lawrence Berkeley National Laboratory. http://epb.lbl.gov/Publications/lbnl-53776.pdf.

Sherman, M, 2003b. ASHRAE's First Residential Ventilation Standard. LBNL Report Number 54331. Energy Performance of Buildings Group, Indoor Environment Department, Environmental Energy Technology Division, Lawrence Berkeley National Laboratory. Berkeley, CA. http://epb.lbl.gov/Publications/lbnl-54331.pdf,

Shields HC, Fleischer DM, and Weschler CJ, 1996. Comparisons among VOCs measured in three types of U.S. commercial buildings with different occupant densities. *Indoor Air* **6**: 2-17.

Siegmann K, and Sattler K, 1996. Aerosol from hot cooking oil, a possible health hazard. *Journal of Aerosol Science* **27, Supplement 1**: S493-S494.

Singer BC, Hodgson, AT, Guevarra KS, Hawley KL, and Nazaroff WW, 2002. Gas-phase organics in environmental tobacco smoke. 1. Effects of smoking rate, ventilation, and furnishing level on emission factors. *Environmental Science and Technology* **35(5)**: 846-853.

Singer BC, Hodgson AT and Nazaroff WW, 2003. Gas-phase organics in environmental tobacco smoke: 2. Exposure-relevant emission factors and indirect exposures from habitual smoking. *Atmospheric Environment* **37**: 5551-5561.

Simcox JJ, Fenske RA, Wolz SA, Lee IC, and Kalman D, 1995. Pesticides in household dust and soil: Exposure pathways for children of agricultural families. *Environ Health Perspect* **103**: 1126-1134.

SJVUAPCD (San Joaquin Valley Unified Air Pollution Control District), 2003. Rule 4901 - Wood Burning Fireplaces And Wood Burning Heaters. http://www.arb.ca.gov/drdb/sju/curhtml/r4901.pdf. Fresno, CA.

Smith DH, Malone DC, Lawson KA, Okamoto LJ, Battista C, and Saunders WB, 1997. A national estimate of the economic cost of asthma. *Am J Respir Crit Care Med* **156**: 787-793.

Smith K, 1988. Air Pollution: Assessing Total Exposure in the United States. *Environment* **30(8)**: 10-38.

Song BJ, Liu AH, 2003. Metropolitan endotoxin exposure, allergy and asthma. *Curr Opin Allergy Clin Immunol* **3**:331-335.

Soukup JM, and Becker S, 2001. Human alveolar macrophage responses to air pollution particulates are associated with insoluble components of coarse material, including particulate endotoxin. *Toxicol Appl Pharmacol* **171(1)**: 20-26.

Sparks LE, Guo Z, Change JC, and Tichenor BA, 1999. Volatile organic compound emissions from latex paint-part 1. Chamber experiments and source model development. *Indoor Air* **9**: 10-17.

Spengler J, Brauer M, Samet JM, and Lambert WE, 1993. Nitrous acid in Albuquerque, New Mexico homes. *Environ. Sci. Technol.* **27**: 841-845.

Spengler JD, and Chen Q, 2000. Indoor Air Quality Factors in Designing a Health Building. *Ann Rev Energy Environ* **25**: 567-601.

Spengler J, Neas L, Nakai S, Dockery D, Speizer F, Ware J, and Raizenne M, 1994. Respiratory symptoms and housing characteristics. *Indoor Air* **4**: 72-82.

Spengler J, Schwat M, Billick I, Colome S, Wilson A L, and Becker E, 1994b. Personal exposure to nitrogen dioxide in the Los Angeles Basin. *J. Air Waste Manage. Assoc.* **44**: 39-47.

Spengler JD, Samet JM, and McCarthy JF, 2001. Indoor Air Quality Handbook. McGraw-Hill. New York.

Spengler JD, Dockery DW, Turner WA, Wolfson JM, Ferris BG, 1981. Long-term measurements of respirable sulfates and particles inside andn outside homes. *Atmos Environ.* 15:23-30.

Spicer CW, Kenny DV, Ward, GF, and Billick IH, 1993. Transformations, lifetimes, and sources of NO_2, HONO, and HNO_3 in indoor environments. *J. Air Waste Manage. Assoc.* **43**: 1479-1485.

Springston JP, Esposito WA, and Cleversey KW, 2002. Baseline indoor air quality measurements collected from 136 metropolitan New York region commercial office buildings between 1997-1999. *Am Ind Hyg Assoc J* **63(3)**: 354-60.

State of California, 2002. Exhibit c: energy efficiency and sustainable building measures tiers one and two. State of California standard agreement, STD 2 RESD. July 2001. Available from: http://www.ciwmb.ca.gov/greenbuilding/design/guidelines.htm.

State of Washington, 2003. Washington State Ventilation and Indoor Air Quality Code (2003 Edition), Section 51-3. Washington State Building Code Council, Olympia, WA. ftp://198.147.238.10/energy.wsu.edu/pubs/code/2003VIAQ.pdf.

Stefaniak AB, Breysse PN, Murray PM, Rooney BC, and Schaefer J, 2000. An evaluation of employee exposure to volatile organic compounds in three photocopy centers. *Environmental Research Section A* **83:**162-173.

Storey E Dangman KH, Schenck P, DeBernardo RL, Yang CS, Bracker A and Hodgson MJ, 2004. Guidance for Clinicians on the Recognition and Management of Health Effects Related to Mold Exposure and Moisture Indoors. Farmington, CT, Center for Indoor Environments and Health at UConn Health Center. http://www.oehc.uchc.edu/clinser/MOLD%20GUIDE.pdf

Strandberg B, Dodder NG, Basu I, and Hites RA, 2001. Concentrations and spatial variations of polybrominated diphenyl ethers and other organohalogen compounds in Great Lakes air. *Environ Sci Technol* **35(6)**:1078-83.

Stratton J, April 1997, Press Release 27-97, California Department of Health Services, Sacramento, CA.

Suh H, 2003. Characterization of the composition of personal, indoor, and outdoor particulate matter exposures. Final report to ARB for contract number 98-330.

Suh H, and Koutrakis P, 2004. Detailed characterization of indoor and personal particulate matter concentrations. Final report to ARB for contract number 00-302.

Summerbell RC, 2001. Respiratory tract infections caused by indoor fungi. In: Microorganisms in Home and Indoor Work Environments. Editors: B Flannigan, RA Samson, JD Miller. Taylor & Francis. New York, NY.

Swan SH, Beaumont JJ, Hammond SK, VonBehren J, Green RS, Hallock MF, Woskie SR, Hines CJ, and Schenker MB, 1995. Historical cohort study of spontaneous abortion among fabrication workers in the semiconductor health study: agent-level analysis. *American Journal of Industrial Medicine* **28:** 751-769.

Switzer P, Klepeis N, and Ott W, 2001. Quantification of population exposure to secondhand smoke. Final Report (Grant 6RT-0118) submitted to the Tobacco-Related Disease Research Program of the University of California.

Takaro TK, Krieger JW, and Song L, 2004. Effect of environmental interventions to reduce exposure to asthma triggers children in Seattle. *J Expo Anal Environ Epidemiol* **14(Suppl1)**: s133-43.

Taylor, C, 2005. Personal communication, ARB Research Division, Sacramento.

Tenbrinke JT, Selvin S, Hodgson AT, Fisk WJ, Mendell MJ, Koshland CP, and Daisey JM, 1998. Development of new volatile organic compound (voc) exposure metrics and their relationship to "Sick Building Syndrome" symptoms. *Indoor Air* **8**: 140-152.

Thacker SB, Hoffman DA, Smith J, Steinberg K, and Zack M, 1992. Effect of low-level body burdens of lead on the mental development of children: limitations of meta-analysis in a review of longitudinal data. *Arch Environ Health* **47(5)**:336-46.

Thatcher TL, and Layton DW, 1995. Deposition, resuspension, and penetrations of particles within a residence. *Atmos Environ* **29(13)**: 1487-1497.

Thayer MA, Chestnut LG, Laza JK, and Van Den Eeden SK, 2003. The economic value of respiratory and cardiovascular hospitalizations. San Diego State University. Report prepared for the California Air Resources Board and California Environmental Protection Agency. http://www.arb.ca.gov/research/apr/past/econ.htm.

The Facts About...Health-Care Cost Inflation. Medical Care Cost Inflation vs. Overall Cost Inflation in the United States, November 1992 - November 2002, Percentage Increase in Consumer Price Index.

Thompson WW, Shay DK, Weintraub E, Brammer L, Cox N, Anderson LJ, Fukuda K, 2003. Mortality associated with influenza and respiratory syncytial virus in the United States. J *Am Med Assoc* **289**: 179-186.

Thurlbeck WM, 1988. Growth, Aging, and Adaptation. In: Murray, JF, Nadel JA, editors: Textbook of Respiratory Medicine, Philadelphia, 1988. W.B. Saunders Company, 37-46.

Thurston GD, Lippmann M, Scott MB, and Fine JM, 1997. Summertime haze air pollution and children with asthma. *Am J Respir Crit Care Med* **155**: 654-660.

Tichenor, BA, 1989. Measurement of organic compound emissions using small test chambers. *Environment International* **(15)**: 389-396.

Tichenor, BA, and Mason, MA, 1988. Organic emissions from consumer products and building materials to the indoor environment. *Journal of the Air Pollution Control Association* **38**: 264-268.

Townsend CL, and Maynard RL, 2002. Effects on health of prolonged exposure to low concentrations of carbon monoxide. *Occup Environ Med* **59**: 708-714.

Traynor GW, 1999. Evaluation of technical literature, indoor air pollution modeling, options for California standards, and recommended standards. Implementation of SB 798: development of standards for unvented gas logs and decorative fireplaces. T. Marshall Associated, Ltd., Pleasanton, CA. Prepared for the California Department of Health Services, Indoor Air Quality Section, Berkeley, CA.

Traynor GW, Apte MG, Carruthers AR, Dillworth JF, Grimsrud DT, and Gundel LA, 1987. Indoor air pollution due to emissions from woodburning stoves. *Environ Sci Technol* **21(7)**: 691-697.

Tsai FC, and Waldman JM, 2004. The California Sierra Radon Study. California Department of Health Services, Environmental Health Laboratory Branch. EHLB Report No. 173.

Tsai FC, and Waldman JM, 2005. The California Sierra Radon Study. Environmental Health Laboratory Report CA/DHS/EHLB/R-173. CA Department of Health Services. 850 Marina Bay Parkway. Richmond, CA 94804.

Tsongas G and Hager WD, 1994. Field monitoring of elevated carbon monoxide production from residential gas ovens. In: Proceedings of the American Society of Heating, Refrigerating,

and Air-Conditioning Engineers, Inc, Indoor Air Quality '94 Conference. Atlanta, Georgia: American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc, 1994.

Tsongas G, 1995. Carbon Monoxide from Ovens: A Serious IAQ Problem. Home Energy Magazine, September/October. Available at: http://hem.dis.anl.gov/eehem/95/950907.html.

Tuomainen A, Seuri M, Sieppi A, 2004. Indoor air quality and health problems associated with damp floor coverings. *Int Arch Occup Environ Health* **77**:222-226.

Turk BH, Brown JT, Geisling-Sobotka K, Froehlich DA, Grimsrud DT, Harrison J, Koonce JF, Prill RJ, and Revsan KL, 1987. Indoor air quality and ventilation measurements in 38 Pacific Northwest commercial buildings. Volume I: measurement results and interpretation. Lawrence Berkeley Laboratory; Final report, LBL-22315.

UL (Underwriters Laboratory), 2002. Standard 2034, Standard for Single and Multiple Station Carbon Monoxide Alarms, ANSI Approved. Northbrook, IL.

USCB (U.S. Census Bureau), 1990. American Fact Finder: 1990 Summary Tape File 1 (STF 1) - 100-Percent data. Available from: www.census.gov.

USCB (U.S. Census Bureau), 2002. Statistical abstract of the United States. http://www.census.gov/prod/www/statistical-abstract-us.html.

USCB (U.S. Census Bureau), 2002. The American Housing Survey. http://www.census.gov/hhes/www/ahs.html

USCB, 2003. California: 2000. Summary Social, Economic, and Housing Characteristics, 2000 Census of Population and Housing. March 2003. PHC-2-6.

U.S. DHHS, 1986. The health consequences of Involuntary smoking: A report of the Surgeon General. U.S. DHHS, Public Health Service, Centers for Disease Control. DHHS Publication No. (CDC) 87-8398.

U.S. DHHS, 1989. Reducing the health consequences of smoking. 25 years of progress: A report of the Surgeon General. U.S. DHHS, Centers for Disease Control, Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. DHHS Publication No. (CDC) 89-8411.

U.S. DOE, 2003a. California weatherization and low-income energy programs. Washington, DC. http://www.eere.energy.gov/weatherization/cfm/index.cfm/state_abbr=ca.

U.S. DOE, 2003b. Weatherization Assistance Program Guidelines & Procedures. Washington, DC. http://www.eere.energy.gov/weatherization/prog_guide.html.

U.S. EPA, 1984. Health effects assessment for benzo(a)pyrene, Cincinnati, OH: Environmental Criteria and Assessment Office, EPA 540/1-86-022, PB86-134335.

U.S. EPA, 1987a. Unfinished Business: A Comparative Assessment of Environmental Problems (Overview report and 4 volumes), US Environmental Protection Agency, Office of Policy analysis, Office of Policy, Planning and Evaluation, Washington DC, February 1987.

U.S. EPA, 1987b. Assessment of health risks to garment workers and certain home residents from exposure to formaldehyde. Office of Pesticides and Toxic Substances.

U.S. EPA, 1987c. Locating and estimating air emissions from sources of polycyclic organic matter (POM). EPA 540/1-86-022, PB86-134335.

U.S. EPA, 1989. Risk assessment guidance for Superfund Human Health Evaluation Manual 1 (U.S. EPA/540/1-89/002).

U.S. EPA, 1991. Building Air Quality: A Guide for Building and Facility Managers. Washington, DC. http://www.epa.gov/iaq/largebldgs/baqtoc.html.

U.S. EPA, 1992. Respiratory health effects of passive smoking: Lung cancer and other disorders. U.S. EPA/600/6-90/006F. U.S. EPA Office of Research and Development, Washington, DC.

U.S. EPA, 1994. Health effects notebook for hazardous air pollutants. Available at http://www.epa.gov/ttn/atw/hapindex.html. Air Risk Information Support Center (Air RISC), Research Triangle Park, North Carolina. Contract No. 68-D2-0065.

U.S. EPA, 1995. Office equipment: design, indoor air emissions, and pollution prevention opportunities. Washington, DC: Office of Research and Development; 1995. Report no. EPA/600/R-95/045.

U.S. EPA, 1996. Safe Drinking Water Act: Amendments of 1996.
Available from: http://www.U.S. EPA.gov/safewater/sdwa/sdwa.html.

U.S. EPA, 1997a. Summary and assessment of published information on determining lead exposures and mitigating lead hazards associated with dust and soil in residential carpets, furniture, and forced air ducts. Washington, D.C.: Office of Pollution Prevention and Toxics, USEPA. EPA 747-S-97-001.

U.S. EPA, 1997b. An Office Building Occupant's Guide to Indoor Air Quality. Washington, DC. http://www.epa.gov/iaq/pubs/occupgd.html.

U.S. EPA, 1998. Building Air Quality (BAQ) Action Plan. (EPA/402-K-98-001, DHHS (NIOSH) Pub. No. 98-123). Available from: http://www.epa.gov/iaq/largebldgs/baqact.html.

U.S. EPA, 1999a. Transport of Lawn-Applied 2,4-D from turf to home: Assessing the relative importance of transport mechanisms and exposure pathways. U.S. EPA 600-R-99-040. Research Triangle Park, NC.

U.S. EPA, 1999b. Reducing Mercury Use in Healthcare: Promoting a Healthier Environment. Available at http://www.epa.gov/glnpo/bnsdocs/merchealth/.

U.S. EPA, 1999c. Integrated Risk Information System, Ethylene glycol monobutyl ether (EGBE)(2-Butoxyethanol) (CASRN 111-76-2), available at http://www.epa.gov/iris/subst/0500.htm#refinhal

U.S. EPA, 2000a. Case Studies - G.W. Carver and Charles Drew Elementary Schools. U.S. EPA 402-F-00-010A. August 2000. http://www.U.S. EPA.gov/iaq/schools/casestudies/caseca.html.

U.S. EPA, 2000b. Case Study: Little Harbour School, Portsmouth School Department, Portsmouth, New Hampshire. http://www.U.S. EPA.gov/iaq/schools/casestudies/iaqharbo.pdf.

U.S. EPA, 2000c. Regulatory impact analysis: heavy-duty engine and vehicle standards and highway diesel fuel sulfur control requirements. EPA-420-R-00-026. U.S. Environmental Protection Agency, Office of Air and Radiation, Research Triangle Park, NC.

U.S. EPA, 2000c. Final Rulemaking Documents and Fact Sheets. Control of air pollution from new motor vehicles: Heavy-duty engine and vehicle standards and highway diesel fuel sulfur control Requirements. Regulatory Impact Analysis (U.S. EPA420-R-00-026), Chapter VII: Benefit-Cost Analysis. http://www.U.S. EPA.gov/otaq/regs/hd2007/frm/ria-vii.pdf. P. VII-50.

U.S. EPA, 2000d. Chlorpyrifos Revised Risk Assessment and Agreement with Registrants. Available from: http://www.epa.gov/pesticides/op/chlorpyrifos/agreement.pdf.

U.S. EPA, 2000e. Proposed Radon in Drinking Water Rule. U.S. EPA 815-F-99-009. Available from: http://www.U.S. EPA.gov/safewater/radon/proposal.html.

U.S. EPA, 2000f. DDT. Persistent bioaccumulative and toxic (PHT) chemical program, http://www.epa.gov/pbt/ddt.htm.

U.S. EPA, 2000g. Aldrin/Dieldrin. Persistent bioaccumulative and toxic (PBT) chemical program, http://www.epa.gov/pbt/aldrin.htm.

U.S. EPA, 2001a. Diazinon Revised Risk Assessment and Agreement with Registrants. Available from: http://www.epa.gov/pesticides/op/diazinon/agreement.pdf.

U.S. EPA , 2001b. Mold Remediation in Schools and Commercial Buildings. (U.S. EPA/402/K-01/001) Washington, DC. Available at www.epa.gov/mold/moldresources.html

U.S. EPA, 2002a. A Citizen's Guide to Radon: The Guide to Protecting Yourself and Your Family From Radon (4th edition). U.S. EPA 402-K02-006. Available from: http://www.U.S. EPA.gov/iaq/radon/pubs/citguide.html.

U.S. EPA, 2002b. Cost of Illness Handbook; U.S. Environmental Protection Agency, http://www.epa.gov/oppt/coi/toc.html.

U.S. EPA, 2003a. Building Assessment Survey and Evaluation (BASE) Study. Available from: http://www.epa.gov/iaq.largebldgs/index.html.

U.S. EPA, 2003b. IAQ Design Tools for Schools. Available from: http://www.epa.gov/iaq/schooldesign/.

U.S. EPA, 2003c. EPA Assessment of Risks from Radon in Home. EPA 402-R-03-003. Office of Air and Radiation. Washington, D.C. http://www.epa.gov/radon/risk_assessment.html.

U.S. EPA, undated. Ozone Generators that are Sold as Air Cleaners: An Assessment of Effectiveness and Health Consequences. http://www.epa.gov/iaq/pubs/ozonegen.html.

U. S. EPA, 2004. National Ambient Air Quality Standards. Available at: http://www.epa.gov/air/criteria.html.

U.S. Green Building Council, 2004. Green Building Rating System: For Existing Buildings Operations and Upgrades (LEED-EB). Available from: http://www.usgbc.org/Docs/LEEDdocs/PublicCommentDraftLEED-EB20040223.pdf.

Van Alphen, 1999. Emission testing and inhalational exposure-based risk assessment for candles having Pb metal wick cores. *The Science of the Total Environment* **243/244**: 53-56.

Verhoeff AP, Burge HA, 1997. Health risk assessment of fungi in home environments. *Ann Allergy Asthma Immunol* **78**: 544-556.

Vette A, Rea A, Lawless P, Rodes C, Evans G, Highsmith R, and Sheldon L, 2001. Characterization of indoor-outdoor aerosol concentration relationships during the Fresno PM exposure studies. *Aerosol Sci Technol* **34**: 118-126.

Viberg H, Fredriksson A, and Jakobsson E, 2000. Developmental neurotoxic effects of 2,2,4,4,5-pentabromodiphenyl ether (PBDE 99) in the neonatal mouse. *Toxicologist* **54**: 1360.

Viberg H, Fredrikkson A, Jakobsson E, Ohrn U, and Eriksson P, 2001. Brominated flame-retardant: uptake, retention and developmental nurotoxic effects of decabromo-diphenyl ether (PBDE 209) in the neonatal. *Toxicologist* **61**: 1034 (abstract).

Vogelzang PFJ, Joost WJ, van der Gulden J, Fogering H, Kolk JJ, Heederik D, Preller L, Tielen MHM, van Schyck CP, 1998. Endotoxin exposure as a major determinant of lung function decline in pig farmers. *Amer J Resp Crit Care Med* **157**: 15-18.

Volberg DI, Surgan MH, Jaffe S, Hamer D, and Sevinsky JA, 1993. Pesticides in Schools: Reducing the Risks. New York, NY: New York Office of the Attorney General.

Vu-Duc T, and Huynh C, 1989. Sidestream tobacco smoke constituents in indoor air modeled in an experimental chamber – polycyclic aromatic hydrocarbons. *Environ Int* **15**: 57-64.

Wainman T, Zhang J, Weschler CJ, and Lioy PJ, 2000. Ozone and limonene in indoor air: a source of submicron particle exposure. *Environ Health Perspect* **108(12)**: 1139-45.

Waldman JM, Buckley TJ, and Lioy PJ, 1989. Indoor and outdoor levels of benzo(a)pyrene in a community of older homes. For presentation at the 82nd annual meeting, Air and Waste Management Association, Anaheim, CA, June 25-30, 1989.

Waldman JM, and Liu KS, 1996. Unintentional Carbon Monoxide Poisoning in California: Determining Risk Factors for Deaths and Hospitalization. Presented at the 6th annual conference of the International Society of Exposure Analysis, New Orleans.

Wallace LA, 1987. The Total Exposure Assessment Methodology (TEAM) Study, Project Summary, EPA/600/S6-87/002. U.S. Environmental Protection Agency, Washington, D.C.

Wallace L, 1991. Comparison of risks from outdoor and indoor exposure to toxic chemicals. *Environ Health Perspect* **95**: 7-13.

Wallace L, 1993. A decade of studies of human exposure: what have we learned? *Risk Anal* **13(2)**: 135-139.

Wallace L, 1996a. Indoor particles: a review. *J Air Waste Manage Assoc* **46**: 98-126.

Wallace L, 1996b. Environmental Exposure to Benzene: An Update. *Environ Health Perspect* **104(6)**: 1129- 1136.

Wallace L, 2000a. Correlations of personal exposure to particles with outdoor air measurements: a review of recent studies. *Aerosol Sci Technol* **32**: 15-25.

Wallace LA, 2000b. Real-time monitoring of particles, PAH, and CO in an occupied townhouse. *Appl Occup Environ Hyp* **15**: 39-47.

Wallace LA, 2001. Human exposure to volatile organic pollutants: implications for indoor air studies. *Ann Rev Energy Environ* **26**: 269-301.

Wallace L, Pellizzari E, Hartwell TD, Sparacino C, Whitmore R, Sheldon L, Zelon H, and Perritt R, 1987a. The TEAM study: personal exposures to toxic substances in air, drinking, water, and breath of 400 residents of New Jersey, North Carolina, and North Dakota. *Environ Res* **43**: 290-307.

Wallace L, Pellizzari E, Leaderer B, Zelon H, and Sheldon L, 1987b. Emissions of volatile organic compounds from building materials and consumer products. *Atmos Environ* **21(2)**: 385-393.

Wallace L, Pellizzari E, Hartwell TD, Whitmore R, Zelon H, Perritt R, and Sheldon L, 1988. The California team study: breath concentrations and personal exposures to 26 volatile compounds in air and drinking water of 188 residents of Los Angeles, Antioch, and Pittsburgh, CA. *Atmos Environ* **22(10)**: 2141-2163.

Wallace LA, Pellizzari ED, Hartwell TD, Davis V, Michael LC, and Whitmore RW, 1989. The influence of personal activities on exposure to volatile organic compounds. *Environmental Research* **50**: 37-55.

Wallace L, Nelson WC, Ziegenfus R, and Pellizzari E, 1991a. The Los Angeles TEAM study: personal exposures, indoor-outdoor air concentrations and breath concentrations of 25 volatile organic compounds. *J Expo Anal Environ Epidemiol* **1**: 37-72.

Wallace, LA, William N, Ziegenfus R, Pellizzari ED, Michael LC, Zelon H, Hartwell T, Perritt R, and Westerdahl D, 1991c. The Los Angeles TEAM study: Personal exposures, indoor-outdoor air concentrations, and breath concentrations of 25 volatile organic compounds. *J Expo Anal Environ Epidemiol* **1(2)**: 157-192.

Wallace LA, Mitchell H, O'Connor GT, Neas L, Lippmann M, Kattan M, Koenig J, Stout JW, Vaughan BJ, Wallace D, Walter M, Adams K, and Liu L-JS, 2003. Particle concentrations in inner-city homes of children with asthma: the effect of smoking, cooking, and outdoor pollution. *Environ Health Perspect* **111**: 1265-1272.

Wargocki P, Wyon DP, Yong KB, Clausen G, and Fanger PO, 1999. Perceived Air Quality, Sick Building Syndrome (SBS) Symptoms and Productivity in an Office with Two Different Pollution Loads. *Indoor Air* **9**: 165-179.

Wargocki P, Sundell J, Bischof W, Brundrett G, Fanger PO, Gyntelberg F, Hanssen SO, Harrison P, Pickering A, Seppanen O, and Wouters P, 2002. Ventilation and health in non-industrial indoor environments: report from a European Multidisciplinary Scientific Consensus Meeting (EUROVEN). *Indoor Air* **12:** 113-128.

Wargocki P, and Djukanovic R, 2003. Estimate of an economic benefit from investment in improved indoor air quality in an office building. *Proceedings of the Healthy Buildings 2003 Conference, Singapore* **3**: 382-387. Singapore, NUS.

Washington DOH, 1995. School Indoor Air Quality Best Management Practices Manual. Washington State Department of Health, Community Environmental Health Programs, Olympia.

Wasson SJ, Guo Z, McBrian JA, and Beach LO, 2002. Lead in candle emissions. *Sci Total Environ* **296**: 159-174.

Weiss KB, and Sullivan SD, 2001. The health economics of asthma and rhinitis: I, assessing the economic impact. *J Allergy Clin Immunol* **107(1)**: 3-8.

Weschler CJ, and Shields HC, 1996. Production of the hydroxyl radical in indoor air. *Environmental Science & Technology* **30**: 3250-3258.

Weschler CJ, 2000. Ozone in indoor environments: concentration and chemistry. *Indoor Air.* **10(4)**: 269-88.

Weschler CJ, 2004. Chemical reactions among indoor pollutants: what we've learned in the new millennium. *Indoor Air* **14 (Supplement 7):** 184-194.

Weschler CJ and Shields HC, 1994. Indoor chemistry involving O_3, NO, and NO_2 as evidenced by 14 months of measurements at a site in southern California. *Environ. Sci. Technol.* **28**: 2120-2132.

Weschler CJ, and Shields HC, 1996. Production of the hydroxyl radical in indoor air. *Environmental Science and Technology* **30:** 3250-3258.

Weschler CJ, and Shields HC, 1997. Potential reactions among indoor pollutants. *Atmos Environ* **31(21)**: 3487-3495.

Weschler CJ, and Shields HC, 1999. Indoor ozone/terpene reactions as a source of indoor particles. *Atmos Environ* **33**: 2301-2312.

Weschler CJ, Shields HC, and Naik DV, 1989. Indoor ozone exposures. *J Air Pollut Control Assoc* **39(12)**: 1562-8.

Weschler, CJ, Shields HC, and Naik DV, 1994. Indoor chemistry involving O3, NO, and NO_2 as evidenced by 14 months of measurements at a site in Southern California. *Environ Sci Technol* **28**: 2120-2132.

Weschler, CJ, Hodgson, AT, and Wooley, JD, 1992. Indoor chemistry: ozone, volatile organic compounds and carpets. *Environ Sci Technol* **26**: 2371-2377.

Weschler CJ, 2004. Chemical reactions among indoor air pollutants: what we've learned in the new millennium. *Indoor Air* **14**: Suppl 7:184-94.

Whitehouse A, 2000. Asbestos-related disease in the community of Libby. Presented at the Libby Asbestos Exposure Scientific Council Meeting, 22 February 2000, Cincinnati, OH.

Whitmore RW, Immerman FW, Camann DE, Bond AE, Lewis RG, and Schaum JL, 1994. Non-occupational exposures to pesticides for residents of two U.S. cities. *Arch Environ Contam Toxicol* **26**(1): 47-59.

Whitmore RW, Clayton A, Phillips M, Akland G, 2003. California Portable Classrooms Study, Final Report. 3 Vols. Prepared by Research Triangle Institute. Final Report to ARB for Contract No. 00-317. : http://www.arb.ca.gov/research/indoor/pcs/pcs-fr/pcs-fr.htm.

Whittemore AS, and Korn EL, 1980. Asthma and air pollution in the Los Angeles area. *Am J Public Health* **70(7)**: 687-696.

WHO (World Health Organization), 1990. Environmental Health Criteria 101. Methylmercury. Available at http://www.inchem.org/documents/ehc/ehc/ehc101.htm, December 29, 2003.

World Health Organization's (WHO), 2000. Air Quality Guidelines for Europe. WHO Regional Publications, European Series, No. 91. Copenhagen, Denmark. http://www.euro.who.int/air/Activities/20020620_1.

WHO (World Health Organization), 2002. The World Health Report 2002, Reducing Risks, Promoting Healthy Life, World Health Organization, Geneva, Switzerland.

Wiglusz R, Igielska B, Sitko E, Nikel G, Jamuszkiewicz I, 1998. Emission of volatile organic compounds (VOCs) from PVC flooring coverings. *Bull Inst Marit Trop Med* **49**: 101-107.

Wiley JA, Robinson JP, Cheng YT, Piazza T, Stork L, and Pladsen K, 1991a. Study of Children's Activity Patterns. Final report, ARB Contract No. A733-149.

Wiley JA, Robinson JP, Piazza T, Garrett K, Cirksena K, and Martin G, 1991b. Activity Patterns of California Residents. Final report, ARB Contract No. A6-177-33.

Wilkins CK, Clausen PA, Wolkoff P, Larsen ST, Hammer M, Larsen K, Hansen V, and Nielsen GD, 2001. Formation of strong airway irritants in mixtures of isoprene/ozone and isoprene/ozone/nitrogen dioxide. *Environ Health Perspect* **109(9)**: 937-941.

Williams R, Suggs J, Rea A, Leovic K, Vette A, Croghan C, Sheldon L, Rodes C, Thornburg J, Ejire A, Herbst M, and Sanders Jr. W, 2003. The Research Triangle Park particulate matter panel study: modeling ambient source contribution to personal and residential PM mass contributions. *Atmospheric Environment* **37**: 5365-5378.

Williams R, Suggs J, Evans G, Creason J, Kwok R, Rodes C, Lawless P, and Sheldon L, 2000a. Indoor, outdoor, and personal exposure monitoring of particulate air pollution: The Baltimore elderly epidemiology-exposure pilot study. *Atmos Environ* **34**: 4193-4204.

Williams R, Suggs J, Evans G, Creason J, Kwok R, Rodes C, Lawless P, and Sheldon L, 2000b. The 1998 Baltimore particulate matter epidemiology-exposure study: part 1. Comparison of ambient, residential outdoor, indoor and apartment particulate matter monitoring. *J Expo Anal Environ Epidemiol* **46(10)**: 518-532.

Williams R, Creason J, Zweidinger R, Watts, R, Sheldon L, and Shy C, 2000c. The 1998 Baltimore particulate matter epidemiology-exposure study: part 2. Personal exposure assessment associated with an elderly study population. *J Expo Anal Environ Epidemiol* **10**: 533-543.

Wilson AL, Colome SD, Baker PE, and Becker EW, 1986. Residential Indoor Air Quality Characterization Study of Nitrogen Dioxide, Phase I, vol. 2, final report for Southern California Gas Company.

Wilson AL, Colome SD, and Tian Y, 1993. California residential indoor air quality study, volume 1: Methodology and descriptive statistics, prepared for the Gas Research Institute, Pacific Gas and Electric Company, San Diego Gas and Electric Company, and Southern California Gas Company.

Wolkoff, P, 1995. Volatile organic compounds – sources, measurements, emissions, and the impaact on indoor air quality. *Indoor Air (Suppl. 3/95)* 9-73.

Wolkoff P, Wilkins CK, Clausen PA, and Larsen K, 1993. Comparison of volatile organic compounds from processed paper and toners from office copiers and printers: methods, emission rates, and modeled concentrations. *Indoor Air* **(3)**: 113-123.

Wolkoff P, Clausen PA, Wilkins CK, and Nielsen GD, 2000. Formation of strong airway irritants in terpene/ozone mixtures. *Indoor Air* **10**:82-91.

Wolkoff P, and Nielsen GD, 2001. Organic compounds in indoor air – their relevance for perceived indoor air quality? *Atmos Environ* **35**: 4407-4417.

Woodruff TJ, Grillo J, and Schoendorf KC, 1997. The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. *Environ Health Perspect* **105**: 608-612.

WRPPN (Western Regional Pollution Prevention Network), 2003. Janitorial Products Prevention Project. Reno, NV. http://www.wrppn.org/Janitorial/jp4.cfm.

Wynder EL, and Hoffman D, 1964. Polycyclic aromatic hydrocarbons. Advances in Cancer Research **8** U.S. Environmental Protection Agency (U.S. EPA), 1987: 312-322.

Wyon DP, 2004. The effects of indoor air quality on performance and productivity. *Indoor Air* **14(s7)**: 92-101.

Yu C, and Crump D, 1998. A review of the Emission of VOCs from Polymeric materials used in buildings. *Building and Environment* **33(6)**: 357-374.

Zanobetti A, and Schwartz J, 2003. Airborne particles and hospital admissions for heart and lung disease in Health Effects Institute Special Report: Revised Analyses of Time-Series Studies of Air Pollution and Health.

Zartarian VG, Ferguson AC, and Leckie JO, 1998. Quantified mouthing activity data from a four-child pilot field study. *J Expo Anal Environ Epidemiol* **8(4)**: 543-553.

Zartarian VG, and Leckie JO, 1998. Feature Article—Dermal Exposure: The Missing Link. *Environ Sci Technol* **3(3)**: 134A-137A.

Zhou JY, Liu KS, and Waldman JM, 1998. Survey of indoor radon concentrations in California elementary schools. California Department of Health Services, Environmental Health Laboratory Branch. *Report EHLB/R-400*, May 1998. Available from: http://www.cal-iaq.org/RadonInCalSchools99.html.

Zhu J, Cao X, and Beauchamp R, 2001. Determination of 2-butoxyethanol emissions from selected consumer products and its application in assessment of inhalation exposure associated with cleaning tasks. *Environ Int* **26**: 589-597.

Zipprich JL, Harris SA, Fox JC, and Borzelleca JF, 2002. An analysis of factors that influence personal exposure to nitrogen oxides in residents of Richmond, Virginia. *J Expo Anal Environ Epidemiol* **12**: 273-285.

Zock JP, Jarvis D, Luczynska C, Dunyer J, and Burney P, 2002. Housing characteristics, reported mold exposure, and asthma in the European Community Respiratory Health Survey. *Journal of Allergy and Clinical Immunology* **110**: 285-292.

10. GLOSSARY

TERM	DEFINITION
Active/Passive Sampling	Active sampling depends on a mechanical process like pumping to collect the sample at a known rate; this is used for VOC and aldehyde sample collection. Passive sampling involves non-mechanical processes, usually diffusion, in which the air is sampled at whatever rate it passes across a badge surface or tube opening.
Air Changes per Hour	ACH, the volume of air moved in one hour. One air change per hour in a room, home, or building means that the equivalent of the volume of air in that space will be replaced in one hour.
Air Cleaners	These are devices designed to remove pollutants from a room. Air cleaners can be portable, or part of a central air system. Air cleaners can be mechanical, employing a filter to remove pollutants, or electronic using a small electrical charge to collect particles from air pulled through a device.
Air Fresheners	These devices are promoted to neutralize odors rather than remove pollutants. Products often emit a fragrance which diffuses into the air. Some styles can spray fragrances and be programmed for fragrance dispersion.
Air Flow Rate	The rate at which air moves into a space. Expressed in units of air changes per hour or cubic feet per minute.
Air Handling Unit	HVAC (heating, ventilation and air conditioning) unit. Refers to equipment that includes a blower or fan, heating and/or cooling coils, and related equipment such as controls, condensate drain pans, and air filters. Does not include ductwork, registers, or grilles, or boilers and chillers.
Allergen	A chemical or biological substance (e.g., pollen, animal dander, or house dust mite proteins) that induces an allergic response, characterized by hypersensitivity.
Ambient Air Quality Standards (AAQS)	State (ARB) and federal (U.S. EPA) enforceable regulations designed to protect the public from the harmful effects of traditional pollutants in outdoor air.
Asthma	A chronic disease of lung tissue which involves inflamed airways, breathing difficulty, and an increased sensitivity to allergens and contaminants in the air.
Biological Contaminants	Agents derived from or that are living organisms (e.g., viruses, bacteria, fungi, and mammal, arthropod, and bird antigens) that can be inhaled and can cause many types of health effects

	including allergic reactions, respiratory disorders, hypersensitivity diseases, and infectious diseases. Also referred to as biological agents.
Comfort Measures	Factors that determine human perception of thermal comfort, including temperature, relative humidity, and draft
Commissioning	Testing the important building systems before occupancy to ensure that they operate the way the designers expect and that they serve the needs of the building occupants. Systems that can be tested include HVAC, electrical, energy management, plumbing, etc.
Fungi	A group of organisms that lack chlorophyll, including molds, mildews, yeasts, mushrooms.
Integrated Pest Management (IPM)	A pest management strategy that focuses on long-term prevention or suppression of pest problems through a combination of techniques such as monitoring for pest presence and establishing treatment threshold levels, using non-chemical practices to make the habitat less conducive to pest development, improving sanitation, and employing mechanical and physical controls. Pesticides that pose the least possible hazard and are effective in a manner that minimizes risks to people, property, and the environment, are used only after careful monitoring indicates they are needed according to pre-established guidelines and treatment thresholds.
Micron	A unit of length equal to one millionth of a meter; a micrometer.
Microorganism	A microscopic organism, usually a bacterium, fungus, or protozoan.
Natural Ventilation	The movement of outdoor air into a space through intentionally provided openings, such as windows and doors, or through non-mechanical ventilators, by wind, air pressure differences, or other natural, non-mechanical means.
Permissible Exposure Limits (PELs)	Enforceable pollutant exposure limits determined by OSHA that are designed to protect healthy adult workers in industrial environments from adverse health effects associated with pollutant exposure. None of these limits are targeted toward protecting children.
Pesticides	A pesticide is any substance or mixture of substances intended to prevent, destroy, repel, or mitigate any pest. Though often misunderstood to refer only to insecticides, the term pesticide also applies to herbicides, fungicides, disinfectants, and antimicrobials. Under U. S. law, a pesticide is also any substance or mixture of substances intended for use as a plant regulator, defoliant, or desiccant.

Polycyclic Aromatic Hydrocarbons (PAHs)	A class of stable organic molecules comprised of only carbon and hydrogen. They are a common product of combustion from automobiles, airplanes, woodburning, cigarettes, and some types of cooking. Many of these molecules are highly carcinogenic and very common.
Portable Classrooms	Classrooms that are designed and constructed to be moveable and transportable over public streets, also known as temporary or relocatable classrooms.
Quality Control (QC)	Internal checks on the operation of sample collection and/or sample analysis. Methods for determining the operation include blanks, spiked samples, flow checks, and duplicate samples. QC measures can be used to determine accuracy, bias, and precision of the data reported.
Real-time Monitoring	This type of environmental measurement gives instantaneous information at the point of sampling; measurements are recorded as often as every minute, every second, or in fractions of a second.
Reference Exposure Level (REL)	The concentration level at or below which no adverse health effects are anticipated for a specified exposure duration. RELs are based on the most relevant, adverse health effect reported in the medical and toxicological literature for the population group known to be most sensitive to the chemical. RELs are designed to protect the most sensitive individuals in the population by the inclusion of margins of safety. Since margins of safety are incorporated to address data gaps and uncertainties, exceeding the REL does not automatically indicate an adverse health impact will occur. OEHHA provides acute (1-hour) and chronic (lifetime, non-cancer), RELs for a number of chemicals, and has developed an 8-hour "indoor" REL for formaldehyde.
Relative Humidity	The measure of moisture in the atmosphere, expressed as a percent of the maximum moisture the air can hold at a given temperature.
Return Air	Air removed from a space by the HVAC system to be recirculated or exhausted.
Sick Building Syndrome	A set of symptoms (including headache, fatigue, and eye irritation) typically affecting workers in modern airtight office buildings, believed to be caused by indoor pollutants (such as formaldehyde fumes or microorganisms).
Supply Air	Air delivered to the conditioned space by the HVAC system and used for ventilation, heating, cooling, humidification, or dehumidification. It is usually a combination of outdoor air and return air.

Traditional Classrooms	Classrooms in permanent, site-built school buildings.
Variable Air Volume System	Air handling system that conditions the air to a temperature using a varying amount of outside airflow based essentially on the outdoor temperature.
Ventilation	The process of intentionally supplying and removing air by natural or mechanical means to and from any space.
Volatile Organic Compounds (VOCs)	Compounds that evaporate quickly from the many housekeeping, maintenance, and building products made with organic chemicals. These compounds are released from products that are being used and that are in storage. Many are carcinogenic, neurotoxins, or mucous membrane irritants.

APPENDIX I

HEALTH AND SAFETY CODE § 39930

The legislative mandate for the report to the legislature on Indoor Air Quality is contained in the Health & Safety Code amendment contained in Section 2 of Assembly Bill 1173 (Keeley, 2002):

39930. (a) The State board shall, not later than January 1, 2004, in consultation with the State Department of health Services, the Office of Environmental Health Hazard Assessment, the State Energy Resources Conservation and Development Commission, any other state agency the state board determines is appropriate, affected indoor emissions sources, and interested members of the public, provide a report to the Legislature summarizing all of the following:

(1) The best scientific information available including, but not limited to, the most recent empirical data, on indoor air pollution including, but not limited to, air contaminants that have been identified as toxic air contaminants pursuant to Sections 39655, 39657, or 39660, or air contaminants for which the state board has adopted ambient air quality standards.

(2) The potential adverse effects of indoor air pollution exposure on public health in the state, including, but not limited to, vulnerable populations, including, but not limited to, elderly persons, infants, and children, based upon the information described in paragraph (1).

(3) Readily available information about the effects of existing regulations and current industry practices in mitigating those exposures.

(4) A listing that references work performed by other state or federal entities regarding biological and radiological substances, including a summary of activities conducted by the State Department of health Services pursuant to Chapter 18 (commencing with Section 26100) of Division 20.

(b) The report described in subdivision (a) shall include all of the following:

(1) A list of indoor air pollutants that are described in the summaries provided pursuant to paragraphs (1) and (4) of subdivision (a).

(2) A list of indoor air pollutants, as defined in Section 39013, ranked in groups designated as high, medium, and lower priorities, that the state board has determined, based upon empirical data or other scientific information, are likely to have the most significant adverse impacts on human health through exposures in schools, non-industrial workplaces, homes, and other indoor locations, and the probable source categories for these pollutants.

(3) An analysis of the indoor emissions, indoor exposures, and potential health effects from the indoor source categories described in paragraph (1), and options for mitigating those health effects in schools, non-industrial workplaces, homes, and other indoor locations, including, but not limited to, a discussion of the feasibility and public health effects of implementing each option.

(4) A description of options for schools and school districts to improve indoor air quality in public schools. The state board shall develop these options in consultation with representatives from school district facility departments, school district maintenance departments, and statewide educational organizations.

(c) (1) The state board shall enter into an agreement with the National Academy of Sciences, the University of California, the California State University, or a similar institution of higher learning that has scientific expertise, any combination of those entities, or with a scientist or group of scientists of comparable stature and qualifications that is recommended by the president of the University of California, to conduct an external scientific peer review of the scientific basis for the report described in subdivision (a).

(2) The state board may not submit the report to the Legislature until all of the following conditions are met:

(A) The draft report is submitted to the external scientific peer review entity described in paragraph (1) for evaluation.

(B) The external scientific peer review entity, within the timeframe agreed upon by the board and the external scientific peer review entity, prepares written comments that contain an evaluation of the scientific basis for the draft report. If the state board disagrees with any aspect of the findings of the external scientific peer review entity, the state board shall include as part of the final report, an explanation of its basis for arriving at the determination, including, but not limited to, the reasons that the state board determined that the report was based on sound scientific knowledge, methods, and practices.

(d) The state board shall present and review the content of the report described in subdivision (a) at a public meeting prior to providing the report to the Legislature.

APPENDIX II
Explanation of Indoor Cancer Risk Estimates

February, 2005

Summary: The toxic air contaminant (TAC)-related cancer risk estimates presented in this report are based on results of the *California Comparative Risk Project (CCRP), Final Report*, May 1994, for indoor indicator pollutants–excluding radon, environmental tobacco smoke (ETS), and asbestos–with adjustments (downward) based on changes in current exposure levels of formaldehyde relative to those from studies used in the CCRP assessment. This information is the best information available for California, and the indoor air concentrations on which these estimates are based are supported by more recent studies from other states. Background information on the estimates developed in the CCRP is discussed below, followed by a detailed discussion of how the cancer risk estimates for this report (AB1173) were developed.

BACKGROUND: the California Comparative Risk Project (report available at http://www.oehha.ca.gov/multimedia/comprisk.html.

- **The CCRP estimated about 268 excess annual cancer cases from indoor residential and consumer product sources** for the 10 indicator chemicals included in the estimate. The majority of that risk was from formaldehyde and p-dichlorobenzene. The excess annual cancer cases were taken from the table "Residential and Consumer Product Sources: II. Human Health Risk Assessment Results for Indicator Chemicals" from the CCRP. The 10 chemicals are:

formaldehyde	*p*-dichlorobenzene
benzene	benzo(*a*)pyrene
1,3-butadiene	chloroform
di-2-ethylhexylphthalate (DEHP)	styrene
tetrachloroethylene (perchloroethylene)	trichloroethylene

- **The risk is that which was attributable only to the emissions from the indoor sources.** In the CCRP Project, the outdoor contribution to the indoor concentrations was subtracted from the indoor levels, with the remainder attributable to indoor sources.

- **The cancer risks estimated are ANNUAL excess cancer cases.** This is somewhat different from the expression we typically use for outdoor air, expressed as cancer risk from 70 years of exposure per million people exposed, but the two can be roughly converted for comparison (see below).

- **The risk from residential and consumer products was ranked in the High Risk category with a high level of confidence** based on the extensive contribution to both cancer and non-cancer risks, the widespread exposure throughout the population, and the consistency of monitoring results across many studies.

- Like other source and media categories in the CCRP, the estimates developed for the Indoor (residential and consumer product) category do not include risk posed by other known carcinogens that occur indoors from indoor sources. **Thus, the cancer risk**

estimated for the 10 indicator chemicals is likely to be an underestimate of the actual cancer risk from indoor sources other than ETS, radon, and asbestos.

- **Notes regarding the CCRP estimates:**

  1. They are based on exposure distributions developed from the best available studies at the time, with greater weight given to California studies. Generally, 3-4 California studies of large numbers of homes in both northern and southern California (totaling about 600-800 homes) were available; these covered a range of seasons, income levels, etc. and were randomly selected using census tract information. For some pollutants, measurements from public buildings, offices, etc. in other studies were also available.

  2. Indoor concentrations tend to be log-normally distributed, meaning a small but decreasing portion of the population experiences particularly high exposures well above the mean. In the CCRP, indoor concentration distributions for a few pollutants appeared to be potentially bi-modal (having a group in the population with especially high indoor concentrations because their house contains large sources of a given pollutant, such as mothballs/*p*-dichlorobenzene...e.g., either the household uses mothballs, or they do not, and if they do, their levels are quite high and that group in the population has distinctly greater indoor concentrations than others). Because of this, concentration distributions (rather than means) were used to develop exposure estimates to achieve more accurate risk estimates.

  3. Although distributions were used to estimate risk, the resulting average individual risk was used to estimate annual cancer cases, and thus these may be conservative estimates, since the average does not necessarily fully capture those at very high risk. Additionally, only the population with an exposure level above the one in ten thousand risk level was included in the risk estimation; thus, those with lower cancer risks were not counted, effectively reducing the total count relative to the population estimate that would be derived if those with lower cancer risks had also been included.

CURRENT ESTIMATE for AB 1173 report:

- We reviewed the estimates for the 10 chemicals and performed a quick literature search to identify more recent data that might indicate that an adjustment is needed to better reflect current indoor exposure levels.

- Formaldehyde is the only one of the 10 indicator chemicals for which there are sufficient new data to develop a more current exposure distribution. Recent emissions studies (e.g., Kelly *et al.*, 1999; Hodgson *et al.*, 2000; Hodgson *et al.*, 2002; Hodgson, 1999) show that indoor formaldehyde emissions from many materials and products have decreased by an average of 49% since the earlier Pickrell emissions study (1983). These and other studies indicate that indoor exposure concentrations have decreased by about 50% since the early to mid-1980s, when the majority of the studies used in the CCRP project were conducted. This was not surprising, since several industry initiatives and some known product changes had occurred since the mid-80s. **Thus, for our new estimate, we assumed that the current formaldehyde risk would be about half of that estimated in the CCRP Project, or about 62 excess cancer cases per year.**

- Recent studies show that levels of some of the other indicator indoor pollutants attributable to indoor sources, such as chloroform and styrene, may have increased in recent years, but there are no new indoor California studies in the last decade that would document this, and information obtained regarding changes in the known sources is mixed. For others, such as benzene, there is reason to believe that indoor levels from indoor sources have decreased somewhat since the earlier studies, due to product composition changes and reduced indoor smoking, but there are no readily available data to support a revised calculation.

  In the CCRP, DEHP was included in the list of chemicals used to develop the cancer estimates. However, IARC recently concluded that DEHP is not a human carcinogen, and downgraded its classification to Group 3 (not classified as a human carcinogen, due to inadequate evidence for human carcinogenicity). In the CCRP, DEHP accounted for one cancer case, so the total cancers estimated from CCRP was decreased by one for the current estimate. Thus, the current total risk posed by the indicators other than formaldehyde and DEHP remains a reasonable estimate of cancer risk.

- **Our current estimate is thus calculated as follows:**
  268 minus 63 (1/2 the previous formaldehyde estimate, and minus one for DEHP) = 205 excess cancer cases per year, times 34/30 (or 1.13) to adjust the original CCRP estimates to the year 2000 California population of 34 million, for a total of 232 excess cancer cases per year due to emissions from indoor sources of the chemicals. This rounds to 230. However, 230 is likely an underestimate due to: a) the conservative nature of the original estimate; b) the fact that there are other indoor carcinogens that are known but not included in the estimate (see bullet below); and c) the uncertainty of the risk estimation process, which is best addressed by using a range where possible. **Thus, at least 230 excess cancers per year are estimated.**

- **There are a number of additional carcinogens known to be emitted from indoor sources that were not included in the indicator** chemicals list for the CCRP Project due to a lack of sufficient indoor data to estimate an exposure level. For example, other PAHs, acetaldehyde and other aldehydes, and asbestos are carcinogenic and have been measured indoors and as emissions from products. Persistent chemicals such as PCBs have been found in house dust, and various toxic metals have been measured at higher levels in both indoor air and house dust. However, the data are not sufficient to estimate population exposure. Others like acrolein are just beginning to be studied in the indoor air. **Thus, the adjusted estimate above is assuredly an underestimate of the actual cancer risk posed by toxic chemicals emitted or produced by indoor sources.**

Comparison to Outdoor Risk Levels (Fig. 2.1 in the report).

- Using the excess cancer cases per million per 70 years in Table 7 of ARB's October 2000 diesel risk reduction plan and the year 2000 California population of 34 million, **current estimated ANNUAL excess cancer cases from diesel exhaust particles total 262** (540/million X 34 / 70), or about 260 excess cancers per year. This figure does not account for recently implemented or planned regulations, but those changes would have little impact on the 70 year lifetime exposures of most adults in the population, and thus this estimate remains reasonable.

- Using the same table, **the non-diesel risk from other outdoor sources is calculated to total 106** excess cancers (218/million X 34 / 70= 106), or about 110 excess cancers per year.

- The total excess cancer cases per year from different sources of air pollutants are thus estimated to be:

Residential and Consumer Product Sources:	**230 /year**
Diesel exhaust PM10:	**260 /year**
Other outdoor sources:	**110 /year**

Figure 2.1 in the AB 1173 report reflect these numbers.

Note: OEHHA has indicated previously that conversion of the outdoor risk estimates to annual cancer cases to allow comparison of the CCRP indoor annual excess cancer estimates is acceptable for this type of general comparison.

RADON, ETS, AND ASBESTOS

Radon and ETS: the exposure and risk estimates have been updated and are included in the text.

Asbestos has not been widely measured in California indoor environments, and there are many measurement difficulties for this set of fibers. There are insufficient data on which to base an indoor asbestos risk estimate (from indoor sources) at this time, just as there was at the time of the CCRP project.

APPENDIX III
Background for Estimates of Indoor Formaldehyde Concentrations

February, 2005

Figure 2.4 is intended to give a general indication of current estimates of indoor formaldehyde levels in several California environments, and to illustrate relative levels among those environments. Data on formaldehyde levels in homes, schools, and offices were obtained from several information sources. A description of the sources used for each category follows. Please note that these estimates were derived only for the illustration provided in Figure 2.4: they were not used for the cancer risk estimates discussed in this report. As discussed in Appendix II, the cancer risk estimates for formaldehyde were based on the estimates from the 1994 California Comparative Risk Project and reduced based on a more recent emissions study.

Manufactured homes
Estimated Average: 37 ppb, Maximum: 227 ppb

Formaldehyde levels in manufactured homes are based on measurements made in California manufactured homes during the early 1980s, reduced by a factor representative of the reduction in formaldehyde emissions in new composite wood products since that time. The resultant value is consistent with recent limited measurements in manufactured homes.

The California Department of Health Services measured formaldehyde levels in approximately 600 mobile homes in 1984 and 1985. Investigators obtained integrated one-week measurements for approximately 600 mobile homes. The geometric mean formaldehyde concentration measured in the summer was 72 ppb (arithmetic mean 91 ppb), and 78 ppb (arithmetic mean 91 ppb) in the winter (Sexton *et al.*, 1985). The maximum value was 464 ppb measured in the summer.

Formaldehyde emissions from new composite wood products are lower today than they were in the early 1980's due to changes in manufacturing procedures. A comparison of emission rates from Pickrell (1983) and Kelly (1999) indicate formaldehyde emission rates from these products have decreased an average of about 49% over the last 20 years. Comparison of data from the two investigators indicate particleboard emissions are 92% of what they were in 1983, interior plywood emissions are 15% of 1983 values, and paneling emissions are 39% of 1983 emissions. An unweighted average of these reductions in emissions yields a gross average estimate that emissions today are 49% of what they were in 1983.

The estimate of an average formaldehyde concentration of 37 ppb in manufactured homes is based on the average of the winter and summer geometric means determined by Sexton, then reduced by 49% to reflect changes in manufacturing practices. The maximum value measured by Sexton (464 ppb) was also reduced by 49% to reflect manufacturing changes.

Hodgson *et al.* (2002) measured formaldehyde levels inside four new manufactured homes in humid climates of the southeastern U.S. The homes were furnished but not occupied (sales models) and had a geometric mean formaldehyde concentration of 34 ppb. Over extended time periods, this level would be reduced as the emissions from building materials decline. However, human activities tend to elevate formaldehyde concentrations due to use of combustion appliances and products that emit formaldehyde, which would offset the decline from building materials. Thus, the level measured by Hodgson is consistent with the concentration estimated above.

Sexton K, Liu K, and Petreas M. (1986), Formaldehyde Concentrations Inside Private Residences: A Mail-Out Approach to Indoor Air Monitoring, *JAPCA* **36**: 698-704.

Pickrell J, Mokler B, Griffis L, Hobbs C, and Bathija A. (1983), Formaldehyde Release Rate Coefficients from Selected Consumer Products, *Environmental Science and Technology* **17(12)**: 753-757.

Kelly T, Smith DL, and Satola J. (1999). Emission Rates of Formaldehyde from Materials and Consumer Products Found in California Homes, *Environmental Science and Technology* **33(1)**: 81-88.

Hodgson AT, Rudd AF, Beal D, and Chandra S. (2000) Volatile organic compound concentrations and emission rates in new manufactured and site-built houses, *Indoor Air* **10**: 178-192.

Classrooms (Inside)
Estimated Average: 18 ppb, Maximum: 110 ppb

The California Portable Classrooms Study (PCS) data from 2001 and 2002 were used to estimate school-year average and maximum concentrations of formaldehyde in California's K-12 classrooms. This is a large, representative statewide data set of formaldehyde measurements obtained across four seasons using both active and passive sampling methods.

School-Year Average Concentration

Because indoor formaldehyde levels increase with increased temperature and humidity, we combined PCS formaldehyde data from Phase I (warmer seasons) and Phase II (cooler seasons) to estimate school-year average concentrations. We excluded most of July and all of August in estimating the school-year average, although 22 % of California's K-12 students attend year-round schools or summer school (CDE, 2002). Therefore, our estimate of school-year average concentration for formaldehyde is likely a conservative estimate for the state.

We used field study data from the PCS Phase II to estimate classroom formaldehyde levels in the 6 months of cooler weather, October – March. In Phase II, formaldehyde was measured using the DNPH method with active sampling over 6-8 classroom hours from October 2001 to early March 2002. The mean concentration for this period was 13 ppb, and the geometric mean was 12 ppb. Monthly breakdowns of Phase II formaldehyde concentrations were not readily available, but they would be limited to only two time periods because of the limited sample size (201 classrooms) in Phase II. Phase II data provide reasonably accurate estimates of classroom concentrations of formaldehyde for the fall and winter seasons when monitoring occurred.

Mail survey data from the PCS Phase I were used to estimate classroom formaldehyde levels in the 6 months of warmer weather (April through September). In Phase I, formaldehyde was measured using a passive monitor deployed for 7-10 days, including nights and weekends, in April to early July 2001. The mean concentration for this period was 27 ppb (geometric mean of 22 ppb).

As expected, both the means and 95th percentile concentrations were notably higher in Phase I compared to Phase II (ARB-DHS, 2003). The higher indoor levels in Phase I were expected because the Phase I sampling was conducted during warmer weather when indoor

formaldehyde levels are usually higher, and because the sample size was substantially larger, increasing the probability of including classrooms with more extreme levels in the sample. Phase I also included nights and one or two weekends in the sampling period, during which the classrooms were probably not ventilated, which could result in higher formaldehyde levels. Also, in Phase II sampling, technicians operated the ventilation system to make flow measurements, which might have reduced formaldehyde levels relative to what they might have been under normal operation conditions.

Phase I measurements confirm that there was a positive bias associated with the weekend sampling included in the 7 to 10 day measurement. The weekend bias was determined by comparing data from classrooms with >25% of the sample days on weekends (2 weekends) vs. those with < 25% weekend days (1 weekend). The first group had mean formaldehyde levels of 30 ppb vs. 25 ppb in the second group, or a difference of 5 ppb due to an additional weekend of sampling in these seasons. These results are from analyses using weighted data.

To estimate the overall effect of the weekend bias on the Phase I mean, we calculated a weighted average of the weekend bias. About half of the sample (52%) included one weekend, and about half of the sample (48%) included two weekends. The weighted average was calculated as follows:

(0.52 of sample x 1 x weekend bias) + (0.48 of sample x 2 x weekend bias)
= 1.48 x weekend bias = 1.48 x 5 ppb = 7 ppb positive bias on Phase I mean

Therefore, the positive bias in the Phase I mean for estimating the mean schoolday (weekday) concentrations was estimated to be 7 ppb. Additional positive bias may have occurred due to sampling overnight, but data are not available to quantify the magnitude of this bias. However, the magnitude of the overnight bias is expected to be much less than that for the weekend bias because the nighttime period is usually much cooler, which would result in reduced formaldehyde concentrations relative to weekend concentrations.

The Phase I data were also adjusted for the lack of data for September. To estimate formaldehyde levels during the warmer season (April – September), the Phase I data were extrapolated to estimate what concentrations would likely be in September, and the 4 month average was then calculated. The monthly means for April, May, and June-early July were 18, 29, and 36 ppb, respectively. July and August were considered to be mostly a vacation period for schools. The monthly mean for September was assumed to be the same as that for June – early July. The average for April, May, June, and September was calculated, and the weekend bias then subtracted, as follows:

(18 ppb + 29 ppb + 36 ppb + 36 ppb) / 4 - (7 ppb for weekend bias in means)
= 30 ppb – 7 ppb weekend bias
= 23 ppb for warmer season, excluding summer vacation

To estimate the school-year average concentrations of classroom formaldehyde in California, we then averaged the estimated concentrations for the warmer seasons and the measured concentration for the cooler season:

(23 ppb in warmer season + 13 ppb in cooler season) / 2
= 18 ppb school-year average estimate

In conclusion, the school-year average concentration of formaldehyde in California K-12 classrooms was estimated to be 18 ppb, after adjustment for weekend bias and the lack of September data. This value is slightly lower than the unadjusted average of the Phase I and Phase II data (20 ppb).

School-Year Maximum Concentration

To estimate school-year maximum concentrations of formaldehyde in California's K-12 classrooms, data from PCS Phase I, PCS Phase II, and case studies in California were considered. The maximum formaldehyde level measured among 199 classrooms in PCS Phase II was 71 ppb (Whitmore, 2003). Because these measurements were made in the fall and winter, and because extreme values are not easily measured with such a small sample size, this value assuredly underestimates the maximum formaldehyde concentration.

In the Phase I mail survey during the spring and early summer, the maximum formaldehyde level measured among 911 classrooms was 138 ppb (Whitmore *et al.*, 2003). However, this value overestimates the maximum because the passive sampler measurement for 7-10 days includes weekends and nights. The highest percentile values reported with statistical confidence were the 95th percentile values for portable classrooms, so these values were used to estimate the weekend bias. The portables subsample with 2 weekends had a 95th percentile of 78 ppb, vs. 62 ppb for the portables subsample with one weekend, a difference of 16 ppb. Using the same approach used above to adjust the average concentrations for weekend bias, a weighted average of 24 ppb is estimated for the weekend bias in the 95th percentile (1.48 x 16 ppb). Subtracting this bias from the maximum value of 138 ppb yields an estimated maximum of 114 ppb. This value may be an overestimate of the maximum concentration because the bias due to overnight sampling is not included. Rounding down, the statewide maximum is estimated to be about 110 ppb.

Although higher than the 71 ppb maximum obtained in the Phase II field study, this value is supported by data from case studies of California schools, which include a maximum of 98 ppb for 6-8 hours. These studies employed active sampling using the DNPH method measurements in 90 classrooms in August, September, or October of 1999 and 2000. Selected classrooms in five school districts, including the Saugus and Beverly Hills districts, were examined.

Because each of these data sets represents a small sample size relative to the total population of classrooms in California, one would not expect the classrooms with the very highest formaldehyde levels to be included in the sample. Samples tend to reflect the mean but not the extreme values existent in the actual population being studied. Thus, the statewide maximum is estimated to be at least 110 ppb, and probably higher.

For more information on the Portable Classroom study, please see:
ARB and DHS, (November 2003) Environmental Health Conditions in California's Portable Classrooms, Report to the California Legislature.
http://www.arb.ca.gov/research/indoor/pcs/leg_rpt/pcs_r2l.pdf, p. 53 et seq.

California Department of Education (CDE). Year-Round Education, 2002-03 Statistics. Based on 2002 CBEDS. Sacramento, CA. http://www.cde.ca.gov/facilities/yearround/yrstat02.htm.

Whitmore R, Clayton A, Phillips M, and Akland G. (2003). California Portable Classrooms Study: Phase I-Mailed Survey and Phase II-Main Study, Final report to ARB for Contract no, 00-317.

Whitmore, R, 2003. Personal communication, February 24. Research Triangle Institute, Research Triangle Park, NC.

Conventional Homes
Estimated Average: 14 ppb, Maximum: 232 ppb.

Formaldehyde levels measured in the National Human Exposure Assessment Survey (NHEXAS) in Arizona and a southern California study were used to estimate current concentrations in Californian homes. The NHEXAS study was conducted from October 1993 through September 1998. Investigators used a probability-based sampling scheme to obtain results that are representative of the entire state of Arizona. Sampling was conducted over different seasons and included 189 homes. Passive sampling tubes with a sodium bisulfite-impregnated disk were used to collect formaldehyde for a 6 - 7 day period. Construction practices, climate, and ventilation practices are assumed to be reasonably similar in California and Arizona. Results from the NHEXAS study indicate the average formaldehyde level for all homes was 17 ppb. The maximum formaldehyde concentration was 331 ppb. Although unusually high, this value is considered a valid result by the authors. They report it is within a factor of 2 of maximum levels measured by other investigators.

Data are also available from a study sponsored by ARB, in which investigators measured formaldehyde levels in approximately 70 homes in limited areas of southern California (Avol et al, 1996). Investigators used the 2,4-dinitrophenylhydrazine method with active monitors. The results are lower than expected for a sample representative of the entire California population of homes for two reasons: sampling was conducted during the summer only with doors and windows open much of the time, and most of the homes were notably older homes, and not new homes. The mean concentration in this study was 9.1 ppb, and the maximum was 31.3 ppb.

Results from both of the above studies were used to estimate indoor concentrations in conventional California homes. The NHEXAS study has great credibility since it used a probability-based sampling scheme for a statewide study conducted in a neighboring state. The southern California study represents the portion of Californians who live in a mild southern California climate in homes that are not new, during under one set of conditions. Results from the two studies are weighted approximately by sample size of the two studies to obtain a current estimate for conventional California homes. The NHEXAS mean and maximum were weighted by a factor of .67, while the California data were weighted by a factor of .33.

Average: 0.67 (17) ppb + 0.33 (9) = 11 + 3 = 14 ppb.
Maximum: 0.67 (331) + 0.33 (31) = 222 + 10 = 232.

Avol, E. (1996), "Residential Microenvironmental and Personal Sampling Project for Exposure Classification", final report to ARB, Contract no. 92-317.

Gordon SM, Callahan PJ, Nishioka MG, Brinkman MC, O'Rourke MK, Lebowitz, MD, Moschandreas DJ. (1999). Residential environmental measurements in the National Human Exposure Assessment Survey (NHEXAS) pilot study in Arizona: preliminary results for pesticides and VOCs, *Journal of Exposure Analysis and Environmental Epidemiology* **9**, 456-470.

Office Buildings
Estimated Average: 13 ppb, Maximum: 26 ppb

The U.S. EPA Building Assessment Survey and Evaluation (BASE) Study examined pollutant levels inside 100 non-problematic office buildings in the U.S. Formaldehyde data from this study has not yet been fully analyzed or published. However, preliminary formaldehyde data were presented in an addendum to a U.S. EPA draft report; "Ranking Risks from Air Toxics Indoors" prepared for Pauline Johnston, U.S. EPA, Indoor Environments Division. Preliminary data available in the addendum were used to estimate formaldehyde levels in office buildings. When analyses of the BASE study data are completed, the final results will be used to represent California office buildings. The value of 26 ppb is likely lower than the maximum.

Source: U.S. EPA Building Assessment Survey and Evaluation (BASE) Study. More information on the U.S. EPA BASE Study can be found at http://www.epa.gov/iaq/largebldgs/index.html.

Outdoor levels
Estimated Average: 3 ppb, Maximum: 15 ppb

Outdoor formaldehyde concentrations are from the air toxics sampling network that was designed to produce a statewide annual average for individual toxic air contaminants. Data from the most recent five years (1998 - 2002) for which data are available were averaged to negate any effects due to global weather influences. Data from the Toxics Network is available at http://www.arb.ca.gov/aqd/toxics/statesubstance.html.

APPENDIX IV

ARB Responses to Comments of the Scientific Peer Review Committee

Report to the California Legislature
Indoor Air Pollution in California
California Air Resources Board
November 2004

The ARB wishes to extend its deep appreciation to the scientific peer review committee for their extensive review of the November 2004 draft Report. The members of the committee included Drs. S. Katharine Hammond, William W. Nazaroff, and Kirk R. Smith, all from the University of California at Berkeley, and Mr. Richard Sextro, from the Lawrence Berkeley National Laboratory. We especially thank Dr. Smith for serving as the Chair of the committee. The committee's comments have improved the Report notably, and will help guide future assessments and actions that may be taken as a result of the report.

COMMENTS:

I. To begin, we commend the California Legislature for commissioning this work. Indoor air quality is an important issue for the health and well being of Californians and the state government can play a significant role in helping to ensure good indoor air quality in both public and private spaces. The topic is not currently well addressed by governments at the national, state, or local level. Consequently, an effort to summarize the state of knowledge and to explore the potential for governmental action is appropriate. California has a proud tradition of leading the country on matters of environmental health, including in the area of indoor air quality.

II. The Air Resources Board has prepared a good draft report. The document assembles a large and diverse body of literature and presents it in a coherent, well-organized, and well-written manner. Particularly impressive are the extensive scope of the pollutant-by-pollutant summaries and the compilation of existing regulations and guidelines.

III. As is generally the case in a scientific peer review, this commentary focuses on aspects of the report that could be improved, rather than discussing the portions that are already in good shape.

IV. In looking over our complete review, below, however, we are concerned that it may be a rather large effort by ARB staff to fully respond. We worry that this would lead to a significant delay in moving forward with efforts in the state to deal with the aspects of indoor air pollution that are well-documented and worthy of action. We recommend, therefore, that the state consider dividing indoor air issues into phases or tiers. In this way, appropriate policy actions can proceed with a first tier, i.e., those pollutants and indoor environments where the evidence is already adequate as a basis for action. Further analysis, needed measurements and other research can be initiated for

contaminants and environments in a second tier. Such division could be made according to judgments of the importance of remaining uncertainties in 1) exposure-response relationships, 2) exposure patterns in California, and 3) effectiveness of available control measures. We would be happy to work with ARB staff in making such judgments or reviewing those made by others.

> **Response:** We agree with the Committee's intent to avoid any considerable delay in moving forward. Consequently, our responses fall into three general categories: 1) immediate changes were made (or will be made in the final version of the report), 2) future research is needed, and 3) comment requires intensive analysis that will not change the report recommendation, but should be carried out in the future.

We start by discussing each of the major pollutants, principally with regard to presentations in Chapter 2 and 3 and then comment on other issues in the executive summary and chapter by chapter. Minor points are collected at the end.

Radon

For the most part, the report relies on the various surveys conducted in CA to assess exposures and to estimate the annual number of lung cancer deaths associated with radon exposure. The overall health effect estimates for CA (p. 90) are done in proportion to the U.S. EPA estimates, adjusting for lower average radon concentrations in the state compared with the US average and correcting for the prevalence of smoking in CA vs. the US as a whole. This latter 'correction' must be done with some care, as the BEIR VI report on which the EPA risk assessment is based only provided risk estimates for two smoking categories - never smokers and ever smokers. Depending upon how many current non-smokers in CA are former smokers, the validity of this adjustment is unclear.

> **Response:** ARB staff consulted with the Department of Health Services, the lead state agency for addressing radon, in preparing these responses to comments on radon. The California radon risk estimate was characterized as preliminary and order-of-magnitude. It was based on an approximate adjustment for different smoking rates in California in recent years. The text was revised to stress the importance of the smoking-radon interaction, and that a truly more accurate assessment would require resources and data not currently available.

In the same paragraph on p. 90, there is a discussion about the estimates being 'worst-case' because "elevated radon regions in California are less populated". The distribution of indoor radon concentrations is irrelevant here, as the risk assessment is based on the average radon concentration, not the underlying concentration distribution. On the other hand, given that only 0.8% of the measured indoor concentrations are above 4 pCi/L in CA, compared with 7% nationwide, the task of determining which regions (and thus populations) are at potentially greatest risk is tractable. To some extent, this has already been done by virtue of the studies in the Sierra Nevada foothills and the Santa Barbara-Ventura counties Rincon.

> **Response:** The text concerning "worst-case" was removed. However, explanation was added to clarify that this estimate is nonetheless very likely an

overestimate, due to more recent data showing substantially lower levels in the Sierras (3% now vs. 25% previously estimated as above the 4 piC/l recommended mitigation level), and other factors as discussed in the revised text.

The report also notes, at p. 90 (3rd para.), that radon in drinking water contributes to the overall risk. However, the report should note that the estimated 168 annual cancer deaths is for the nation as a whole. Furthermore, based on the assessments of radon-in-drinking-water concentrations in CA, one would expect very few of these cases to occur in CA due to the low dissolved Rn concentrations. Note also that these cases are based on public water systems, not wells serving individual homes.

Response: Clarification was added that the 168 is a national estimate.

It is interesting to note that if the overall risk assessment approach were applied to average outdoor concentrations (0.4 pCi/L – which represents the 'lower limit' to radon risk reduction), the expected annual cancer death rate is still ~800 for CA (~600 if the smoking prevalence adjustment is made). According to the EPA radon risk assessment report (EPA 2003c in the ref. List), the estimated lung cancer deaths among never-smokers is 2900 nationwide or ~350 in CA. Adjusting this for the outdoor air concentration (0.4/1.25), the number of lung cancer deaths per annum in CA among never-smokers due to exposures equivalent to those outdoors is ~100.

Response: The text was revised to include an estimate of deaths if indoor radon levels were reduced to outdoor radon levels. It illustrates that, because radon is naturally-occurring and occurs indoors and out, there is a substantial background risk that cannot reasonably be mitigated, other than through reduction of exposure to tobacco smoke.

Finally, with respect to radon risk estimates, there are several places in the report narrative (p. 5, para. 2; p. 28, para. 1; p II-4) that state that radon risks have been reduced and that it is closely associated with smoking. The EPA risk assessment has, if anything, increased the risks associated with radon exposures. The previous risk assessment (circa 1992) yielded a central estimate of ~13,000 lung cancer deaths per year – the current estimate is ~21,000 and the relative risk estimate for never smokers has increased (see Pawel and Puskin 2004). With respect to never smokers, the estimated radon-related lung cancer death rate is 350 per year for CA, which is on par with the estimates for ETS-associated lung cancer. Finally, while there is uncertainty in all risk estimates for exposures to contaminants, radon is the least uncertain. There have been statewide and regional surveys, so the distribution of exposures can be reasonably estimated. The dose-response information is largely based on human exposures – uranium and hard-rock miners at high exposure levels and residential studies at the low end.

Response: As indicated above, ARB consulted with DHS on the responses to comments on radon. The NRC estimates for radon potency (and estimated deaths), on which EPA bases their estimates, have oscillated over the years. Between 1980 and 1987 (NRC's BEIR IV and BEIR V reports), the estimated risk and numbers of deaths were reduced, based on the NRC's reconsideration of differences between uranium miners and the general public, and other factors.

Subsequently, estimates increased, for various reasons. The point is that, despite the agreement that radon is a potent carcinogen, the most knowledgeable scientists have decreased and increased their estimates for radon over the years. Additionally, the estimates for number of deaths in the U.S. has also increased because of improved exposure estimates in high radon states, which is where EPA has focused much of its attention...but California has much lower levels of radon. EPA has changed their interpretation of the BEIR results over the years as well. It is important to note, though, that the majority of the risk accrues to active smokers; it is not spread across the population evenly. Thus, the risk numbers must be considered in light of other factors, such as the inseparability of the risk between smoking and radon, as noted by the NRC in its various BEIR reports. As the panel correctly notes, the risk to never smokers in California is much lower: 350 cancer deaths based on EPA's estimates. Most recently, the NRC (p. 19, BEIR VI report, Health Effects of Exposure to Radon, 1999a) stated that:

1) the deaths from radon-attributable lung cancer in smokers could most efficiently be reduced through tobacco-control measures, in that most of the radon-related deaths among smokers would not have occurred if the victims had not smoked;
2) the committee's...approach to assessing lung-cancer risks posed by indoor radon and cigarette smoking are subject to considerable uncertainty because of gaps in our scientific knowledge of effects at low levels of exposure; and
3) the attributable risk for smoking, the leading cause of lung cancer, is far greater than for radon, the second leading cause.

Specific comments

1. p. 5, middle paragraph. Better to refer to "radium-containing" rock and soil as the source of radon. (Uranium is the ultimate progenitor, but geochemistry can cause separation between uranium and radium, the immediate parent.)

Response: The text was revised.

2. p.20. The cancer risk from lifetime radon exposure, even at 1 pCi/L is remarkably high in comparison with the 1/100,000 used as the NSRL for Prop 65.

Response: Comment noted.

3. p.20. The State of California has frequently developed its own environmental quality standards and guidelines, and these are often more stringent than the federal ones. It is curious that the federal 4 pCi/L guideline has not been seriously scrutinized by CA agencies.

Response: Comment noted.

4. §2.3.9. Radon-222 emanates from the decay of radium-226 (not uranium-238). Radon is not directly a carcinogenic hazard; rather it is its short-lived radioactive decay products that accumulate in the lungs and irradiate epithelial cells.

Response: Text changed.

5. Based on the discussion above, the characterization of the risk estimate as "preliminary" on p 160 is unwarranted.

Response: The text was revised to characterize the estimate as a rough, order-of-magnitude estimate.

6. The radon discussion in Appendix II (p. II-4) is inconsistent with the exposure and risk estimates performed elsewhere in the report.

Response: The Appendix was revised.

Volatile organic chemicals

Formaldehyde

Appendix III presents a method for estimating HCHO concentrations in the current building stock. For manufactured homes, an overall reduction factor of 49% is applied to the concentrations measured in the 1980s, on the grounds that manufacturing processes have reduced average emission rates by this amount. However, this reduction applies to new manufactured homes and not to the existing manufactured home stock. A better (more defensible) basis for estimating the changes in indoor HCHO concentrations in these residences would be to estimate the annual rate of new manufactured home construction since the Sexton, et al. survey and combine these with the concentrations in the existing stock. The 49% likely didn't occur all at once, so some sort 'phased' reduction factor should be derived. Some accounting for the removal of older manufactured housing is also necessary, assuming the data are available on which to base an estimate.

The application of the 49% reduction to the peak concentration doesn't appear to be legitimate. Given that the 49% reduction applies to new construction, it is still possible to have high concentrations in older manufactured homes. In addition, since the 49% is an average, there are still likely to be new homes in which low-emitting products were not always used, etc. A better way to estimate the 90 or 95th percentile peak concentrations (not the highest) would be to use the concentration distribution given by the data from Sexton (GM and GSD) and add in an estimated distribution for new manufactured homes. This could be done year by year with the GM adjusted downward to account for the emission changes. As a first estimate, keeping the GSD the same is reasonable. In the end, there will be a new distribution of HCHO concentrations updated to 2000, from which one can derive an estimate for the 90 or 95th percentile peak concentration.

Response: New homes, not older homes, are the primary concern for exposure to formaldehyde. A more complex calculation that takes into consideration reductions associated with the percentage of older homes in California is not considered necessary, because most building materials have completed their majority of off-gassing after about five years. The changes in composite wood manufacturing processes occurred in the early 1980s; as a result, decreased initial off-gassing rates did occur substantially at one time. Manufacturers have

continued to change their processes to a lesser extent, so emissions have declined a small amount since the 1980s, but not enough to warrant a more complex mathematical approach associated with varying ages of housing stock. For the same reasons, a 49% reduction of the peak values measured by Sexton should be representative of today's high-end formaldehyde concentrations. An older house will not have high concentrations of formaldehyde as a result of composite wood materials that had a high initial emission rate 20 years ago.

One issue not addressed by the report directly is how much HCHO concentrations decline with building age due to out-gassing, etc. Some discussion of the aging effect is necessary, hopefully with some quantitative estimate of the decline in HCHO with age. The discussion on p. III-1, para. 6 – that human activities generating HCHO offset the aging effect – is incorrect on its face, unless there has been an increase in such activities with time. Besides, if such activities are important, they deserve their own set of exposure estimates.

Response: A paragraph has been added to describe the observed decrease in home formaldehyde levels during the first five years in new manufactured homes (Sexton *et al.*, 1986). An old (1980s) analysis of a small number of Oak Ridge, TN homes showed a distinct drop from years 1-5, relative to formaldehyde levels in older homes; however, while we have the slide based on that study, we could not locate the reference, and so have not yet included that study in the revised report. The report also contains other references to chamber studies and measurements in test homes taken eight to ten months after an initial measurement.

As building materials age and formaldehyde emissions decrease, people's activities have a more significant impact on indoor formaldehyde concentrations. For example, cigarette smoking, use of combustion appliances, the introduction of new clothing and fabrics, and the result of indoor air chemistry will become the major formaldehyde sources (not the building materials). Older homes can also experience substantial increases in their formaldehyde levels when new bookcases or furnishings are purchased, or when remodeling occurs.

Appendix III also presents an estimate for HCHO concentrations in conventional homes, which is based on a sample-size weighted average of two studies. Given the different natures of the two studies, this averaging is not justified. The report notes the limitations of the study conducted in southern CA – limited by the fact that it was summer only with potentially high ventilation rates due to doors and windows being open. The only legitimate comparison that can be made with these data are with data collected under similar conditions in the AZ study – either as a reality check or possibly to add the data from the two studies together for the same seasonal conditions. The AZ study also has its limitations with respect to its application to CA housing and these need to be discussed. While it appears reasonable to argue that construction practices and ventilation conditions may be similar (enough), a probability based sample for AZ will be heavily biased toward houses in Phoenix and Tucson, whose climates may be similar to some areas in southern CA, but not likely the coastal population centers like San Diego, LA and especially the SF Bay Area. Given that both temperature and humidity affect HCHO emission rates, these issues deserve further discussion in the report – perhaps with some emission-rate-based adjustments.

Response: In essence, this comment highlights the lack of recent California data for this topic. Reasons for including the two studies follow. The Arizona study is a recent study, and it is weighted to be representative of the entire Arizona population. To the extent new homes are included, the study includes accurate indoor concentrations for newly constructed homes. It is possible that differences in temperature, humidity, and ventilation considerations relative to California would lead to different indoor formaldehyde levels in California. However, those values are certainly within the same order of magnitude, and probably are within a factor of 2 of the Arizona homes. As far as we know, the study conducted in Southern California is the most recent California study. We felt it should be included, despite its shortcomings as to older housing stock and only summer sampling. As you mention, these facts are explained in the appendix, then a weighted combined estimate is calculated.

With respect to the peak concentration, there is certainly no good basis for adjusting the peak measurement from the AZ study. However, the report is inconsistent in its derivation and use of peak concentrations/exposures throughout. In those cases where sufficient measurements have been made, it is best to use a 90 or 95th percentile approach, which can be estimated directly from the data (if there are sufficient numbers of measurements) or from a cumulative distribution plot. Such an approach reduces the impact of an extreme measurement and provides a more statistically sound means of describing elevated concentrations.

Response: Your comment is well taken. We could leave the Arizona peak result unadjusted. However, not knowing the conditions surrounding that measurement, for consistency we adjusted it slightly downward by a weighted measurement from southern California. The net effect is that we report a peak of 232 ppb instead of 331 ppb (which industry has commented is much too high). Peak concentrations are used in Figure 2.4 to illustrate maximum levels that have been measured; they are used in Figure 2.4 because 90th or 95th percentile values are not available for most of the categories shown. In Table 2.7, on the other hand, 90th percentile concentrations for VOCs were available and are presented. Each is clearly identified.

In the end, given the potential importance of HCHO to indoor contaminant exposures and risks, a well-defined statewide survey of HCHO concentrations would be the best way to resolve these questions.

Response: Although we agree that a well-defined statewide survey of formaldehyde concentrations would provide the best results, such an effort is outside the scope of this report. The California Portable Classroom Study was recently completed, and provides a recent, statewide, comprehensive study of indoor formaldehyde levels. The results are disturbing and compelling: using just the field study results, which were the most conservative measurements, we found that over 4% of the classrooms had formaldehyde levels above OEHHA's acute REL for irritant effects, and all classrooms exceeded the acceptable cancer risk guideline. While a new residential statewide survey of formaldehyde concentrations would undoubtedly be enlightening, the body of knowledge associated with formaldehyde, along with cancer risk estimates, indicate that

formaldehyde is a chemical of major concern in the indoor environment and warrants prompt reduction.

Other VOC/TAC

The review of VOC and TAC is quite extensive in the report. In many respects, however, it presents an undiscriminating treatment of VOCs, even though it is clear that the authors recognize the strongly varying potency among different compounds. In addition to being careful to discriminate among the different compounds that *have been* measured, it is also essential to point out that many potentially important compounds for human health and comfort are not routinely measured. Carslaw (2003) presents a nice (brief) summary of this issue. Weschler (2004) is also a good source for the latest information on indoor air chemistry and its relationship to human health and comfort. (He has referred to this issue under the heading of "stealth chemicals.") Overall, the sections on sources, emissions and concentrations of various VOCs need context, which is provided by health information, and the levels at which health effects occur. In some cases there are reports of emissions that have been measured on VOCs for which no toxicologic or health data are reported; the report should either restrict itself to compounds for which adverse health effects are known or strongly suspected, or at least clearly separate these from compounds with known health effects, so the reader is clear.

Response: The VOC section is complex due to the many pollutants, sources, and health effects that are included within the category. Statements have been added to clarify that many compounds in indoor air are not routinely measured, and reactive chemistry is a new field of research. The report states that by definition there is no safe exposure level to carcinogens; it discusses the concept of exposure and dose, and refers the reader to the OEHHA website for specific information. The report quotes work by Hodgson and Levin (2003) stating that in most cases indoor levels of most VOCs are below health benchmarks such as chronic Reference Exposure Levels, odor thresholds, and irritancy levels (Section 2.3.2.1). However, as noted, some are frequently above acceptable cancer risk levels, and as noted by the panel members, there are many potentially harmful chemicals that are not routinely measured.

The risk estimates for these materials include HCHO, which is already discussed above. For the remaining chemicals, the concentrations and exposures are considerably less certain. The report notes, for example, that with the changes in smoking habits, some of these exposures (e.g., benzene) may be much smaller than that found in the 1992 study (Sheldon et al., 1992a). The overall risk estimate (annual cancer deaths) presented in Table 3.2 (p. 99) of 115 has large uncertainties associated with it (as acknowledged in the report) yet no estimates are provided for either the low or high case – implying a much greater precision on the central estimate than is warranted. At a minimum, one could use formaldehyde exposure as the limiting case (assuming these exposures are more ubiquitous than are exposures to other chemicals).

Response: As indicated in the report and Appendix II, the risk estimates are taken from the Comparative Risk Project. That document did not present the full breadth of information developed during the project, including confidence intervals or other measures of variability for each carcinogen, although such uncertainty was stressed in the text of that report. However, the panel's comment

is noted, and caveats were added in the report and the appendix to emphasize that the values used are estimates with potentially substantial uncertainty.

Specific comments

1. p 20, "architectural coatings." The "low VOC" products were developed to minimize ambient ozone forming potential. This is a very different goal than minimizing toxicity associated with exposure to primary or secondary emissions indoors. The presumption in this document is that these low VOC products have yielded benefits in improved indoor air quality. That presumption requires scientific support, or else it should be presented as speculation or inference.

   Response: Specific information was added regarding ARB restrictions against using certain toxic air contaminants in consumer products.

2. Missing from the discussion in this section are two source categories that seem important: outdoor air pollution and attached garages (and their contents). The only place attached garages are mentioned is on p. 65, second paragraph.

   Response: A discussion of information regarding attached garages was added for both the CO and VOC sections of the report. Some additional information regarding outdoor pollution was added as well; however, the focus of this report is on indoor pollution.

3. Health Effects of Formaldehyde (pp. 58-59). Given the large number of people exposed over the guidelines, as given later, and OEHHA calculations, compare the estimate the number of cancer deaths from formaldehyde with the actual # of nasopharyngeal cancer deaths in CA (shouldn't exceed)

   Response: Between 1998-2002 there was an average of 98 nasopharyngeal cancer deaths per year in California. Our estimate of 62 cancer deaths due to formaldehyde per year compares very well with this figure. The remaining cancers are most likely attributable to occupational formaldehyde exposures. We will add this information to the final version of the report.

4. Indoor Formaldehyde Concentrations (pp 60-61). End of first paragraph: Classrooms and offices should be of nearly equal concern as homes, given the distributions shown in Figure 2.4.

   Response: Text was adjusted in the report to indicate that classrooms and conventional homes also are a concern. However, offices have lower formaldehyde levels as would be expected, due to the different materials used.

5. Page 60, first bullet in 2nd list. Should not equate "emissions" with "concentrations." What does it mean to have "carpet emissions were generally below the limit of detection of 1 $\mu g/m^3$?"

   Response: Text was changed, thank you.

6. p. 67, last paragraph What levels of 2-methoxyethanol and 2-ethoxyethanol were found in these cleaning products? These are the solvents associated with

spontaneous abortion in the semiconductor health study (Swan et al., 1995; Eskenazi et al., 1995); these effects were observed at quite low levels, well under 1 ppm (Hammond et al., 1996). Note that 2-ethoxyethanol is mentioned again on p.70, 3rd bullet; once again, given its demonstrated toxicity, report levels

Response: Only 2-butoxyethanol was found in the cleaning products. Text was clarified and the concentration in the cleaning products was added. On page 70, the compound is 2-ethoxyethylacetate. The reader should refer to the citation for additional information.

Biological Agents

The treatment of "biological agents" in the Executive Summary effectively states the main points. Here are a few items that should be considered in revision:

- The list of biological contaminants (p 9) should also include microbial VOCs and SVOCs, which are often associated with unpleasant odors. (Although the link between odor and health is not strongly established, odor has well-accepted historical legitimacy as a basis for air pollution control measures.)

  Response: Although firm data associating indoor microbial VOC exposures with human health effects is not available, the reviewer's comments relative to malodors, especially considering individual variation in sensitivity to odors, are reasonable. MVOCs will be added to the list of biological contaminants. However, evidence for potential health effects from microbial SVOCs is too preliminary to include in this document.

- Transmission of infectious disease and exposure to pathogens from poorly maintained ventilation systems merit attention, as noted. In addition to the points made in the executive summary (p 9), it might be worth noting concerns about indoor air transmission of emerging infectious agents, as evidenced by the recent SARS outbreak. The broader relationship to security concerns and possible exposure to biological agents (e.g. anthrax spores) may also be worth mentioning.

  Response: Added SARS as an example of an emerging infectious agent. A broader discussion of indoor exposure to bioterrorism agents is beyond the scope of this document.

- Table ES-2 (p 11) lists the total costs associated with "ETS: asthma episodes" as 0.001 billion $/y and associated with "mold and moisture: asthma and allergies" as 0.22 billion $/y. It is not credible that the cost of mold and moisture-induced asthma is 200 times that of ETS-induced asthma. See further discussion below.

  Response: The medical costs for ETS-induced asthma were revised upward. Additional detail will be added in the future to include the explanation discussed below regarding potential underestimates.

  Because of differences in the types of data available, the original cost estimates in Section 3 and Table ES-2 for asthma due to ETS exposure and to mold-moisture exposure were calculated using different methods. The medical cost estimates for ETS used a unit cost for a doctor's visit ($42) multiplied by the

number of children with an asthma episode due to ETS exposure. The ETS estimate does not include adults, and does not include mortality, because such data are not available. Mortality cost estimates were not made for ETS-induced asthma because the ARB/OEHHA (2004-2005) risk assessment did not include premature deaths in its summary table for health. For the mold-moisture estimates, the medical and mortality costs were derived by taking a fraction of the total U.S. costs (direct and indirect) for asthma, and multiplying that by the fractions of asthma attributed to mold and moisture.

In the revised estimate, the medical cost estimates for ETS-induced asthma were revised upward using a unit cost of $640 (1990 dollars), the per capita cost of asthma in the U.S. (direct and indirect) cited by Weiss and Sullivan (2001). The mold-moisture estimate is now 10 times greater than the cost of ETS-induced asthma episodes. This remaining difference between the ETS and mold-moisture total estimates for the medical cost of asthma is due to the following:

- The attributable fraction of asthma due to mold-moisture is higher than that for asthma due to ETS (13% vs. 3.5%). The ETS value is taken from ARB/OEHHA (2004-2005).
- The ETS estimate is for children's cases only, so it does not include the substantial indirect costs for lost work days, as shown in the cost estimates by Weiss and Sullivan (2001).
- The direct cost for treating children is not fully reflected in the ETS estimate; the unit cost is based on the population average cost in the U.S., but children may require more medical care than adults.
- The unit cost used for the ETS estimate has not been adjusted from 1990 to 2000 dollars.

Biological Agents: Chapter 2

- In Table 2.1 (p 33), consider adding "building occupants" to the "major indoor sources" column for "biological agents." Certainly this is the major source category for infectious disease transmission. Another important source is "infestations" by cockroaches.

  Response: The recommended additions were made.

- The underlying reason for the increase in asthma is not well understood. It is misleading to suggest that indoor and outdoor air pollution are understood to be important causes. The opening paragraph on p 34 creates an inaccurate impression.

  Response: No change was made relative to this comment. The sentence in question (last sentence in the paragraph) was worded carefully, is already qualified, and is well-supported by literature citations. We believe the panel may have misinterpreted the statement, which reads "...indoor and outdoor air pollution have been identified as potentially important contributors to the increase of asthma." Nonetheless, we will clarify this statement in the final version.

- Care should be taken to distinguish information on asthma initiation (becoming an asthmatic) from information on asthma exacerbation (attacks in asthmatics). Different pollutants have different impacts, which in turn have different implications

for age distributions, burdens, and policy. If a study being examined does not provide information allowing us to tell whether the effect was on initiation or exacerbation, it should be so stated in the discussion.

Response: We agree with the panel's preference, and that is why both Tables 2.2 and 2.3 (from IOM 2000) are included in the report. Due to time constraints, we were not able to revisit all papers throughout the report that discuss asthma, but will make edits accordingly in the final version of the report.

- The specific reference to Tables 2.2 and 2.3 from the IOM report is appropriate. However, it is important to note that the IOM report only found sufficient evidence of a causal relationship for several factors: house dust mite (for development and exacerbation), ETS (for exacerbation among preschoolers), and cat and cockroach (exacerbation). The text on p 34 overstates the case of causality: "The committee found that, in addition to the known biological asthma triggers such as mold, house dust mites, and animal dander, chemicals such as ... *can exacerbate* asthma in sensitive individuals." (Emphasis added here.) This sentence strongly suggests causality, but for molds scientific evidence only supported a finding of an association, rather than causality.

  Response: The original sentence combined the results of several categories in a summary sentence, as the first sentence in a paragraph, followed by a specific discussion of each trigger, which is very explicit. The first sentence has been revised. Molds were included in the introductory sentence because the focus of that clause was biologicals vs. chemicals.

- In addition to the IOM, a large review was recently published in the EU related to this topic (Bornehag et al., 2004). A key conclusion: "Dampness in buildings is a risk factor for health effects among atopics and non-atopics both in domestic and in public environments. However, *the literature is not conclusive* in respect of causative agents, e.g. mites, microbiological agents and organic chemicals from degraded building materials." (Emphasis added here.) Given the existence of authoritative reviews by scholarly committees such as this EU review and also the IOM reports in 2000 and 2004, it seems inappropriate to give comparable weight to the review findings of a single scientist (Delfino, 2002) (p 35-36).

  Response: Reference to the Bornehag review was added, including the absence of an identified specific causative agent in damp buildings. The EuroExpo review (basis for the Bornehag et al. article, 2004) specifically sought studies that evaluated building dampness and health effects. Thus their conclusions that damp buildings increased the risk for respiratory symptoms in non-atopics and asthma exacerbation, but the underlying agent in <u>damp</u> was not identified. The Delfino review included a different population of studies looking at the relationship between indoor airborne chemicals and asthma regardless of moisture problems in the building.

- The discussion of Sick Building Syndrome (SBS) relies on relatively old literature of limited scope (p 41). More recent investigations that add substantially to the literature include Mendell et al. (2002), Seppanen and Fisk (2002), and Wargocki et al. (2002). While the causal connections with pollutants have not been elucidated,

Mendell et al. summarize the state of understanding for biologically plausible connections. All three of these papers make a strong case for associations between ventilation system deficiencies and SBS symptoms. The present review should incorporate some of this more recent evidence.

Response: The suggested references were added to the discussion of Sick Building Syndrome.

- Section 2.3.4 begins by listing biological contaminants (p 74). Properly, one should refer to fragments or excreta from house dust mites and cockroaches, rather than the entire organism. (Also applies at bottom of p 75.)

  Response: This additional detail has been added.

- The discussion of health effects of biological contaminants (p 74-81) relies heavily and appropriately on the recent IOM (2004) review. Overall, this section makes its major points effectively and accurately. It could be further improved in some specific details, as described here:
  - It is stated (p 75) that "colds are more often transmitted by direct contact." In our reading of the literature, the mode of transmission of rhinovirus is not well known. The statement should be supported by a reference to an authoritative source, or removed.

  Response: Text was changed to acknowledge that the mode of rhinovirus transmission is not fully understood.

  - A conference paper by Myatt et al (2002) is cited in relation to communicable disease transmission (p 75). A recent peer-reviewed journal article by the same investigators is a more scientifically compelling source (Myatt et al., 2004).

  Response: Updated reference included.

  - In discussing toxic responses to "damp or moldy" buildings (p 75), endotoxins and mycotoxins are listed as possible causative agents. This presentation leaves the impression that the problem is clearly of biological origin. The literature leaves open the possibility that the underlying cause of health problems in damp buildings is chemical, rather than biological. This point should not be lost in the presentation of evidence. (This is done well in a paragraph on p 77.)

  Response: The text states that exposure to toxic bacterial and/or fungal elements in indoor environments is controversial and not proven to be linked to human health effects.

  - Section 2.3.4.2 (p 75-77) discusses "sources" of biological contaminants. The subsection on house dust mites should note that the allergens are carried on excreta and body fragments that may become resuspended by simple indoor activities such as walking and cleaning. The discussion of cockroaches does not present information about sources. The review should

briefly explain what is allergenic about cockroaches and how exposure might occur. Similarly, the discussion of viruses does not say anything about sources of viruses.

Response: Clarification of mite excreta and body fragments as source of allergen has been added. Comment on potential for mite allergen resuspension is not included due to the rapid resettling rate and the large size of resuspended particles (IOM, 2000, p 139). Additions to text were made to augment sections on cockroach and viral exposure.

- On p 79, a statement is made that "healthy children in damp or moldy buildings sometimes report having more respiratory infections...." This statement requires a reference to an appropriate study; otherwise, it is inappropriate in a scientific review.

Response: Sentence revised and literature citation added.

- Unless you really think pulmonary hemorrhage is a concern (in which case more support is needed), drop the sentence appearing at the end of the 3rd paragraph, p 79.

Response: The sentence will be dropped or additional support added in the final version of the report.

- Section 2.3.4.3 (p 77) opens with the statement that indoor mold is experiencing "increasing occurrence at problem levels." This statement requires supporting evidence.

  Response: It is based on the great demand for mold inspection and remediation services that did not exist 10-15 years ago, and the increasing number of calls received from the public by state agencies regarding very serious mold problems (extensive visible or hidden mold) in homes in California. Mold was hardly mentioned at ARB's 1994 indoor air quality symposium, yet at a similar symposium in 2000, it was the unintended focus of discussion. However, we will revise the sentence in the final version.

- The discussion of "mold concentrations" (p 80-81) makes the important point that objective measures of moldy indoor conditions that would pose health problems are lacking. If indoor mold is deemed a high priority issue (as listed in Table 6.1, p 147), then research is likely warranted on methods of diagnosis.

  Response: The need for additional research in mold environmental assessment methods was added to the recommendation for further research.

Biological Agents: Chapter 3

- The review determines that 13% of asthma cases are attributable to residential mold and moisture-problems/dampness. The basis for this determination is an analysis of four large studies, as summarized in Table 3.4 (p 105). This 13% finding is important in the context of this review, as it drives the estimates of cost associated with

mortality (Table 3.2, p 99) and morbidity (Table 3.3, p 103). Several concerns arise that should be considered in revising the draft:

- The estimated asthma costs for ETS is $1.3 million per year, less than 1% of the estimated cost of asthma for mold & moisture ($190 million per year). It simply seems implausible that the true difference in costs could be so large, given these facts. (1) Epidemiology is the basis for both ETS and mold/moisture to be investigated as "causes" of asthma. (2) The prevalence of ETS exposure (e.g. in one's residence) and mold/moisture exposure is comparable in magnitude (~ 10-30%). (3) The odds ratio for asthma to be associated with mold/moisture is small in epidemiological terms (1.3-1.6). The differences should be reviewed and either the estimates revised or the differences explained.

  Response: See the response above, under Biological Agents.

- In presenting cost estimates for mold/moisture, the implication is that there is a causal association. (This is what is implied in the term "attributable.") Yet IOM (2000) could only conclude that there is an association between exposures to fungi/mold and exacerbation of asthma (Table 2.2, p 35).

  Response: Subsequent studies have strengthened the link between mold/moisture and asthma. Clarification of any causal association will be added to the report at a later date.

- The studies cited in Table 3.4 are not specific to California. Are construction and climate conditions sufficiently similar to justify the direct application of these studies to estimate conditions in California? Even if so, a caution should be added.

  Response: The text was modified to explain the applicability of these studies to California conditions.

- The presentation states (p 106) that the estimate "does not include the costs of other indoor allergen sources..." Please confirm that there was careful control in all of these studies on all other allergenic agents. Mold and moisture would tend to indicate poor operation and maintenance and this would likely correlate with the prevalence and levels of other allergenic agents.

  Response: Due to time constraints, those studies will be reviewed in the future and the report will be revised if needed to add any necessary clarification.

- At the top of p 106, the figure $24 million should be $240 million.

  Response: Thank you, the text was changed.

- The discussion of the potential for control of moisture and mold problems (p. 106) is inappropriately simplistic. Yes, of course, in principle mold and moisture problems can be controlled. We can also build a fleet of motor vehicles that don't emit

excessive pollutants, and we can eliminate smoking-related diseases by having everyone quit smoking. But these feasible solutions are very challenging to implement in part because they rely on informed action by large segments of the population. Any discussion of control should acknowledge the real and substantial challenges. Specifically, the statement that it is "probably feasible to eliminate at least 50% of the particle exposures that contribute to asthma" (p 107) is unsupported speculation and should be removed.

Response: We did not intend to be overly simplistic, but rather, brief. However, we do believe that preventive maintenance and proper attention to building operation and maintenance are more readily achievable than some other resolutions to indoor pollution that require new technologies, for example. The examples cited above...smoking cessation (where people are addicted), and a new fleet of low-emitting cars (where new technologies, and actions by both manufacturers and the public must all be implemented) would in our view be in a different, more long-term category, because they are more difficult to achieve.

- There is not sound scientific evidence to support the claim that biological particles are the dominant source of the adverse health effects associated with dampness and mold. (Furthermore, that statement is not necessary in the discussion here.) (p 104)

  Response: One paragraph was revised to remove reference to biological particles. Other similar references in the document will similarly be revised before the report is finalized.

Environmental Tobacco Smoke

Health Effects of ETS

The report has properly listed some of the most well known effects of ETS, e.g., lung cancer and heart disease, but some of the others are dealt with less systematically; some, e.g. SIDS, asthma induction, are mentioned in one section, but are not included in the calculations of the Costs of Indoor Air Pollution (Chapter 3). The California EPA has produced two excellent reviews of the health effects of ETS, *Health Effects of Exposure to Environmental Tobacco Smoke*, 1997, and the recent update, *Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant* SRP Version, October 2004. The information in these reports should be incorporated into this report, at least as a complete summary and with references to the report. We suggest a table of these health effects, including the estimated number of Californians affected by each disease (with upper and lower confidence estimates where available); this has already been done in the Cal/EPA report on Proposed Identification of ETS as a TAC, October, 2004. This document on Indoor Air Pollution should be congruent with the two documents cited above. Note that the effect of ETS on heart disease has a very profound public health impact, which deserves more attention than has been given in this report.

Response: The report summarized and cited the information from the October 2004 Part B report by OEHHA. This information has been updated using the more recent OEHHA estimates, and a table with OEHHA's case estimates attributed to ETS exposure has been added to the ETS Health Effects section.

The text and estimates will be further revised as appropriate after the Scientific Review Panel has approved the ARB/OEHHA ETS document.

Exposure to ETS

Environmental tobacco smoke is a major indoor contaminant/risk factor in CA for which the exposure estimates currently are substantially uncertain. The report notes this, but spends considerable time describing studies conducted before smoking was banned/reduced (pp 71-74). For the most part, these studies have little quantitative relevance today. Not only have workplace and public (e.g., bars and restaurants) ETS exposures been reduced to essentially zero, surveys indicate that smoking behavior in homes is being changed to reduce ETS exposures to non-smoking members of the household. ETS exposures of nonsmokers should also be adjusted to account for the observation that cigarette consumption in CA is about half that of the US.

Response: We concur that smoking is nearly gone from California's public buildings, and that fewer people are allowing smoking in their homes. Nonetheless, there are still significant exposures to ETS in some buildings in California. Older studies and studies of ETS in casinos and bingo parlors were included in the report for completeness, because some California environments continue to allow smoking. Some new casinos in the state allow smoking, and there remains a percentage of homes and other environments where smoking is allowed. Children are especially at risk of being exposed in homes and cars if an adult in their family smokes and does not avoid smoking around the children.

It is unclear how the panel's comment on ETS relates to the pages indicated, which discuss VOC levels in homes, not ETS. However, if the concern is that the VOC levels are outdated, cigarettes are not the primary indoor source of most of the VOCs discussed. Only the benzene measurements (and to a lessor extent xylene and toluene) would be expected to be substantially different in homes now. Additionally, the VOC study indicated was the last large, population-based indoor/personal VOC study to be conducted in the state, and thus warrants discussion. The Energy Commission is funding, and ARB is managing, a study of indoor air quality, including VOCs, in new California homes, which should provide more current information.

ETS: Chapter 3

Table 3.1 of the report lists the Unit Costs for Health Effects; these have been treated unevenly. At the very least, the costs of visits to physicians should be included, as these have been well studied. As an example, Table 6.11 of the document *Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant* reports that there are over 50,000 ETS attributable office visits for otitis media for children under the age of 3; even at $100 per office visit this comes to a cost of over $5 million. There are ways to incorporate also the lost work time due to having to take the child to a doctor. Similarly, Table 6.09 lists doctor consultations for respiratory symptoms, and reports a 15% increase if a smoker is in the household, and a 38% increase if two or more smokers are present. More seriously, young children have over twice the risk of developing bronchitis or wheezing if they are exposed to a pack or more a day (Table 6.08).

Response: The text and tables have been revised to include unit costs and total cost estimates for all but one of the health outcomes with estimated case numbers from Table ES-2 of the OEHHA ETS risk assessment. Otitis media (middle ear infection) was added. The unit cost for asthma cases has been increased to $640 per year, based on national per capita averages, as discussed above. Respiratory symptoms and bronchitis are not included in this table, so cost estimates were not developed. SIDS was not included in the cost estimate because cost estimates were not available. Assuming that each case of premature death from SIDS would have a valuation of $6.3 million, the cost would total about $130 million. This amount would not significantly affect the total cost estimate for ETS (over $25 billion in Table 3.6).

The cost of premature delivery was not estimated separately because it overlaps with the costs of low birth weight to a large degree. Caveats were added to the text for the additional costs of premature delivery that could not be quantified currently.

How was the cost for low birth weight children ($118,000 per case) derived? How low birth weight? Most ETS related low birth weights are small decrements in birth weight—are these the number of cases of babies born with a weight under a given weight, or with a statistically significant lower birth weight?

Response: The cost is based on U.S. EPA's Cost of Illness report, as summarized in Section 3. U.S. EPA compared the costs for low birth weight infants (less than 2,500 g) vs. other infants. OEHHA (2004) attributed a 20-100 g decrement in birth weight due to ETS exposure, and a downward shift in the distribution of birth weights. The final report will clarify this discussion.

Parts of Table 3.3 seem inconsistent. For example, the medical cost for ETS asthma episodes is estimated at $42 each, yielding an estimated cost per year of $1.3 million, while asthma costs related to mold and moisture are estimated over 100 times greater--$190 million. Does this really make sense? What about emergency room visits for asthma attacks? Different methodologies may have been used to derive these numbers (see page 106), but they need to be reconciled when they are compared and contrasted, as in this table.

Response: The text and tables have been revised, as discussed above.

ETS risk assessment

The basis for the risk estimate in this report (p. 100) is from OEHHA, based only on spousal smoking (incorrectly referred to as 2004 – should be 2003c), although this has apparently not yet been peer reviewed by the external review panel for OEHHA.

Response: The text and tables have been revised to reflect the latest OEHHA risk assessment, which has been revised in response to the panel comments and is still under review.

The risk estimate given on p. 100 is based on estimated US lung cancer rates, re-scaled for the CA population fraction, corrected to the year 2000. No correction was made for

the differences in smoking rates, although the data are discussed in para. 5 and the *same data are used to correct the risk estimates for radon (described earlier).* This would reduce the central estimate to ~ 275, based on the estimated number of smokers. An even lower estimate would result from the observation that cigarette consumption in CA is about half that of the US.

> **Response:** Changes, clarifications and caveats will be to added to the report at a later date to reflect the latest OEHHA risk estimates and with the radon approach.

Table 3.2 (p. 99) surprisingly does not provide upper or lower bound estimates for annual lung cancer cases associated with ETS. Clearly such bounds are necessary, as the current estimates are misleading as to the precision of the central estimate. At the lower end, it is possible that behavior modification by smokers in their homes has reduced non-smoker exposures. At the upper end, estimates of ETS exposure based on nicotine measurements may be underestimated, based on recent work reported by Apte, et al (2004). This work demonstrated that for situations where the interior surfaces are not chronically exposed to ETS, nicotine sorbs to these surfaces more rapidly, thus leading to lower measured nicotine concentrations in the air (and hence, lower ETS-RSP exposure estimates).

> **Response:** The text was revised to show the range in the latest OEHHA estimates. OEHHA used the lower bound because it was based on the best study available.

Particles (PM)

We were concerned that the health effects of indoor PM were not discussed in detail. Although PM epi has not been done with indoor-generated PM (IPM) per se, there is little reason to think that IPM is somehow not a health hazard, given its sources (combustion, for example). And, if less hazardous than outdoor PM, it is unlikely to be so by a large factor. Extrapolating from outdoor PM to IPM is less uncertain than extrapolating, e.g., from high dose-rate studies to low dose-rate environmental conditions, or from animal studies to humans.

This raises a variant of the drunk-looking-under-the-streetlamp principle, i.e., it is a bit of an anomaly that we have such excellent and extensive work on outdoor epi. It is simply because, unlike most risk factors, a small number (often just 1) of existing measurement stations can be used to characterize reasonably well the changes in exposure to huge *populations with reasonably good health records for important outcomes. The area* illuminated by this strong streetlamp, however, should not be confused with the area where the biggest impacts from PM exist (which include different locations, populations, and diseases). With appropriate care, we should attempt to extrapolate the results to the darker parts of the street (e.g., indoor PM, vulnerable populations, and, even, end points not usually examined, childhood pneumonia, for example, as done in the WHO Comparative Risk Assessment (Cohen et al., 2004). Such kinds of extrapolations are already accepted as necessary and useful from occupational settings and animal studies to indoor environments, arguably larger stretches than from outdoor PM to IPM.

If CARB were to extrapolate outdoor PM epi to indoor concentrations from indoor sources, even if there were to be some discounting due to different particle mixes, the results are likely to be much larger than the relatively small contributions to frank health risks from VOCs. Given that it is based on epi with similar populations and exposure levels (and not animals and high-exposure occupational settings), it would likely be more convincing as well to most observers.

> **Response**: We agree that indoor PM assuredly has large impacts on health. Accordingly, the November 2004 report included a full three-page discussion of the health effects of ambient PM (more than any other pollutant in the report), because there is no such body of information for indoor PM. That was followed by a discussion of the likelihood that indoor PM has similar effects. In response to the panel's comments, we have added additional studies and text to bolster those sections of the report. However, as discussed in the report, ARB convened a panel of expert scientists in indoor PM in February 2004 to advise us regarding the conclusions that can be drawn at this time regarding the health impacts of indoor PM. They concluded that there is insufficient information on which to base any type of quantitative estimate of the health impact of indoor PM. Additionally, a European group conducted a similar assessment, and came to a similar conclusion (Schneider et al., 2003). We also consulted with the OEHHA's PM staff, who concurred with those conclusions. Thus, we are not able to include a quantitative estimate in this report. However, ARB is in the process of funding research into the health impacts of indoor-generated PM, beginning with indoor combustion emissions, and we hope to begin to shed some light on this critical area very soon. We encourage others to pursue this area of research as well.

Indoor and Personal PM Concentrations

The report overstates the similarity between indoor and personal PM, especially if one examines CA data as presented in Table 2.5. Thus, the third line of the report inaccurately states that personal exposures "often exceed both indoor and outdoor concentrations." However, examination of Table 2.5 for PM2.5 reveals that while this is true for studies in Boston, Detroit, and Baltimore (Midwest and east coast), it is less true for studies in California. In the Suh 2004 and the Linn 1999 studies in LA and the Evans 2000 study in Fresno, the personal exposures were less or comparable to the outdoor concentrations; only the Suh 2003 study of COPD subjects in LA found personal exposures greater than outdoor concentrations. Similarly, the report should note that, while Boston and Detroit had *higher indoor* than outdoor concentrations of both PM10 and PM2.5, California cities generally had *lower indoor* concentrations for PM10 and PM2.5 than ambient concentrations.

> **Response**: Changes were made to the text to clarify these relationships, and additional edits may be made in the final version of the report. However, the statement that personal exposure levels are often higher than both indoor and outdoor concentrations is correct, for the general population and for certain seasons. Additionally, personal exposures are more often correlated with indoor concentrations than outdoor, particularly in population-based studies, although a few studies have found stronger correlations with outdoor PM. Study design, season, and study population are all key characteristics that affect the PM levels measured. The PTEAM study, a large population-based study of the general

population conducted in Riverside, found a much higher personal PM exposure levels relative to indoor and outdoor levels, which were similar. Since PTEAM, most studies have improved the study design (by using a longitudinal rather than cross-sectional design), but have focused on sensitive populations (many elderly or with compromised health), rather than the general population. Members of sensitive groups often have different activity patterns than the general population: they may be less active, and conduct fewer activities that generate indoor PM, and they may operate their homes differently (more or less window-opening), resulting in lower personal exposures and indoor concentrations than healthy families with active children and pets might generate. There are also distinct seasonal differences in the relationship among indoor, outdoor, and personal concentrations, and also differences between PM2.5 relationships and PM10 relationships: these are both reflected in the Fresno data, for example. Finally, it should be noted that the Suh studies (2003 and 2004) both involved very small sample sizes...about a dozen homes in each (the sample size indicated is the number of daily measurements...this will be clarified in the final report).

We have several suggestions for Table 2.5

- The values reported should be geometric means or medians, not means, which can be elevated by one or two high values.

  Response: Some studies did not report geometric means, so arithmetic means are shown. Additionally, the arithmetic mean is more reflective of the entire distribution, including the high end values that may represent the most impacted portion of the population.

- The entries should be grouped first by regions of the country
- All studies in one city (e.g., LA, Boston) should be grouped together to ease comparisons

  Response: We are not attempting to compare indoor concentrations by region of the country. Season, urban vs. rural location, subject type (COPD, etc) and activity level, and several other factors are at least as important, if not more so, in determining indoor PM concentrations. Regional groupings might be easier for identifying California studies, and may be provided in the final version of the report; however, studies of indoor PM are sufficiently new that studies in other regions of the country remain very informative.

- Some measure of the dispersion of the data should be included (e.g., SD, GSD)
- The number of samples should be reported

  Response: Done.

- Residential data should be separated from office building data
- Include the other office building data mentioned on page 50

  Response: The office building data from the BASE study are now shown at the bottom of each table. The other two studies are not included in the table because they were conducted in the early to mid-80s, in buildings where substantial

smoking occurred, and are not particularly relevant to current conditions in California.

- Where possible, report data separately for buildings where smoking did and did not occur during the sample collection

Response: Clarification has been added in the text that most of the studies shown in the table are from homes or buildings where little or no smoking occurred, most often because of the health problems of the participants. One exception is the PTEAM study, in which a very small percentage of homes were smoking homes. A footnote will be added to the table in the future.

Other points:

- p. 49 top: Spengler et al. reported measurements of personal, indoor, and outdoor PM sampling long before the PTEAM study—we think it was the 6 City data reported in the 1980s
- p. 49 3rd paragraph, 6th line:, rather than "for each of the three groups," actually,, for 3 of the 4 groups for outdoors, and all groups for indoors
- p. 49, 3rd paragraph, 6th and 7th lines: the data on PM10 personal are missing from the Tab le—please include
- p. 50 2nd line: 19.6 here but 19.5 in table—reconcile
- p. 50, 8th and 9th lines: a percentage is missing

Response: Corrections and additions have been made, thank you.

- p. 50, 2nd paragraph: The data discussed here are quite different from other comparable data, where the personal exposures were comparable to the indoor, and less than the outdoor; the discussion is therefore misleading

Response: Comment noted.

- p. 50 3rd paragraph, line 8; the indoor concentrations reported should be compared to outdoor concentrations. Did the BASE study uniformly exclude buildings with smoking (we think it did not)? This should be considered in it evaluation here.
- Do we have data on smoking for any of the home studies reported? If so, report whether smoking occurred during sampling, and separate reports from homes with smoking from those without smoking and place on adjacent lines
- The last two points may lead to an observation along the lines that smoking is a major contributor to indoor PM. The Spengler study alluded to earlier also provides data on this point, and estimates an increase in PM of about 1 ug/m^3 per cigarette smoked per day, or 20 ug/m^3 per pack.

Response: Information from Spengler et al was added in the PM source emissions section. Where smoking occurs, it is indeed the greatest contributor to indoor PM, as shown in many earlier indoor PM studies not included in Table 2.5. However, as mentioned above, most of the studies in Table 2.5 had little or no smoking in the homes during monitoring. We will review these studies and

determine whether there is sufficient additional information to warrant separation of smoking and non-smoking locations in this table.

Carbon Monoxide (CO)

- Page 52. It seems unlikely that the epidemiological studies cited here determined CO *exposures* with a high degree of accuracy.
- *Owing to indoor sources and also variable proximity to vehicular emissions*, one doubts that ambient CO is a good proxy for CO inhalation exposure.

  Response: The California CO study was a study of death certificates, not an epidemiology study, where CO poisoning was the known cause of death. Ambient CO was not measured or discussed in this study. Rather, blood CO levels and the decedents' immediate history were used to assign a cause of CO poisoning in the death certificates.

- First two lines on p. 52 state natural gas, propane and kerosene fuels add to > 99%, but motor vehicles "also took a substantial toll" (31% in figure 2.2)

  Response: The summed percents relate to the preceding sentences, which discuss the percent of the indoor deaths attributable to the different types of indoor sources. Those indoor sources contributed about 50% of the total deaths due to accidental CO poisoning, as stated in the preceding sentences. Additional clarification will be added to the final version of the report.

- How many homes had indoor CO greater than the state standard of 20 ppm?

  Response: It is unclear which study this comment is referring to. The CO mortality study did not involve measured data, as indicated above. In the Wilson et al study, several homes had levels over 20 ppm; we will insert the actual number of homes in the final report.

- Page 53, §2.2.4.3. The first line ("most homes have relatively low CO levels") begs for support. CO levels in the wintertime may often be persistently elevated owing to high ambient levels (e.g. in Los Angeles) and to the use of improperly vented heating devices (e.g. in the mountains).

  Response: Our statement is correct and well-supported. In the two large California studies cited (Wilson et al 1993 and Sheldon et al 1993), both of which included winter data and sample sizes of 277 and 280 homes, respectively, neither showed more than a few homes with CO levels above health-based standards. Ambient CO levels have decreased sufficiently in Los Angeles that there are very few winter excursions above the ambient standards.

- Perhaps move figure 2.2 and the discussion of types of appliances to section 2.2.4.3, Emissions.

  Response: We believe it fits best with the discussion of the results of the mortality study, but the information from that study could be split as suggested.

- The paragraph that refers to Springston et al. (2002) should point out that these were commercial buildings (in which combustion appliances might have been absent altogether).

  Response: We will check that for the final version of the report.

- Related to vehicle emissions, probably more important than CO in ice rinks is accidental poisoning deaths associated with idling motor vehicles in attached garages. See Marr et al. (1998).

  Response: We have added a section on attached garages to the report.

- Page 98, §3.1.1. We see no rationale for excluding CO poisoning deaths from motor vehicle emissions as many of these occur in indoor spaces. See Marr et al., 1998.
- Page 98, middle. A better reference (than CPSC, 1997) to the recent state of accidental CO poisoning deaths nationwide is Mott et al., 2002.

  Response: In the California CO mortality study, vehicle-related CO deaths often occurred outdoors, under circumstances such as individuals trying to warm up the car (using the heater while idling a long time), and many were associated with alcohol use. The statistics on the number of deaths that occurred in an attached garage were not readily available, and would likely add very little to the total CO mortality estimate.

Pesticides

Pesticides are substantially discussed in the report, but notably absent from either the "high priority" or "medium priority" source lists for mitigation. The omission seems inappropriate. The underlying reason for the omission is unclear.

Response: Pesticides were added to the medium priority sources for mitigation.

Health effects section (p. 81, para 5 ff) on pesticides is weak. More CA data on pesticide concentrations should be available now from the ongoing studies by Pat Buffler and Brenda Eskenazi at UC Berkeley, however.

Response: Information was added to the pesticide health effects section. However, time did not permit consulting with the investigators suggested above.

Non-industrial Workplace Exposures

With respect to workplace exposures, the report focuses mostly on office exposures, and these are treated well. However, there are other non-industrial workplace exposures that should be addressed. Brief mention is a made of a few of these occupations (janitors, barbers, and beauticians), however, a more systematic effort is needed to address the exposures of these workers, and to evaluate other non-industrial occupations with important exposures, e.g., auto repair shops, indoor construction work. This would be, however, an enormous task, and perhaps the scope of the report should

actually be restricted to home and office exposures, although the original charge to ARB was broader. A clear statement is needed at the start of what is covered.

> **Response:** We realize the information on non-industrial workplaces was weak. Thank you for acknowledging that inclusion of this information could be "an enormous task". We have included a section, 2.4, to highlight some of the exposure information that is known regarding non-office, non-industrial workplaces. While not a robust assessment, the new section provides a reasonable handle on the scope of the many indoor air quality issues that arise in non-industrial workplaces in California. Additional information may be added to the final report.

COMMENTS BY SECTION

Executive Summary

As the summary stands now, a reader cannot get a good overview of either the health outcomes or of the sources.

The report contains a large amount of data on a very broad topic. However, it does not synthesize the data well. Thus the second bullet states 230 excess cancers occur from indoor carcinogens, yet that excludes those from ETS, radon. The authors should choose a template to summarize the data, either by health outcome or by source—currently it is mixed, and not all the data are presented. To the degree possible, numbers should be presented for all of the outcomes for all of the relevant exposures (e.g., numbers of lung cancers attributable to VOCs and radon, but not ETS, are presented; no numbers are presented for the number of coronary heart disease deaths and cases attributed to indoor air problems, yet this is likely to be a much larger number).

> **Response:** It has been a challenging task to organize the information in this report. We have attempted to organize information by pollutant with Table ES-1 serving as a summary for what is known about the pollutants, sources, and associated health effects. Methods for mitigation are organized by source categories (Table ES 3.1 and 3.2) because manufacturing changes have the potential to address multiple pollutants (if more than one is present) associated with a given product. The numerical estimates for the major health endpoints were specified throughout the summary.

Page 6, penultimate paragraph. The reference to ambient PM mortality should not refer to an association with "exposures" but rather with "concentrations." (It is very important to be precise in distinguishing among all of the parameters of concern.)

> **Response**: Statement corrected, thank you.

Page 8, first paragraph. Worth mentioning here that ozone produces PM as a byproduct as well.

> **Response:** Text now includes PM.

Chapter 4 Existing Regulations

The discussion of current regulations, guidelines and practices seems very complete and the review committee did not identify any major omissions. As a general observation however, the discussion presented in this chapter is not always careful to note that many of these regulations, etc. are explicitly focused on outdoor air quality and only secondarily – if then – on indoor air quality.

Response: The text was revised.

Transference of standards based on outdoor epi to indoor air should be done with caution. Outdoor epi is done by looking at changes in health based on changes in outdoor levels, not changes in exposure. Since most people spend most of their time indoors and the indoor concentrations of outdoor pollutants is less than they are outdoors, the real impact per unit exposure is higher. Consider, for example, that a change of 50 ug/m^3 of PM causes so much ill-health in the outdoor epi. If the penetration level is 80%, however, it is actually showing the effect for only a 40 ug/m^3 change in exposure. Indoor levels are much more closely related to real exposure, however, and so, arguably the same amount of ill-health represented by 50 ug/m^3 of outdoor PM would occur with a 40 ug/m^3 of indoor pollution (from indoor sources). Standards should be modified accordingly. All this of course does not take into account the different populations that may exist in certain indoor environments (more vulnerable) or different toxicities of indoor and outdoor particles.

Put another way, having a less stringent standard for indoor than outdoor air does not meet the "laugh test" for protecting public health. Would then an appropriate approach for pollution control be to ban chimneys and other methods of taking pollutants out of buildings where people spend time? To the extent that indoor environments can be considered a public good (and there is a substantial basis for doing so to a significant degree), the only possible reason to have less stringent standards indoors would be because it has been shown that IPM is less unhealthy than outdoor PM because of its chemical/physical characteristics. Arguments about population vulnerability and less than 100% penetration from outdoors to indoors only argue for more strict standards indoors.

Response: Ambient air quality standards were used as approximate benchmarks in our indoor air quality guidelines, similar to what was done for the WHO guidelines for indoor and outdoor air quality. Ambient air quality standards are comprised of two parts: a concentration and an exposure duration, or averaging time, that are relatively safe, with a margin of safety. A person exposed to a certain level of PM for one hour would experience the same health impact whether he/she is standing indoors or outdoors. As long as the averaging time is specified, there is no need to adjust an indoor guideline level....the level is set based on the duration of exposure, or averaging time, and the concentration. Also, to clarify, this section discusses indoor air quality guidelines, not standards: there are no indoor air quality standards.

Specific comments

1. Page 111. The limitations of workplace regulations are more severe than those listed. They do not apply to indoor environments that are not workplaces. Also, they have been developed from an industrial hygiene perspective in which one or a few key chemicals dominate exposure. They seem entirely ill designed to serve as a basis for evaluating the health and comfort risks posed by the complex mixtures found in, e.g., modern office buildings.

   Response: There are few buildings that are not workplaces covered by Cal/OSHA's regulations. Unlike federal OSHA, Cal/OSHA's rules apply regardless of the number of employees. Only federal buildings are not covered by state regulations. As indicated in our text, Cal/OSHA has not only PELs, which we agree (and have stated in the report) are inadequate to protect many people, but they also have other regulations such as the Minimum Building Ventilation Standard, which applies broadly, and is protective of, or provides benefits to, any person in the building.

2. Page 113. Have the radon in drinking water regulations been promulgated and finalized?

   Response: Not yet. Will clarify in final report.

3. §4.3 Emission Limits. Much of this section is far from the issue indoor air pollution (e.g. the discussion of ARB's consumer products and architectural coatings programs). Note the earlier comment on whether the coating program has a positive impact on indoor air.

   Response: Language has been added regarding the impact on indoor air (ARB has prohibited some TACs in certain products, for example).

4. p. 125. The section on "DHS non-binding guidelines" does not discriminate among VOC emissions based on toxicity. Nor does it consider the possibility of secondary emissions caused by oxidation (e.g. owing to ozone exposure) of materials.
5. Page 126, first full paragraph. This sentence doesn't make sense as written: "emissions from a single material or product cannot exceed one half the chronic REL." Emissions would be expressed in mass per time. The REL is expressed in mass per volume. They cannot be directly compared.
6. Page 127, §4.3.3.3. This statement requires substantiation: "These programs have been successful in reducing emissions from their products over the last few decades."
7. Page 128, bottom. As in comment 64, the discussion of the GEI emissions criteria mixes measures. The implication is that emissions are to be limited so that individual VOCs "must meet the criteria of less than $1/10^{th}$ of the threshold limit values...." In addition to the problem of equating emissions to concentrations, this also seems like an ineffective criterion for ensuring good indoor air quality. (An indoor environment in which multiple chemicals approached $1/10^{th}$ of their respective TLVs would not be healthful for general occupancy.)
8. Page 131, lower half. The citation Maeda, 2004 does not appear in the reference list. (The committee did not audit the concordance between citations and references in general.)

9. Page 132, §4.4.1.3. Weatherization and duct sealing would affect not only indoor levels produced by combustion appliances, but from other sources, too.

Response: Text was revised for most of these comments; remainder will be made in final version.

Chapter 5 Methods to Prevent and Reduce Indoor Air Pollution

In a document that is painstakingly detailed with respect to regulations and guidelines (30 pages), it is disappointing to find control and mitigation so thinly discussed (5 pages in the main report, plus 2 in the executive summary). The major themes are more or less correct: source control, ventilation, public education, air cleaning devices, and building operation and maintenance. But the level of treatment of these topics is superficial to the point of being simplistic. This section is the weakest part of the report.

Response: We agree that the section needed work, and have revised and expanded the entire section.

Detailed critique is provided below for the first two topics.

- *Source Control (p 15 and 141)*
  - "There are many actions that can be taken to reduce indoor air pollution and, in some cases, completely eliminate sources." The problem of source control is much more complex and challenging to implement than acknowledged in this section.

Response: Text was revised.

  - Actions taken to "completely eliminate sources" are only pertinent for indoor emissions, and only for a subset of pollutants. When sources are outdoor air or the occupants themselves, then complete elimination is not an alternative.

Response: Section was revised, but it was never stated that all sources could be eliminated.

  - "The most effective preventive measure is to use building materials, consumer products, and appliances that emit little or no air pollution." This might be a practical alternative for some indoor air quality problems, but only if such information were widely available, which is not the case. Specifying reliable and practical test methods that work across the many diverse source categories is but one of many challenges.

Response: Several available methods are mentioned in the options for mitigation section, but we agree that others are needed.

  - Reformulation of manufactured products and processes to eliminate certain toxic compounds and minimize overall emissions is an appropriate strategy. The report should acknowledge that not all indoor air problems are of the sort that can be controlled by this means (e.g., SBS, for which the causative agents are not yet known). It should also acknowledge that emitted species

vary markedly in their toxicity and so it is the composition of what is emitted in addition to the total amount that matters. Third, it should acknowledge the potential significance of secondary emissions, occurring because of reactions among the constituents or because of degradation/oxidation with time following installation or use.

Response: Some revisions have been made, and additional clarification will be added in the future. A more detailed assessment of potential risk reduction measures would be conducted before specific product reformulation is recommended.

- *Ventilation. (p 15, p 142-143)* Ventilation and its role in influencing indoor air quality is a much richer subject than can be adequately addressed in the brief treatment it receives in this report. Overall, relative to its importance, the document undervalues ventilation as a part of the overall indoor air pollution issue, particularly in office buildings and other structures with mechanical ventilation. While source control is an important part of an overall IAQ management strategy, so is the reliable provision of an adequate supply of good quality ventilation air to indoor spaces. Emissions control is clearly most important to avoid extreme problems (e.g., CO poisonings). But emissions will never be reduced to zero. Ventilation has an important role affecting IAQ in typical indoor environments. Important elements of the ventilation dimension of the IAQ issue that are inadequately discussed in this report include the following:
  - Data on current ventilation rates in the California building stock and trends.
  - Relationship between ventilation and indoor pollutant levels.
  - Challenges of providing adequate ventilation in the new residential building stock, given tighter construction practices.
  - Tensions between reducing ventilation for energy efficiency (CEC's focus) and the need for adequate ventilation to ensure good IAQ (and the technical potential for improving ventilation efficiency or effectiveness).
  - Challenge of providing good quality ventilation air when ambient air is unacceptably polluted, a common condition in California.

  Response: Information and Figure 5.1 were added. Some additional information may be added in the final version.

  - Consideration of "ambient air" as either a "high priority" or "medium priority" source category for mitigation.

  Response: The mitigation of pollution in ambient air is already a high priority...it is the reason for ARB's existence. However, it is not the focus of this report. Improved air filtration was included in recommendations for improved building operation and maintenance and indoor clean air technology development.

  - (p 22-23) Ensuring adequate ventilation in the building stock is glaringly absent from the 9 recommended "elements of an indoor air pollution reduction program." (Brief mention of proper venting of combustion sources under point 6, and low-noise fans under point 9 are not commensurate with the importance of this issue.)

Response: Recommendation No. 7 was revised to specify ventilation as a high priority. However, ventilation problems are largely ones of enforcement and diligence, not lack of laws or regulations. The Energy Commission has authority to establish minimum levels of outdoor air, and Cal/OSHA has authority over HVAC operation and maintenance.

- *Litigation.* Needed here also is a section discussing the history of litigation and its threat as an intervention for IAQ, which is referred to several places in the text, but never discussed directly. One of the arguments for keeping the government out of IAQ is that the courts work well enough to keep us safe. Clearly, there have been successes, which should be acknowledged, but also there are failures. It would also be good to examine how well this can be expected to work in future and whether there might be legislation that could make it work better.

  Response: Such information was added to the text.

Chapter 6 Prioritization of Sources and Pollutants

The criteria for choosing priorities need to be made a bit more clear as well as how they were applied. Indeed, it is the committee's view that there are really two (or possibly more) tiers – Tier 1 – where the indoor exposures for which the contaminant concentration or exposure distributions are fairly certain and the exposure-health-risk relationships are reasonably well understood and Tier 2, where there are uncertainties in these characterizations that would prevent informed actions (based on current knowledge). Within these tiers, prioritization criteria could be defined and applied.

Response: We believe that the two tiers of prioritization by source is the best approach, because then multiple pollutants emitted from a single source can be addressed at once. It is unclear where the panel believes the known health effects to be less certain.

- The EPA has done some interesting graphs showing the result of multiplying the range of exposures to a substance times the range of potencies expected to derive a range of impact. Can CARB do something similar?

  Response: Such an effort would take substantial time. (EPA spent two years on such an assessment for indoor pollutants, which has not yet been released.) This approach would be a useful approach for a comprehensive program.

- Are there (hidden) criteria of cost-effectiveness, i.e. are some things left off because they are considered undoable at reasonable cost?
- Are there (hidden) criteria of political feasibility, i.e. are there some things left off (e.g., candles, incense) because they would not be acceptable?

  Response: No, nothing is hidden. This analysis was not intended to be as detailed as indicating all sources in a category that might be addressed.

- What happened to lead? It is clearly important, but perhaps A) it was determined not to be an air issue because most exposure is by other routes or B) it was felt that it is being handled already. Explanation needed.

- Mercury, although perhaps less important, raises the same questions.

  Response: DHS manages the lead health program for the state. Mercury is handled by DHS and some local health departments.

- Pesticides are conspicuous by their absence on either the high or medium list of priorities.
- What happened to attached garages? And the related issue of gasoline vapor exposures? Nasty stuff, gasoline.

  Response: Both were added as suggested in the revised report.

How would all the proposed activities be organized and coordinated within the state? This seems like an extremely important but unaddressed issue.

Response: An agency in charge of a comprehensive program would clearly need to routinely coordinate with other agencies and interested parties. For state agencies, the California Indoor Air Quality Interagency Working Group serves as a good example of how such coordination might be accomplished. That group meets quarterly and is coordinated by DHS to foster communication and collaboration among agencies in addressing indoor air issues. Additionally, ARB has convened state agency review groups for this report and for the Portable Classrooms Study report, and received excellent input and suggestions on both reports. Finally, the state Sustainable Building Task Force and other interagency groups have successfully involved all agencies needed to assess specific issues or to provide input on various projects, and serve as successful prototypes for effective interagency communication and coordination.

Chapter 7 Options to Mitigate Indoor Air Pollution

(Pages 22-25 and 153-159) This portion of the report advances recommendations about actions that can be taken to improve indoor air quality in general, and in schools in particular. These recommendations are consistent with underlying science, to the extent that it is currently understood. A few observations about this section are worth considering during final revision of the report.

- (p 153) An effective overall management system for indoor air quality requires not only knowledge of pollutant sources, behavior, and consequences, but also strong expertise in building sciences. Part of the difficulty in effectively responding to the indoor air pollution problem is not only because of a lack of clear authority for the issue within any state agency, but also because of fragmented and incomplete relevant expertise.

  Response: Comment noted.

- (p 156-157) The emphasis on source control throughout this report should be tempered by the findings reported here for schools. Take note that the key problems reported in schools could not be addressed merely by effective source control. In addition, effective (and quiet) ventilation systems and proper maintenance are reported to be of importance.

Response: Our report did not indicate that source control would handle all indoor air problems. The report indicates throughout that ventilation, public education, and other measures are all part of an effective, comprehensive program to reduce indoor pollution. The text has been edited to emphasize this. Also, regarding schools, one of the most important problems identified was the elevated levels of formaldehyde, which require source control for effective risk reduction.

- (p 22 & 153) This sentence, which appears in both the executive summary and in the main report should be reconsidered: "The approach used to reduce toxic air contaminants in ambient air, in which source emissions are reduced without setting enforceable air quality levels, seems most applicable to indoor air." his statement seems to reflect an ARB-centered perspective in which only policy tools that have been developed for ambient air pollution control can be employed. The indoor environment shares some attributes with ambient air pollution, but it is sufficiently distinct to invite fundamental rethinking of policy approaches, rather than an effort to find the best fit from the current portfolio.

  Response: This statement was intended to differentiate from an approach more like the criteria standard-setting approach, in which strict standards are established and must be met. Instead, our TACs program allows identification of exposures and risks, with mitigation based on best available technology, not meeting a pre-set standard. Thus, as technology improves, additional reductions can be implemented.

Other Points

Methodological issues: When discussing mortality impacts, it is a bit misleading to sum simple deaths across diseases and risk factors. The disturbing issue about exposures to pollution or other risk factors, of course, is actually the premature mortality they cause. Thus, at the least, the term "premature" morality (or deaths) should be the wording.

Response: Text was revised.

As everyone dies, however, the degree of prematurity is critical, which is why lost-life-year measures are becoming more widely used. It would not seem possible that ARB can change the favored metric in one report, but it would seem worthwhile to offer a lost-life-year evaluation in parallel to the simple mortality tabulation, which requires knowledge/ assumption about the age distribution of the premature mortality. Even better, of course, would be a combined measure (QALY, DALY, etc) that combines lost years due to premature mortality with those due to illness/injury. The committee recognizes that doing so is probably beyond the resources and time available for this report, however.

Response: Comment noted. However, adding such an approach is beyond the time and resources available for this report.

There seems to be no discussion of "counterfactual" (CF) values in the assessments, but rather an implication that the entire pollutant level is up for grabs. Is it a reasonable

assumption that it would be possible to achieve zero concentrations of pollutants indoors with any conceivable set of interventions? We think not as there is no indoor environment in the world that has ever achieved it, bar perhaps Level 5 laboratories or some such. Just as it is inaccurate and misleading to account 100% of outdoor concentrations to "air pollution", so it is for indoor pollution (even not accounting for the outdoor pollution going indoors). The WHO Outdoor Air Comparative Risk Assessment (CRA), for example, used 7.5 $\mu g/m^3$ PM2.5 as the counterfactual level for outdoor air pollution (Cohen et al., 2004). It also did a sensitivity analysis that showed that changing the CF up or down has a big effect on the resulting overall estimate of burden, as you might expect. Zero, however, would be clearly wrong and misleading.)

The best CF to use would be one actually demonstrated in real interventions, preferably determined as a shift in the exposure distribution as it would be in the real world (also done in the CRA for some risk factors). In lieu of that, you could take the current 10% level or some such as the CF level with the assumption that it would be possible to reach that level with feasible interventions.

> **Response**: We did not mean to imply that risk can be reduced 100%. Clearly, the level of a pollutant outdoors will limit the levels that can be achieved indoors, in many cases. We have added some information reflective of a "counterfactual", such as the estimate of the remaining risk if indoor levels of radon were decreased to the ambient level, and will add additional information in the final report.

Section 2.1 The Key Health Impacts identified are asthma and cancer, and both are in fact very important; two others are cited, irritant effects and sick building syndrome (which is not itself a health effect). However, there are some other very important health impacts that should be carefully considered: heart disease, reproductive effects and diseases of the immune and nervous systems. Asthma and heart disease deserve more attention, given the large numbers of people affected, and therefore the public health impact of these diseases. Where there is uncertainty about the effects of indoor air pollutants on these other diseases, but some evidence exists, the uncertainty should be acknowledged.

> **Response**: Additional information was other impacts was added to the text.

The committee is also worried about Section 2.1.2, which seems to conflate chemicals with quite different levels of IARC carcinogenic status (table 2.4, p. 38) without noting the uncertainty in doing so.

> **Response**: Table 2.4 is simply to inform the reader regarding the IARC status of some common indoor air pollutants.

Mercury: It has been shown that the concentration can reach nearly 20 mg/m^3 indoors where liquid mercury has been spilled, and the Threshold Limit Value is 0.025 mg/m^3 for elemental mercury. Clearly seriously high levels of mercury are possible if elemental mercury is left in place, e.g., from a broken thermometer.

> **Response**: This is an accidental situation, and is currently being addressed through a program in the state to collect old mercury thermometers.

Risk perception: You may want to explore at bit more the anomaly that we heavily control hazardous waste dumps/leaking gas tanks/etc. and yet allow the very same chemicals almost free rein inside our houses within easy access of our young children, and so on. There is a natural tendency to be more concerned with uncontrolled "waste" but we are fooling ourselves if we act as if the material in our house is really under our control or somehow safer because we are still using it. This stretches a bit past IAQ per se, but is an important aspect of the risk perceptions related to it

> **Response**: We agree with the statement and the public's (and decisionmakers') lack of information on indoor air quality or their perception that it does not present a problem. However, public concern is growing as the public becomes more aware of the problem. This is evident in the number of public phone calls we receive from those seeking information, and the large and increasing number of visits to our indoor air quality website.

A double solidus should be avoided, as it can be ambiguous. Instead of $g/m^2/h$, for example, use $g/(m^2\text{-}h)$, which has no ambiguity. (One could use $g\ m^{-2}\ h^{-1}$, of course, but this might be too technical-looking for the intended audience.)

> **Response:** The representation of units was not changed. We felt this would be more understandable by the lay audience.

The word "data" is used as plural in some places and as singular in others.

> **Response:** We try to treat "data" as a plural term, and will edit the final version carefully.

Minor comments on Executive Summary

- Page 1, 1st paragraph. Not clear what is meant by "will be considered by the California Air Resources Board."

  > **Response**: The report will be discussed by our Board at their March meeting, to receive Board member comments before it will be finalized and sent to the Legislature.

- Page 1, final paragraph. Rather than "much less diluted," it is better to say that indoor emissions are "diluted much more slowly". (Also, P 28, first full paragraph.)

  > **Response:** Change was made.

- Page 1, final paragraph. Smith's "rule of 1000" has been substantiated by other investigators. The point made has much broader support than "one investigator has calculated." Sample references: Bennett et al., 2002; Lai et al., 2000. (Also, P 28, middle.)

  > **Response:** Additional support by other investigators was noted.

- Page 2, Figure ES-1 (and elsewhere). Please provide references for figures and tables that display published data.

  Response: Some notations were added to figures, and more will be cleaned up for the final version.

- Page 3, Table ES-1. Another major source of carbon monoxide is attached garages. Another major source of "formaldehyde and other aldehydes" is environmental tobacco smoke.

  Response: Suggestions were added to table.

- Page 5, Figure ES-2. Apparently the basis for this figure is detailed in the main report. The executive summary should point to the main body of the report where the estimates are developed.

  Response: So noted.

- Page 5, middle paragraph. Better to refer to "radium-containing" rock and soil as the source of radon. (Uranium is the ultimate progenitor, but geochemistry can cause separation between uranium and radium, the immediate parent.)

  Response: Some changes were made, but text remains uranium in some places for ease of understanding by lay readers.

- Page 8, first paragraph. Worth mentioning here that ozone produces PM as a byproduct as well.

  Response: PM was added to text.

- Page 9, final paragraph. To the best of my knowledge, swamp coolers are relatively uncommon in California. Better to focus on the main ventilation processes: infiltration, natural ventilation, and flow induced by fans (central air, and local exhaust).

  Response: Swamp coolers are fairly common in some parts of the state. An ARB-sponsored study specifically identified swamp coolers as a factor in moving the outdoor air (and ozone) to the indoors. Swamp coolers have also been associated with increased levels of dust mite allergen in homes.

- Page 10, final paragraph. Specify a time period when referring to IAQ costs as "potentially in the billions of dollars" (per year?).

  Response: Yes, per year. Will clarify here and throughout the report in the final version.

- Page 12, first paragraph under V. Should point out that workplace standards also don't apply to where children spend most of their time.

Response: Workplace standards in California do apply in schools and in publicly operated day care centers. They of course do not apply in homes, where children spend most of their time.

- Page 13. Proposition 65 is not mentioned in this list, although it seems that it has had a salutary effect on air pollutant exposure associated with certain consumer products, such as the elimination of TCE from "White-Out."

  Response: Prop 65 is discussed later in the report. We agree that is can have substantial positive impacts on indoor air, although it was not intended to address indoor air.

- Page 15, "reduction at the source". Control through source reduction is not so simple as reducing total mass emission rates. Toxicity can vary by orders of magnitude among species, and so the toxicity of emissions needs to be factored into any source-control strategy.

  Response: We agree. We would add that the percent of the population affected and the typical duration of exposure are also important considerations in determining an appropriate source control strategy.

- Page 16, "air cleaning devices". The discussion in the executive summary could also acknowledge the potential for future improvements in air cleaner technology (as discussed on p. 155 (ICAT for IAQ).

  Response: We will add a statement to that section in the final version of the report. Mitigation option no. 9 (indoor ICAT) includes a statement regarding the need for effective, low noise air cleaners.

- Page 17. Groundwater is also an important source of radon. Building materials are potential sources when they contain elevated levels of radium (not "radon gas").

  Response: Both suggestions were included in the tables.

- Page 18, first paragraph. Meaning of phrase is unclear: "the gap in reducing exposure and risk from categories of indoor sources."

  Response: That phrasing was deleted.

- Page 19, first paragraph. Contrary to what is stated, disease transmission can certainly occur because infectious agents are "emitted into the indoor environment per se."

  Response: Text has been corrected.

- Page 23, point 8. Good to mention the importance of indoor chemical reactions, in particular the importance of pollutant-surface interactions as an area in need of further study (e.g. ozone-carpet).

Response: We agree that pollutant surface interactions are an aspect of indoor chemistry that needs further research. However, we believe that is a fine point not appropriate for the executive summary.

Minor comments, Chapter 1

- Page 27, middle. There are not "several journals" that are "devoted exclusively" to the field of indoor air quality. Only one is first-rate: *Indoor Air.* Other journals carry IAQ articles, but are not devoted to it.

  Response: Text was adjusted accordingly.

- Page 29, bottom. Careful: Children do not "inhale a greater quantity" in an absolute sense (although they do per unit body weight).

  Response: Text indicates their increased inhalation is relative to body weight.

- Page 30, "children's activities". Reference group is unclear for "younger children spend more time near indoor sources...."

  Response: We will recheck and clarify in the final version.

Minor comments, Chapter 2

- Page 32, second sentence. Not clear what the antecedent is of "some."

  Response: Corrected.

- Page 32, third paragraph. Not clear what the basis is for the statement "only a fraction of indoor pollutants have been identified."

  Response: Clarification made.

- p. 32, para 3, end. This is quite misleading. With our growing analytic capability we are approaching a stage where we can essentially measure just about anything just about everywhere. This is not an indication of concern, however, for it is dose and toxicity that drive risk, not occurrence.

  Response: Indeed dose and toxicity drive risk and that point is made elsewhere in Chapter 2. However, to the extent that any toxic air contaminant is introduced into an indoor environment, especially one with children present, it provides the potential for exposure, and contributes to whatever burden may already exist in that environment.

- 34, line 3: asthma increase called "tremendous" before any discussion of it.

  Response: "Tremendous" was removed.

- p. 34, para 2: Careful, increase in simple asthma prevalence does not track directly to increases in asthma burden, which is driven by severe asthma attacks and deaths.

  Response: We remain concerned about the rise in asthma prevalence, however, for all of the reasons discussed.

- Page 39. On the theme of ETS exposure, it may be worth noting the estimates that have been made of population intake to specific hazardous air pollutants from residential ETS (Nazaroff and Singer, 2004).

  Response: Such information was added to the ETS discussion in section 2.3.3.1.

- Page 40. Not sure that isoprene does not react rapidly with ozone, in comparison with air-exchange rates. (See Atkinson and Arey, 2003, for confirmation.)

  Response: The mention of isoprene relates to the work of Wilkins et al., (2001) who studied its reactive chemistry.

- 40, last line: What terpene?

  Response: The point is that at room concentrations the reactivity products are probably more irritating than the precursors. The 'terpene' refers to those generally studied, pinene, limonene, etc.

- Page 41. In addition to forming formaldehyde, and secondary PM, the ozone-terpene reaction system generates the OH radical, which is a major story line for indoor air chemistry. This was an important aspect of the Fan et al. (2003) paper. The seminal paper on this topic is Weschler and Shields (1996).

  Response: Comment noted.

- p. 42, para 2, end: need citations here.

  Response: Several citations were added.

- Page 42, bottom. Worth noting is another key difference between indoor PM and outdoor PM: age of aerosol. Particles with organic content often tend to be emitted with the carbon in a chemically reduced form. As particles age in the atmosphere, the carbon is slowly oxidized. This changes the polarity and water solubility of the organic surface of the particle and could conceivably affect the toxicity. In this way, a typical indoor combustion particle might be quite different than a typical outdoor combustion particle.

  Response: Good point, will incorporate in final revision.

- Page 45, lower third. The statement that NAAQS for PM are "often exceeded in California's indoor environments" is not well supported by evidence presented. If true, then the current empirical basis is limited, since there are not too many

indoor PM measurements in California. (The PTEAM study is a noteworthy exception to this statement; however, that is a special case since the measurements were made during the autumn in Riverside, conditions that would tend to produce higher indoor PM levels than typical for California as a whole.)

Response: The studies shown in Table 2.5 for California do support that statement. All of the Los Angeles studies of indoor PM2.5 show indoor averages that exceed the California standard for PM2.5, annual average of 12 $\mu g/m^3$. Similarly, the indoor averages of all of the studies of PM10 conducted in Los Angeles (not just PTEAM) exceed the state PM10 annual standard of 20 $\mu g/m^3$. Finally, as indicated in the statement, homes with indoor sources would be especially likely to exceed the standards, based on the very high indoor PM emissions measured in the studies discussed in section 2.2.2.

- Page 45, §2.2.2. Is this line justifiable from existing data? "Indoor PM concentrations are typically equal to or higher than concurrently measured outdoor levels." Evidence suggests that residential PM levels are comparable between outdoors and indoors (lower without smoking; higher with smoking), but that in commercial buildings w/o smoking, indoor PM levels are systematically lower than those outdoors.

  Response: See response in earlier responses on PM.

- Page 46, first full paragraph. There are a few recent papers on indoor particles of outdoor origin that provide a stronger basis for the discussion than those papers cited here. See, for example, Riley et al., 2002; and Ott et al., 2000.

  Response: We will add these citations in the final version of the report.

- Page 46, first full paragraph. The discussion of residential ventilation here is a bit skewed. Dominating are infiltration, natural ventilation (windows), and mechanical flow induced by central air systems, exhaust fans, and vented combustion devices (e.g., fireplaces). Swamp coolers and whole house fans are rather less common (although perhaps not in Riverside).

  Response: There are few fireplaces in southern California. Swamp coolers and whole house fans are listed as examples of mechanical ventilation devices in homes that would influence indoor-outdoor relationships.

- Page 46 and elsewhere. No mention is made of the degree to which pollutants penetrate from outdoors to indoors along with infiltration air. Several papers have been published on this topic since the mid 1990s, among them being Liu and Nazaroff (2001).

  Response: Information will be added.

- Page 46, 2nd full paragraph. A thorough review of the role of ETS as a source of indoor PM can be found in Nazaroff and Klepeis (2004).

  Response: Thank you. Citation will be added.

- Page 55. Missing from the discussion of nitrogen oxides is the potentially important chemistry involving the nitrate radical (NO_3), which would be formed indoors whenever NO_2 and O_3 are found together. See Weschler (2004).

  Response: Revisions will be added to the text.

- Page 56, §2.2.6. Reference to "swamp coolers" seems misplaced here, as it would seem to be a relatively uncommon configuration for the California housing stock.

  Response: The number of homes with swamp coolers is not insignificant, and the Energy Commission previously encouraged their use, because they are more energy efficient than refrigerant coolers. Also, in the USC study Children's Health Microenvironmental Study, Steve Colome found that homes with swamp coolers in use had ozone nearly as high as outdoor levels on high ozone days.

- Page 57. Suggest updating the reference of Weschler et al. 1989 to Weschler, 2000.

  Response: We will update this and others in the final version of the report.

- Page 57, last paragraph. Ozone from any source can react with indoor surfaces (not only ozone from ozone generators), including carpets (Weschler et al., 1992, as cited; and Morrison and Nazaroff, 2002).

  Response: Will clarify in final version.

- Page 60, first bullet in 2nd list. Should not equate "emissions" with "concentrations." What does it mean to have "carpet emissions were generally below the limit of detection of 1 $\mu g/m^3$?"

  Response: Correction made.

- p. 65, para 2: this seems to have about the only mention of attached garages, which we understood to actually be a major source of exposure. Is this not so?

  Response: A section has been added on attached garages.

- Page 72, first paragraph. The result cited from Gilpin et al. (2001) is reported here differently than on the next page. Here it says that 78% of households with children didn't permit indoor smoking in 2001. (Implication: 22% of all households with children permit indoor smoking.) On the next page, it says that 22% of *smoking parents* still allowed smoking inside the home.

  Response: Will be corrected in the final version.

- Page 72, bottom. Substantially more detail on ETS emission factors of toxic air contaminants is now available in the papers by Singer et al. (2002, 2003).

Response: Thank you.

- 72, para 3: Not clear why there is a discussion in this report about active smoking in pregnant women. Will lead to confusion.

  Response: The one sentence about smoking during pregnancy serves to illustrate health effects associated with ETS.

- p. 74, Table 2.8: what are the averaging times?

  Response: Nicotine and RSP concentrations in this table represent different averaging times because they are from multiple studies. The ETS document prepared by ARB to identify ETS as a TAC has a full summary of the averaging times.

- Page 75, first bullet. The statement that "colds are more often transmitted by direct contact" should be supported with a reference. My understanding is that the mode of transmission of rhinovirus is largely unknown.

  Response: Text was changed.

- Page 85, §2.3.6.2. Should also mention candles with metal wicks as a source of lead exposure. (Van Alphen, 1999)

  Response: This was added to the report.

- Page 88, §2.3.8. Cooking (hot oils) should also be mentioned as a potentially important source of PAHs (it is in §2.3.8.2, but not in the opening paragraph). (Reference: Siegmann and Sattler, 1996).

  Response: Cooking oils were added to opening paragraph. Citation was included in source discussion.

- Page 91, Asbestos: This section should first explain the relevance of asbestos fibers >5 microns long. Explain the standards and the restriction to biologically relevant fibers. Keep the units constant, e.g., fibers/ml or per m^3.

  Response: Text was modified to discuss the impact of fiber length and the use of fiber length for standard setting. The units are the same as those used by the authors of various papers.

- Page 93 Health effects of PBDEs: Even if NOELs are not available, give LOELs. Again, some context is needed to understand other exposures.

  Response: Additional information will be added to the final version of the report. A statement from Rudel et al. (2003) was added in the concentration section that indicates 15 compounds measured in her study exceeded guideline levels. Dr. Rudel also notes that existing guidelines do not consider endocrine effects.

- Page 94 Sources of PBDE: Should explain why PBDE is added to these materials (fire retardant). PBDE Concentrations: Concentrations should be given here, and contextualized with health data/standards/health studies.

  Response: Text has been added to the source section to indicate PBDEs are fire retardants. Concentration data were added and contextualized with a statement about guideline levels.

Minor comments, Chapter 3

- Page 98, §3.1.1. Why exclude CO poisoning deaths from motor vehicle emissions, as many of these occur in indoor spaces? See Marr et al., 1998.

  Response: See previous response.

- Page 98, middle. A better reference (than CPSC, 1997) to the recent state of accidental CO poisoning deaths nationwide is Mott et al., 2002.

  Response: Thank you, we will incorporate this.

- Page 100, middle. "In addition, cigarette consumption by California adults was found to be about half of the US average...." On what basis? Per smoker? Per adult?

  Response: We will clarify in the final version.

- Page 106-107. Where is this statement substantiated?: "with proper measures, it is probably feasible to eliminate at least 50% of the particle exposures that contribute to asthma exacerbation, and likely more."

  Response: See earlier response; however, this statement will be removed.

- Page 107. At a few points in this discussion, it is suggested that the 2% productivity reduction owing to SBS is "conservative." Is the evidence sufficiently strong to conclude whether this is a conservative estimate or not.

  Response: The estimate was based on a mid-point value, so it is a conservative estimate in that context. Subsequent studies (summarized in Wyon 2004) suggest that even greater worker performance improvements, on the order of 5-10%, are achievable. This more recent information has been included in the text.

- Page 110. The last sentence on this page seems speculative, especially regarding IQ effects. Improved IAQ in schools will lead to higher intelligence quotients among students?!

  Response: Yes, the sentence does refer to "potential benefits." There are studies that indicate students have higher test scores when in more comfortable environmental conditions. Lead exposure, which would be reduced by improved

building maintenance and dust control in schools and homes with lead present, has specifically been associated with decrements in children's IQ's.

Minor comments, Chapter 4

- Page 111. The limitations of workplace regulations are more severe than those listed. They do not apply to indoor environments that are not workplaces. Also, they have been developed from an industrial hygiene perspective in which one or a few key chemicals dominate exposure. They seem entirely ill designed to serve as a basis for evaluating the health and comfort risks posed by the complex mixtures found in, e.g., modern office buildings.

  Response: The second and third paragraphs in this section discuss the shortcomings of Cal/OSHA regulations relative to full protection of workers.

- Page 111-112. I'm pleased to see the discussion of Prop 65 and the new EPA radon in water regulations in this section. (Have the radon in water regs progressed since 2000; update?)

  Response: The regulations have not been promulgated yet.

- §4.3 Emission Limits. Much of this section is far from the issue indoor air pollution (e.g. the discussion of ARB's consumer products and architectural coatings programs).

  Response: These two ARB programs do limit emissions from products. They serve as examples of how the agency can work with industry to derive products with less adverse environmental impacts. Additionally, ARB has restricted the use of several TACs in certain types of products.

- Page 126, first full paragraph. This sentence doesn't make sense as written: "emissions from a single material or product cannot exceed one half the chronic REL." Emissions would be expressed in mass per time. The REL is expressed in mass per volume. They cannot be directly compared.

  Response: Text was changed to indicate the emissions are put through a modeling process to estimate a room concentration.

- Page 127, §4.3.3.3. This statement requires substantiation: "These programs have been successful in reducing emissions from their products over the last few decades."

  Response: Text was changed.

- Page 128, bottom. As in comment 64, the discussion of the GEI emissions criteria mixes measures. The implication is that emissions are to be limited so that individual VOCs "must meet the criteria of less than 1/10th of the threshold limit values...." In addition to the problem of equating emissions to concentrations, this also seems like an ineffective criterion for ensuring good

indoor air quality. (An indoor environment in which multiple chemicals approached 1/10th of their respective TLVs would not be healthful for general occupancy.)

Response: Text was changed.

- Page 132, §4.4.1.3. Weatherization and duct sealing would affect not only indoor levels produced by combustion appliances, but from other sources, too.
  Response: text was changed.

Minor comments, Summary

- Page 160, near bottom. The underlying reason for the "rule of 1000" is not because of the factors cited, but rather because the rate of ventilation provided to buildings per occupant is about 1000 times less than the amount of wind-supplied "ventilation" to an urban area, per inhabitant.

  Response: Text has been changed to reflect this correction.

Additional references that might be used in the report: most cited above.

Apte, M.G., Gundel, L.A., Dod, R.L., Russell, M.L., Singer, B.C., Sohn, M.D., Sullivan, D.P., Chang, G-M, and Sextro, R.G., Indoor Measurements of Environmental Tobacco Smoke, Final Report to the Tobacco Related Disease Research Program, Lawrence Berkeley National Lab report LBNL-49148, Berkeley, CA, March 2004.

Atkinson R., Arey J. Gas-phase tropospheric chemistry of biogenic volatile organic compounds: A review. *Atmospheric Environment* **37 (supplement 2)**, 197-219, 2003.

Bennett D.H., McKone T.E., Evans J.S., Nazaroff W.W., Margni, M.D., Jolliet O., Smith K.R., Defining intake fraction, *Environmental Science & Technology* **36**, 206A-211A, 2002.

Bornehag CG, Sundell J, Bonini S, Custovic A, Malmberg P, Skerfving S, Sigsgaard T, Verhoeff A, Dampness in buildings as a risk factor for health effects, EUROEXPO: a multidisciplinary review of the literature (1998-2000) on dampness and mite exposure in buildings and health effects, *Indoor Air* **14**, 243-257, 2004.

Carslaw N., New directions: Where next with indoor air measurements? *Atmospheric Environment* **37**, 5645-5646, 2003.

Cohen AJ, Anderson HR, Ostro B, Pandey KD, Krzyzanowski M, Kuenzli N, Gutschmidt K, Pope CA, Romieu I, Samet JM, Smith KR, *Mortality Impacts of Urban Air Pollution, in* Ezzati M, Rodgers AD, Lopez AD, Murray CJL (eds) Comparative Quantification of Health Risks: Global and Regional Burden of Disease due to Selected Major Risk Factors, Geneva: World Health Organization, Vol 2 of 3, pp.1353-1433, 2004.

Eskenazi, B., Gold, E.B., Lasley, B.L., Samuels, S., Hammond, S.K., Wight, S., Rasor, M.O'N., Hines, C.J., Schenker, M.B. Prospective monitoring of early fetal loss and clinical spontaneous abortion among female semiconductor workers. *American Journal of Industrial Medicine*, **28**:833-846, 1995.

Hammond, S.K., Hines, C.J., Hallock, M.F., Woskie, S.R., Kenyon, E., and Schenker, M.B. Glycol ether exposures in the semiconductor industry. *Occupational Hygiene* **2**:355-366, 1996.

Lai A.C.K., Thatcher T.L., and Nazaroff W.W., Inhalation transfer factors for air pollution health-risk assessments, *Journal of the Air & Waste Management Association* **50**, 1688-1699, 2000.

Liu DL, Nazaroff WW, Modeling pollutant penetration across building envelopes, *Atmospheric Environment* **35**, 4451-4462, 2001.

Marr L.C., Morrison G.C., Nazaroff W.W., and Harley R.A., Reducing the risk of accidental death due to vehicle-related carbon monoxide poisoning, *Journal of the Air and Waste Management Association*, **48**, 899-906, 1998.

Mendell MJ, Fisk WJ, Kreiss K, Levin H, Alexander D, Cain WS, Girman JR, Hines CJ, Jensen PA, Milton DK, Rexroat LP, Wallingford KM, Improving the health of workers in indoor environments: Priority research needs for a national occupational research agenda, *American Journal of Public Health* **92**, 1430-1440, 2002.

Morrison G.C., and Nazaroff W.W., Ozone interactions with carpet: Secondary emissions of aldehydes, *Environmental Science & Technology*, **36**, 2185-2192, 2002.

Mott J.A., Wolfe M.I., Alverson C.J., et al., National vehicle emissions policies and practices and declining US carbon monoxide-related mortality, *JAMA* **288**, 988-995, 2002.

Myatt TA, Johnston SL, Zuo ZF, Wand M, Kebadze T, Rudnick S, Milton DK, Detection of airborne rhinovirus and its relation to outdoor air supply in office environments, *American Journal of Respiratory and Critical Care Medicine* **169**, 1187-1190, 2004.

Nazaroff W.W. and Singer B.C., Inhalation of hazardous air pollutants from environmental tobacco smoke in US residences, *Journal of Exposure Analysis and Environmental Epidemiology*, **14**, S71-S77, 2004.

Nazaroff W.W., and Klepeis N.E., Environmental tobacco smoke particles, *Indoor Environment: Airborne Particles and Settled Dust*, Morawska L. and Salthammer T. (Eds.), Wiley-VCH, Weinheim, Germany, 2004, pp. 245-274.

Ott W, Wallace L, Mage D, Predicting particulate (PM_{10}) personal exposure distributions using a random component superposition statistical model, *Journal of the Air & Waste Management Association* **50**, 1390-1406, 2000.

Riley W.J., McKone T.E., Lai A.C.K., and Nazaroff W.W., Indoor particulate matter of outdoor origin: Importance of size-dependent removal mechanisms, *Environmental Science & Technology*, **36**, 200-207, 2002. [See errata on p. 1868.]

Seppanen O, Fisk WJ, Association of ventilation system type with SBS symptoms in office workers, *Indoor Air* **12**, 98-112, 2002.

Siegmann K. and Sattler K, Aerosol from hot cooking oil, a possible health hazard, *Journal of Aerosol Science* **27 suppl. 1**, S493-S494, 1996.

Singer B.C., Hodgson A.T., Guevarra K.S., Hawley E.L., and Nazaroff W.W., Gas-phase organics in environmental tobacco smoke: 1. Effects of smoking rate, ventilation, and furnishing level on emission factors, *Environmental Science & Technology*, **36**, 846-853, 2002.

Singer B.C., Hodgson A.T. and Nazaroff W.W., Gas-phase organics in environmental tobacco smoke: 2. Exposure-relevant emission factors and indirect exposures from habitual smoking, *Atmospheric Environment*, **37**, 5551-5561, 2003.

Swan, S.H., Beaumont, J. Hammond, S.K., Von Behren, J., Green, R.S., Hallock, M., Woskie, S.R., Hines, C.J., Schenker, M.B. Historical cohort study of spontaneous

abortion among fabrication workers in the semiconductor health study: Agent-specific analysis. *American Journal of Industrial Medicine*, **28**:751-769, 1995.

Van Alphen M. Emission testing and inhalational exposure-based risk assessment for candles having Pb metal wick cores, *The Science of the Total Environment* 244, 53-65, 1999.

Wargocki P, Sundell J, Bischof W, Brundrett G, Fanger PO, Gyntelberg F, Hanssen SO, Harrison P, Pickering A, Seppanen O, Wouters P, Ventilation and health in non-industrial indoor environments: report from a European Multidisciplinary Scientific Consensus Meeting (EUROVEN), *Indoor Air* **12**, 113-128, 2002.

Weschler C.J. and Shields H.C., Production of the hydroxyl radical in indoor air, *Environmental Science & Technology* **30**, 3250-3258, 1996.

Weschler C.J., Chemical reactions among indoor pollutants: What we've learned in the new millennium, *Indoor Air* **14 (Suppl 7)**, 184-194, 2004.

APPENDIX V

RESPONSES TO PUBLIC COMMENTS on the NOVEMBER 2004 REPORT

We appreciated the comments received on the November 2004 draft report. They have helped further refine the content and strength of the report.

ACCESS BUSINESS GROUP

Comment: ARB continues to cite consumer products as a source of TACs without considering the abundance of evidence obtained in the VOC reduction program for ambient air quality. Exposure of consumers to TACs is unlikely due to a variety of US product safety regulations and voluntary efforts of manufacturers. ARB has focused reformulation on the highest use products.

> **Response**: *Based on information available, the report accurately characterizes sources* of these compounds and uses caveats such as "at the time the study was conducted". "Consumer products" is a broad term, and, as noted in the comment, not all types of consumer products are regulated by ARB. Additionally, the U.S. product safety regulations referred to are generally labeling requirements or other requirements that do not require removal of potential TACs from the product.
>
> We acknowledge in the report the achievements at eliminating TACs in consumer products. However, accessing the ARB database of solvents that can be used in consumer products, we find well over 100 solvents of varying properties and health effects are listed in the database. The database also indicates the mass emissions of various VOCs from consumer and commercial products. Data indicate that tons of VOCs are released on a daily basis, with spray hair styling products being the highest emitter of pounds of VOCs.

Comment: Table ES-1 is a confusing collection of data. Endocrine disruptors are listed as pollutants without much evidence that they are found in indoor air. DDT has been discontinued as a chemical in US commerce for many years. "Organic chemicals" seems misleading since most of the products listed do not contain the listed pollutants.

> **Response**: Table ES-1 is designed to give the reader a quick overview of the many categories of indoor air pollutants. ARB believes it has highlighted the categories of pollutants most often discussed and studied in the scientific literature. To meet the intended purpose of the table, pollutants, sources, and potential health effects have been grouped together. A footnote was previously added that explains that each pollutant may not cause all of the health effects listed in the third column. A good summary of concentrations of endocrine disruptors in indoor air and on house dust is provided by Rudel et al. (2003). Although the sale of DDT was prohibited many years ago, it is detected in house dust in virtually every study that analyzes for it. It is extremely persistent in the environment.

Comment: The listing of personal care products and cosmetics as a source of formaldehyde is speculative. Such use has largely been discontinued. When it was present, emissions were trivial.

> **Response**: Fingernail products were very high emitters of formaldehyde in the Kelly et al study of formaldehyde emissions funded by the ARB and completed in 1996. In an unpublished study of products purchased from California retail stores, Akland and Whitaker (2000) found formaldehyde in several fingernail products and a few other personal care products as well. Emissions were not necessarily trivial, and most importantly, personal exposures of the users of the products clearly could be quite high.

Comment: The report states, "Low emission product designs or reformulations can usually be accomplished by the manufacturer, with minimal impact on the consumer, often with only minor increased costs." This is not likely to be true if further reductions in consumer product emissions above those already achieved in the ambient air VOC emissions reductions. Further reductions at each phase of regulations have been more difficult and more costly.

> **Response**: We agree that as regulated products are further regulated, the incremental reductions are more costly per unit of pollutant reduction. However, ARB regulates only a limited number of consumer product categories; there is room for emissions reductions in categories that are not currently regulated. There may also be opportunity for further reductions in building materials and appliances.

Comment: In Section 1.4 "Environmental Justice Considerations Relative to Indoor Air Quality", the citation of pesticide residues in carpeting and room freshener use seem unnecessarily critical. It seems appropriate to encourage those who live in substandard housing to use appropriate consumer products to deal with pests and malodors.

> **Response**: Data relating indoor air quality to environmental justice issues are scarce. The two citations provided on pesticide and air freshener use are appropriate to include in this section. The Department of Health Services and private organizations participate in asthma intervention programs to educate low-income individuals to improve their environment by reducing asthma triggers. Proper use of cleaning products is undoubtedly part of the educational curriculum. However, preventive measures are preferred over pesticide use.

Comment: Section 2.3.3, "Volatile Organic Compounds", seems confusing as VOCs are cited when specific toxic or irritant VOCs are intended. Most VOCs have no adverse health effects at the levels found in indoor air.

> **Response**: VOCs studied by the scientific community in indoor air are reported in this section. In many cases indoor concentrations are below health benchmark levels. However, studies indicate that those homes at the high end of distributions may experience unsafe levels of specific VOCs. Additionally, those with carcinogenic potential are sometimes elevated above one in a million or one in a hundred thousand risk levels. .

Comment: The section on VOC emissions from consumer products has many examples of misinformation. For example, chloroform is cited as a consequence of laundry and dish washing when neither would be a likely source, absent chlorine bleaching. Instead the use of municipally treated water is the likely cause of ambient chloroform. ARB has the opportunity to use the

survey information on VOC from the consumer products ambient air program to correct these errors.

> **Response**: The release of chloroform from the use of chlorine-containing cleaning agents during wash cycles does indeed occur and the report provides appropriate citations. Prior to discussing this source, the report discusses the release of chloroform from treated domestic water, particularly during showers. Additional citations will be added to the report.

AMERICAN CHEMISTRY COUNCIL, PHTHALATE ESTERS PANEL

ARB Comment regarding phthalate esters: The Office of Environmental Health Hazard Assessment, reviews and assesses the toxicity of chemicals and conducts risk assessments. Questions related to the toxicity of DEHP and other phthalate esters have been referred to OEHHA for response. OEHHA has not been able to respond to all of ACC's detailed comments on phthalates. Additionally, many of those detailed comments go beyond the scope of this report. ARB has corrected the IARC classification of DEHP and made other edits accordingly. Responses to some key comments are provided here. Following OEHHA's response to other comments, the final report will be revised as appropriate.

Comment: The report is technically correct in stating that DEHP is in IARC Group 3, not classifiable as to human carcinogenicity. The summary fails to present the heart of the IARC's conclusion – that "the mechanism by which di(2-ethylhexyl phthalate increases the incidence of hepatocellular tumours in rats and mice is not relevant to humans" (IARC, 2000). Suggested alternate wording for the report is provided.

> **Response**: OEHHA has not responded to the comment on mechanism. Based on the IARC assessment of phthalates, it is correct to say that there is inadequate evidence of carcinogenicity of DEHP in humans, but that there is sufficient evidence in experimental animals for carcinogenicity. This information is included in the report on pages 38-39.

Comment: The final report should not indicate that other phthalates are carcinogenic. Three pages in the report are identified where it is implied that phthalates are carcinogenic.

> **Response**: The reference to other phthalates as potential carcinogens was removed from Appendix II, but the text of the main report was inadvertently left as before. We will review the final version of the report for those statements and correct as needed. Also, based on the IARC reclassification of DEHP to Group 3, the one case of cancer from DEHP in the Comparative Risk Project was subtracted from the indoor cancer risk estimate, leaving a total of 232, instead of 233, estimated cancers. Because of rounding, this change did not affect the estimate of 230 excess cancers per year used in the main report.

Comment: It is misleading to refer to all phthalates as endocrine disrupters. Comments provided in June indicate the weight of evidence demonstrates that phthalates neither mimic nor interfere with estrogen and androgen; some, but not all, phthalates have been shown to cause lowered testosterone levels when administered to rodents at very high doses.

> **Response**: We will change the wording to reflect this in the final version of the report.

Comment: Measured levels of phthalates in indoor air are far below animal effect levels. A previous ARB response indicates that a complete evaluation of their prevalence in indoor air would be undertaken prior to any recommendations regarding these chemicals. The levels are so low, they are a very low priority, consideration should be given to removing phthalates entirely from the report.

> **Response**: We agree that a complete evaluation would be undertaken prior to any recommendations specific to phthalates. However, because DEHP and other phthalates may act as endocrine disruptors, phthalates should remain in the report.

AMERICAN CHEMISTRY COUNCIL, SOLVENTS COUNCIL

Comment: The draft report still fails to adequately assess potential health risks because it fails to compare indoor air VOC concentrations to relevant health benchmarks. The mere inclusion of benchmarks with no additional discussion to put these benchmarks into perspective does little to help the reader accurately assess the potential risk, if any, posed by exposure to indoor air. The comment then lists five specific chemicals and requests additional discussion of those chemicals relative to the benchmarks identified for them.

- **Response**: The appropriate benchmark comparison for a 1 in 100,000 cancer risk level was provided for VOCs in Table 2.7. Additionally, the benchmarks for formaldehyde are included in the text, and comparison to Figure 2.4 is made simple because the benchmarks are included in the figure. Benchmarks were included in the text for other chemicals as well, as noted in the commentor's comments. When benchmarks are presented in the text, it is assumed the reader can determine if the relevant concentration is above or below the benchmark. We do not believe that the additional detailed discussion requested for each chemical is appropriate for this report. The report also states that there is no level of exposure that is known to be absolutely safe for carcinogens. Thus, for those compounds identified as carcinogens, any elevated concentration above the concurrent outdoor levels is undesirable, and indicates the presence of one or more indoor sources. Whether such sources need to be, or should be, mitigated, depends on many factors.

Comment: The report presents a misleading impression of VOC health risks because it lumps "VOCs" together in a single, undifferentiated group. For example, the reference to "VOCs: cancer" in Table ES-2 should be changed to "Selected Known or Suspected Carcinogens," and a footnote added to explain the cost estimate is the sum of cost estimates derived for a certain number of compounds. The mention that indoor and outdoor air pollution have been identified as contributors to an increase of asthma is overly broad and undefined. More discussion should be included on the Institute of Medicine report regarding pollutants and asthma.

> **Response**: The report reflects that VOCs are comprised of a large number of chemicals. The heading "VOCs: cancer" is explained in the health and cost sections, and is more informative and specific than the heading suggested. Appendix II clearly lists the chemicals that were included in the cancer risk estimate. Tables 2.2 and 2.3 are copied from the Institute of Medicine (IOM) report, with the citation provided and accompanying discussion in the text. If a reader needs to further explore indoor exposures and exacerbation or development of asthma they should read the IOM report.

Comment: The prioritization scheme is improved but still fails to represent accurately the relatively minor risks and costs associated with indoor air VOC exposure. Products should be

listed as "high" priority only if it can be demonstrated that exposures exceed or can be *reasonably anticipated to exceed relevant health benchmarks. Hodgson and Levin* (2003) conclude that "for a very few compounds, such as acrolein and formaldehyde, the evidence based on sensory irritation and chronic toxicity is sufficient to warrant efforts to reduce and otherwise control the sources of these compounds in buildings." A quantitative prioritization is needed to determine whether a "comprehensive program" is required.

> **Response**: The purpose of the report was to provide an overview of sources and health effects of indoor pollutants, list existing regulations and practices for mitigating exposures, and describe options and recommendations to improve indoor air quality. A quantitative prioritization is needed if a comprehensive program is implemented, but that work is outside the scope of this report. The statement by Hodgson and Levin (2003) is based on sensory irritation and chronic toxicity, but does not address carcinogenicity. As mentioned above, for carcinogens, there is no level of exposure that is known to be absolutely safe.

Comment: The report still fails to explain adequately that its estimate of excess cancer cases from indoor air VOCs is very conservative and probably greatly overestimates actual risks, which could be zero. The report should include more detailed discussion of the uncertainties inherent in estimating potential excess cancers and the highly conservative nature of its upper-bound excess cancer estimates. The report should include more discussion of Wallace (1991) which states that the risk may be zero.

> **Response**: The VOC cancer risk estimate is based on risk calculations in the Comparative Risk Project (CCRP), and only the excess cancers for the population estimated to be exposed above a one in 10,000 risk level were counted. The procedures used in the CCRP were peer-reviewed, and represent standard practice in health risk assessment. Additionally, as stated in Appendix II, the CCRP estimate does not include all VOCs and semi-volatile chemicals that are known or suspected carcinogens. For example, PAHs other than B(a)P, and aldehydes other than formaldehyde, are not included, yet there are known indoor sources of those compounds.

Comment: The report does not demonstrate a need for new legislative authority to regulate VOCs. The majority of VOCs, when compared to health benchmarks present little or no health risk. Focusing on VOCs will divert resources away from more hazardous air pollutants, such as ETS and radon.

> **Response**: The report does not focus on VOCs. In the high priority group, only building materials are sources of VOCs. However, as indicated in the report, while indoor VOC levels typically do not exceed non-cancer health benchmarks, a number of them have been measured indoors at levels above their respective cancer risk levels of concern.

ASSOCIATION OF HOME APPLIANCE MANUFACTURERS (AHAM)

Comment: AHAM disagrees with the prioritization of sources. Air cleaners and combustion appliances should not be on the high priority list and vacuum cleaners should not be on the medium priority list.

> **Response**: The focus of the air-cleaners category is on those air cleaners that generate ozone, especially those that generate ozone purposely, to supposedly clean the air

(stated in the table). Vacuum cleaners are included in the group of household appliances and office equipment, although the focus of the group is computers, copy machines, and printers, which can be sources of pollutant emissions. While the group is medium priority, vacuum cleaners were included, since they can sometimes re-suspend substantial amounts of particles. There are some very good vacuum cleaners on the market; vacuum cleaners would be a low priority in this group. The report also recommends the development of innovative clean air technologies for indoor air, which could include improved air cleaner, vacuum cleaner, and combustion appliance technologies.

Comment: AHAM believes there is disagreement regarding the validity of activity pattern study data from the 1980s and 1990s, and recommends further research and discussion with stakeholders to update information on time spent indoors.

Response: We are unaware of studies showing any substantially different results from the early activity pattern studies, in terms of time spent indoors and outdoors. Subsequent to ARB's studies, a large national surveys and local field studies have replicated the general findings on time spent indoors. We believe that the other areas indicated in the report warrant higher priority for research.

Comment: The ARB response to an earlier comment indicates that recommended mitigation options for improved public education and professional training would address the misuse of outdoor cooking appliances. However, the draft report still does not clearly reflect this important point. As another example, the public should be educated to follow industry-recommended cleaning guidelines for humidifiers. Consumers must bear some responsibility for maintaining their appliances.

Response: The text was revised to include more discussion of public and professional education and outreach strategies. However, we believe it is largely industry's responsibility to educate their customers regarding recommended cleaning and use of appliances.

Comment: The report fails to mention, by definition, the combustion of natural gas creates by-products and therefore, zero-emitting gas appliances are not technologically feasible. A recent study by the Gas Research Institute found the ANSI/Z21/83 standards have such a high margin of safety that they do not need to be changed. (Citation provided)

Response: Direct-vented (sealed-combustion) appliances are virtually zero-emitting in terms of indoor emissions. For example, direct-vent gas furnaces and gas fireplaces are very energy efficient, are resistant to backdrafting, and have been used widely for years. Direct vent gas stoves were developed by the Canadian Gas Research Institute in the 1990's but have not been marketed widely. The report will be revised to clarify this topic.

It is not clear that the ANSI/Z21/83 standards would assure healthful levels of indoor NO_2 and aldehydes as well as CO, especially over the lifetime of the appliance with typical maintenance levels.

Comment: Two letters are provided from the Consumer Product Safety Commissions regarding studies conducted at NIST. These studies state that the current ANSI Z21.1 standards for emission contain sufficient safety margins. CPSC concluded that no changes are necessary to

the voluntary safety standards. AHAM believes a full survey of homes and buildings is needed to develop a baseline data set.

Response: While the standards may be sufficient for safety (prevention or death) purposes, we believe they are not sufficient to adequately protect health. The referenced modeling studies address indoor CO, and, in fact, show that the ARB's IAQ guideline and state ambient air quality standard would be exceeded. For example, the 1-hour and 8-hour guideline level would be exceeded in the baking scenarios with smaller kitchen volumes and lower air exchange rates. The medium and lower air exchange rates used in the modeling are not as low as those are for some new or weatherized homes in California. This modeling estimate does not include the contributions of outdoor CO levels, which can be substantial at times. Further, these studies do not address what is typically the limiting factor for combustion appliances: indoor NO_2.

A full statewide survey of gas appliances in homes and other buildings is not necessary. Rather, testing, development, training, and verification are needed to improve the design, operation, and maintenance of combustion appliances and their venting systems.

Comment: AHAM objects to most of the proposals in Section 7, "Options to Mitigate Indoor Air Pollution". Establishing emission limits specifically for California is not justified. Stakeholders participate in standard development groups such as ANSI and ASTM, which are designed to ensure products do not diminish indoor air quality. ARB staff are encouraged to participate with these standards development groups.

Response: We commend those working on ASTM and ANSI standards that include consideration of indoor air quality. However, ANSI and ASTM standards are not based on California housing and environmental factors, and may not adequately protect our citizens' health. Additionally, they are not required to be met by all manufacturers. ARB actively participated in the development of UL/ANSI standards for indoor products in the past, but unfortunately did not result in standards; we will participate in the future as resources allow.

Comment: The report utilizes incorrect data and assumptions. Specifically, it states that there are no industry consensus standards for vacuum cleaners. ASTM International has a technical committee, ASTM Committee F11 for Vacuum Cleaners (established in 1972). This committee has 28 industry consensus standards for vacuum cleaner performance. The committee undertakes new initiatives. Currently under review is a standard for determining the change in room air particle counts as a result of vacuum cleaning.

Response: The upcoming standard for airborne particles appears to be the only standard that addresses IAQ directly. The final report will be revised to incorporate information on any existing performance standards that protect indoor air quality at that time.

Comment: The ASTM F11 committee members undertake new initiatives aimed at developing standards for those performance characteristics not yet covered by the current standards. One of those....is the development of new standards for determining "the change in room air particle counts as a result of vacuum cleaning." The lack of such basic information (about the initiatives) in the report indicates the ARB should work with stakeholders to gather current and accurate information rather than seek authority for emission standards. As an example, ARB should

work with industry to get more reliable data on usage patterns of range hoods in homes before making any recommendation.

> **Response**: We commend the ASTM F11 committee for moving forward in this area. However, development of consensus test methods is different from developing limits that must be met. Such information is not "basic information" on indoor air quality that negates the scientific findings in the report. Regarding the specific example given, there is sufficient information available to conclude that range hood use and design are often inadequate. The ARB activity pattern studies and other studies confirm that range hood use in homes is very limited. Additionally, the ARB residential cooking study and other studies show that range hoods are not always very effective. New and existing information on this topic would be considered in developing a risk reduction strategy.

CalAG, LLC

Comment: CalAg, LLC has developed a patented technology for the production of MDF from California rice straw. The product uses a formaldehyde-free polyurethane resin in the binding process. Panelboard product will be on the market by early 2007. MDF made with agricultural fibers and MDI resin will have no off-gassing and will be a substitute for any application that currently uses wood based MDF. Laminates could be applied for decorative purposes, but would not be needed to reduce off-gassing.

> **Response**: Thank you for providing panelboard products that do not emit formaldehyde for consumers. The report mentions some alternatives currently available, thereby indicating it is feasible to produce low-emitting products. Your products further substantiate that alternatives are available.

CALIFORNIA WOOD INDUSTRY COALITION (by Venable)

Comment: Please explain the Chemical Industry Institute of Toxicology (CIIT) risk assessment from 1999 in the final report. The CIIT report's quantitative results should be expressed in addition to and in distinction to those of OEHHA and its acceptance by other national and international governmental bodies be acknowledged.

> **Response**: In November 2002, ARB and OEHHA denied a petition to formally review the CIIT formaldehyde risk assessment. The petition was based in part on the potency estimate change associated with the CIIT 1999 report. OEHHA determined that the report was a new analysis of old evidence rather than new evidence. OEHHA stated that more information is needed to evaluate the risk assessment model used by CIIT, and that it needs to be peer-reviewed and validated. In the future, if formaldehyde risk assessments are reevaluated, information stemming from this work may be considered.

Comment: Recommend removal of the point estimates of risk, or if used, put them into context by also referencing the Maximum Likelihood Estimate (MLE) expression of the California model and the CIIT results in close proximity. Although statements are included regarding 95% Upper Confidence Limit, they are not carried throughout the report. Appendix II discussion reflects the CRP report as a basis of point estimate of risk, a practice the CRP authors considered Inappropriate. The Appendix does not even mention the fact that a statistical UCL expression was used.

Response: In the CCRP, the cancer potency value used to estimate risk was the 95th percentile value, which is standard practice in cancer risk assessments. The CCRP methods and procedures were reviewed and approved by a peer review committee. However, the full distribution of exposure values were used for the calculation (not a high end value), and only the portion of the population with estimated risk above the 1 in 10,000 level were included in the calculation of estimated cancer cases. Had cases with less than a 1 in 10,000 risk level been included, the estimate would have been much higher. In reviewing available data on formaldehyde and other carcinogens, ARB staff found that the CCRP provided the most robust, California-specific analysis of indoor exposure and risk, and that is why the CCRP estimates were used as a starting point. The formaldehyde estimate was cut in half, as described in Appendix III, based on reductions in emissions quantified by an ARB-funded study that showed that emissions from the most common formaldehyde-emitting products and materials had decreased by 49%. The resulting estimate of 62 excess cancers per year compares very well with the 98 nasopharyngeal cancers per year (average of reported cancers from 1998-2002) reported by the California Department of Health Services' cancer registry. The remaining nasopharyngeal cancer deaths are likely the result of occupational exposures to formaldehyde, which may at times be higher than residential exposures. However, workplace users of formaldehyde typically have the benefit of engineering, administrative, and personal protection controls not used by occupants of new mobile homes, classrooms, or other buildings. Thus, some new building occupants can experience higher exposures to formaldehyde over a longer period of time (more hours per day, and more days per week) than would many workers who use formaldehyde in their workplace.

We will review our discussions further and include additional caveats regarding the uncertainty in any such estimates in the final version of the report. However, we will not remove the formaldehyde estimates provided, because, as explained below, they are relatively robust, and supported by current nasopharyngeal death rates.

Comment: Regarding independent laboratory testing for emissions, it is not practical to test a complex product such as furniture. Pieces vary considerably by size, composition, construction techniques, etc. It would be impossible to develop a test protocol that would capture meaningful information on consumer furnishings, per se.

Response: Multiple protocols may be needed to test all types of furniture, but this is not an impossible task. Some types of furniture have already been tested for emissions. Precedent has been set for emissions testing of modular office panels and furniture for the Capitol East End Project. These pieces were purchased in a very large order and manufactured under controlled conditions. We acknowledge other types of furniture may have more variety in the components and are manufactured under a variety of conditions. This topic has been discussed related to the formaldehyde Air Toxics Control Measure. ARB will pursue continued discussions with industry if the recommendation to develop an emissions testing procedure is implemented.

Comment: We are curious as to why some references to the ongoing ARB ATCM proceeding have been eliminated (pages 26 and 104 of June draft report). The Coalition is proud of the cooperative effort that is underway in working with ARB staff over the last four years.

Response: ARB appreciates the cooperative attitude the composite wood industry has maintained in working on the formaldehyde ATCM. Reference to the effort was removed from the Executive Summary because the ATCM is not yet in effect, and the specific timeline is uncertain. It is still included in the body of the report in Section 4.3.3.1.

Comment: Thank you for adding results of the ARB survey in context of the ATCM proceeding on page 60. The results are of a chamber concentration, and could be mistakenly interpreted as the ambient level in a home. Please clarify.

Response: A statement was added to the report to clarify that chamber concentration does not equal the level in the interior of a home.

Comment: There is one inaccuracy regarding industry standards. The CPA lowered the large chamber emission limits for certain particleboard products, thus they are not identical to HUD standard for mobile homes.

Response: Wording was appropriately adjusted in the report.

CLOROX

Comment: The correlation between asthma and bleach (page 37) is inaccurate. The Rosenman et al study cited in the report lacks scientific rigor.

Response: Authors of the Rosenman et al. (2003) study examined a database of work-related asthma cases associated with cleaning products, reported primarily by physicians between 1993 and 1997. The article discusses limitations of the database: limited documentation of pulmonary function; identification of the causal agent is based on history; and the database contains only a minority of the work-related asthma cases for the study states. However, the paper provides further citations for new-onset work-related asthma occurring after a documented exposure to cleaning products, and more citations for exacerbation of pre-existing asthma from such exposures. The large number of cases reported would indicate an association exists between asthma, bleach, and other cleaning products.

Comment: Cleaning products when used in the manner directed are part of the solution and not the cause of asthma. The report fails to recognize the benefits of cleaning products to mitigate the effects of asthma. Two citations provided.

Response: The role of cleaning properly with spray and liquid disinfectants and other cleaning products is included in Section 5.5, *Building Operation and Maintenance,* and highlighted in the Executive Summary. Cleaning products can reduce bacteria and mold on surfaces, and reduce exposure to them. Despite this benefit, some can influence indoor air quality in a manner that requires attention to proper use and potential trade-offs. Thus, preventive measures that reduce the need for cleaning products are highly desirable.

CONSUMER SPECIALTY PRODUCTS ASSOCIATION

Comment: CSPA continues to be concerned about the undue emphasis on indoor emissions that present little or no health risks while failing to highlight more significant risks. ARB fails to fully appreciate the rigorous federal product safety regulations that govern formulated consumer products as well as the efforts of manufacturers of those products to assure product safety. The report understates the regulations affecting products used indoors. The Executive Summary dismisses U.S. Consumer Product Safety Commission (CPSC) regulations.

> **Response**: It is not clear which indoor emissions CSPA believes are insignificant. We believe that all indoor emissions of carcinogenic compounds and irritant compounds are potentially significant, and must be considered in light of other factors such as the presence of multiple indoor sources, extent of infiltration from outdoors, length of duration of exposure, and so on. A brief synopsis of the jurisdiction CPSC has over consumer products is presented in the Executive Summary. More detail is provided in Section 4.3.1.1 including information on the Federal Hazardous Substances Act. This Act provides guidelines for determining if a substance is "toxic", and has labeling requirements including the use of specific signal words. However, CPSC's actions are focused more on safety than health, and CPSC only infrequently prohibits a specific component in a product.

Comment: The report fails to adequately consider the public health and indoor air quality benefits of many of the formulated consumer products, articulated in Attachment C to our previous comments. Please include Attachment C and A to the website and in the next version of the report.

> **Response**: The report acknowledges that many consumer products have beneficial effects. Section 5.5 "Building operation and maintenance" discusses the need for regular building maintenance, as well as the beneficial aspects of regular and effective cleaning. Attachments A and C were added to the Clorox comments on our website.

Comment: Table ES-1 improperly aggregates large classes of pollutants with a wide range of properties, toxicity, sources, and health effects. Only the most toxic pollutants likely to be present indoors should be listed and individually paired with its sources and effects. The category of "Organic Chemicals" is particularly misleading. Issue is taken with air fresheners and cleaning agents, aerosol sprays, candles, and endocrine disrupters.

> **Response**: Table ES-1 is designed to give the reader a quick overview of the many categories of indoor air pollutants. ARB believes it has highlighted the categories of pollutants most often discussed and studied in the scientific literature. To meet the intended purpose of the table, pollutants, sources, and potential health effects have been grouped together. The footnote explains that each pollutant may not cause all of the health effects listed in the third column. The table contents are supported by information presented in the rest of the report. Research has shown that "air fresheners" contain VOCs and can have irritating effects on some members of the population. Candle smoke and aerosol sprays contribute to particulate matter. Further information on these sources and on endocrine disruptors, including citations, is in the relevant sections of the report.

Comment: Results of the Rosenman et al. (2003) study and Delfino (2002) review have not been accurately characterized in the Draft Report. There is no clear evidence that these VOCs

are associated in any causative way with asthma. Epidemiological studies such as this are often subject to confounding factors that mask actual causes.

> **Response**: The report does not indicate that these studies purport a causal relationship, but rather an association with asthma symptoms (in individuals with asthma), or exacerbation. See the response to comment submitted by Clorox, above, for further information on the Rosenman et al study as well.

Comment: The report is correct to note that the specific causes of SBS have not yet been firmly identified. However, it is contradictory and speculative in implying that "irritant chemicals" are suspected (Executive Summary).

> **Response**: Refer to Section 2.1.3.2 for a more complete discussion of SBS. Irritation of the eyes, nose, and skin is generally associated with factors such as low ventilation rate, recent renovation or remodeling, and sources such as photocopiers. These factors lead the scientific community to conclude that some component of gaseous or particulate pollution is the source of the irritation.

Comment: The report lists candle burning as a potential source of harmful PM. It is not clear what size particles are being discussed. In the Executive Summary, the report does not analyze the differences in the physics and chemistry of particles outdoors and indoors, including the greater tendency indoors for particles to agglomerate and fall out, and to be captured in sinks such as fabrics and carpeting. Studies have shown that particulates from candles are above the one micron range, and therefore are not considered respirable.

> **Response**: The information requested is discussed in the body of the report in Section 2.2.1. PM from indoor sources, including that from combustion sources, is emitted in a variety of size ranges, including sizes above and below one micron, all of which can contribute to health impacts, as described in the report.

Comment: The term "Pesticides" is a broad term. Pesticides are regulated by state and federal agencies. They undergo numerous tests and are not capable of producing "adverse developmental and neurological effects". Pesticides used indoors do not persist for long in the environment...synthetic pyrethroids last only six to seven weeks before degrading.

> **Response**: In Section 2.3.5, the report briefly discusses different categories of pesticides and their concentrations measured in air and house dust. Indeed, pesticides are extensively regulated and the results of many types of toxicity tests are required to register a pesticide. Nonetheless, they can cause adverse effects on humans when exposure is elevated. The California Department of Pesticide Registration maintains a Pesticide Illness Surveillance Program. In 2002 (the most recent year data is available), there were over 1,000 doctor-reported illnesses associated with occupational and non-occupational exposure to pesticides. Although some newer pesticides do not last as long as some older ones, they nonetheless persist for some time, and that time may be longer in real homes than anticipated. At the 2004 annual meeting of the International Society of Exposure Analysis, several speakers noted their findings that some pesticides appear to last longer indoors than previously estimated, possibly due to the lack of weathering (rain, sun) and temperature extremes in indoor locations.

Comment: Expand the review of biological contaminants in the Executive Summary.

Response: We believe the discussion of biological contaminants in the Executive Summary is an appropriate length.

Comment: The discussion of environmental justice properly notes that many biological asthma triggers are likely to be present in locations where lower income individuals most often live. Unfortunately, by stigmatizing cleaning products that eliminate many biological contaminants, ARB is reducing the likelihood of mitigating these pests.

Response: Environmental justice issues associated with asthma triggers are far more complex than indicated here. Spengler and others have shown that preventive measures are most effective in reducing asthma triggers and preventing exposure to them in low income households. Asthma intervention programs managed by the Department of Health Services and private organizations are working to educate low-income individuals to improve their environment by reducing asthma triggers. We do not believe we have stigmatized cleaning products in our report, nor do we believe that low income households will throw away their cleaning supplies due to the content of this report.

Comment: Table ES-2 and Table 3.6 show that the vast majority of estimated costs are associated with ETS and radon. It is unfortunate that cost estimates for biological contaminants such as infectious diseases were not included. The CDC estimates that infectious diseases result in Americans spending more than $950 billion on direct medical costs.

Response: We acknowledge the magnitude of the health impacts and costs of infectious disease in the U.S. However, infectious diseases can be transmitted through routes such as personal contact, and shared food and utensils among families. It is not feasible in this document to identify the portion of infectious disease that is solely attributable to indoor air quality.

Comment: Table ES-2 implies that all VOCs cause cancer. The few VOCs contributing to this alleged risk should be specified here.

Response: The Executive Summary relays the highlights of the major findings in the report. Please refer to Section 3.1.2, "Deaths From Volatile Organic Compounds (VOC) Exposure" for details on the cancer calculation. Appendix II clearly lists the chemicals included in the cancer calculation. However, as noted in Appendix II, not all VOCs that are carcinogens were accounted for in the estimate.

Comment: In the Executive Summary, it is stated that low-emission product designs or reformulations can usually be accomplished by the manufacturer, with minimal impact on the consumer, often with only minor increased costs. This statement is patently false. Industry's experience working with ARB regulations indicates it is difficult to maintain product efficacy during reformulations. Costs in many categories have been substantial.

Response: That sentence refers to the increased costs to the consumer, which typically are minimal. Costs for reformulations and product changes vary by industry, by facility, and by other factors, such as whether the changes can be implemented as part of other planned modernization or other product changes. In the ARB regulatory framework, ARB conducts economic analyses for proposed regulations. A full analysis would be conducted if regulatory changes were implemented. It should be noted that, in a review of the estimated vs. actual costs of implementing mobile source rules, US EPA found that the actual costs of changes required by rules were much less than those estimated

by the relevant stakeholders, and even less than those estimated by EPA (JF Anderson and T Sherwood, 2002, Comparison of EPA and Other Estimates of Mobile Source Rule Costs to Actual Price Changes, Presentation at the SAE Government Industry Meeting, Washington, DC, May 14, 2002.)

Comment: CSPA likes the segregation of sources into high and medium priority tables. The high priority source categories are well-documented and should be the focus of any state activities for the remainder of the decade.

Response: We are glad you are in agreement with our prioritization.

Comment: The report states that "non-toxic cleaning products are currently sold in the marketplace." Unfortunately, that viewpoint does not take efficacy into account. The legitimacy of "non-toxic" claims is questionable.

Response: Websites for environmentally friendly products and sensitive populations tout "non-toxic" alternatives to many products. The message is that alternatives to standard products are available to do the intended job, though they may take more abrasive action, a period of contact before removing, etc. We agree with your question about legitimacy. A self-proclaimed statement about safety carries little weight. That is why we are promoting several recommendations to mitigate indoor air pollution through emission limits, emission testing, and a comprehensive management system for indoor air quality.

Comment: Table ES-3.2 and Table 6.2 should be edited because very few of the consumer products listed emit the pollutants listed as examples. Also, the table does not acknowledge the comprehensive federal regulatory system in place.

Response: The CPSC has regulated some products, but usually they have used labeling requirements instead of emission limits or content limits, although some such actions have been taken. Many products are not regulated for their impact on indoor air quality. Therefore, federal regulatory efforts have not been adequate for indoor air quality concerns.

Comment: We do not need a management system for indoor air quality nor need to require consumer products to be tested for emissions.

Response: Noted. Our reasons for our recommendations are spelled out in the report.

Introduction and Background

Comment: We are uncertain which "room fresheners" are described on page 32 when referring to Wiley et al. (1991).

Response: In this case, room fresheners included any of several products such as aerosol, plug-in devices, or those that work through evaporative diffusion.

Health Impacts, ETS

Comment: It is not true that a high concentration of an aerosol spray is inhaled during product use. The spray from these products is highly directional to provide product to the intended target.

> **Response**: The report states that "a high concentration of the chemicals consequently may be inhaled during product use before the chemical has a chance to become more dilute in the air". Despite the fact that the spray is directional, solvent is released into the air in the vicinity of the user, where it is likely that some fraction of it will be inhaled. People also move around when using some aerosol sprays, such as when washing windows, thus increasing the likelihood of inhaling the aerosol.

Comment: CDC data indicate that the rate of Americans experiencing an asthma attack in a 12-month period has declined in the last few years.

> **Response**: The rate of asthma attacks may have gone down due to efforts to use preventative medicines to manage asthma. Asthma treatment is not the focus of this report.

Comment: Tables 2.2 and 2.3 indicate asthma is associated primarily with agents of biological origin. There should be more focus on biological contaminants in this discussion.

> **Response**: There is substantial discussion of biologicals in the report.

Comment: Discussion based on the 10-year-old CCRP is of limited relevance because many product reformulations have occurred. Consumer products have very limited amounts of formaldehyde, greatly reduced para-dichlorobenzene, and no trichloroethylene. An updated study should be a condition for any new initiative on indoor air pollution.

> **Response**: As discussed above and in the report, the CCRP estimates were adjusted (down) where new data are available. Some products used in homes and public buildings still contain toxic air contaminants, so we believe our estimates are appropriate. However, as stated in the document, development of a comprehensive program would include collection of additional information and further assessment.

Comment: The referenced study by Fan et al. (2003) is described in a very misleading manner. The authors conclude that the best strategy to reduce indoor reactivity would be to lower outdoor ozone levels.

> **Response**: The report lists the conclusions of Fan et al. (2003) in the same order that the author lists them. The first suggested method for mitigation states "it is prudent to limit the use of products that emit high-reactivity alkenes during episodes when outdoor ozone levels are elevated".

Comment: The section on particulate matter contains only a single paragraph on biological contaminants. o provide an accurate and balanced perspective, a more thorough review of biologicals should be provided here.

> **Response**: Biological contaminants are discussed fully in Section 2.3.4, hence they are given cursory treatment in the PM section.

Comment: It is inaccurate to say that consumer products are a source of formaldehyde. Some coatings and adhesives may emit formaldehyde, but most of our industry's formulated household consumer specialty products to not.

> **Response**: Some types of coatings, sealants, caulks, adhesives, cosmetics, and wallpaper can all emit formaldehyde. Whether or not formaldehyde is an ingredient in a consumer product, it also can be produced through reaction of some emitted chemicals with other chemicals present in the indoor environment.

Comment: Use of the broad term "Volatile Organic Compounds" to represent a very narrow class of chemicals that may present health concerns is misleading. "Sources and Emissions of VOCs" contains dated or inaccurate information. Some chemicals such as benzene, toluene, and para-dichlorobenzene have been greatly decreased in consumer products since the 1991 EPA TEAM studies were conducted.

> **Response**: Based on information available, the report accurately characterizes sources of these compounds and uses caveats such as "at the time the study was conducted". The report cites more recent studies than the TEAM studies for all chemicals. The paragraph on para-dichlorobenzene includes citations from Wallace, 2001 and Gordon et al., 1999. The paragraph on benzene states that the overwhelming source of benzene exposure for smokers is mainstream cigarette smoke, as well as exposure from auto exhaust and gasoline vapor emissions. Akland and Whitaker (200) detected toluene most frequently in auto care products, glues, and personal care products, such as in fingernail polishes.

Comment: The section on VOCs is grossly misleading because it fails to acknowledge that the mere existence of a VOC in indoor air does not mean there is a risk of adverse health impacts. Many VOCs are not Toxic Air Contaminants and do not present health impacts at the levels at which they exist in indoor air.

> **Response**: The report does not state that all VOCs are harmful. The report highlights the chemicals that have been studied in indoor air and are considered by the general scientific community to be indoor air pollutants. In some cases, chronic RELs or other health benchmarks are included so the reader can compare reported concentrations to levels that are expected to be free of adverse health impacts. However, if a chemical is a carcinogen, there is no known safe exposure level. For a full review of levels associated with health impacts, please search the OEHHA website.

Comment: Issue is taken with results reported from several studies cited in the report (Akland and Whitaker, 2000; Zhu et al., 2001; Cooper et al, 1995).

> **Response**: The report reflects findings from those studies that were conducted on products available at the time. Akland and Whitaker (2000) was included because, although not published, all data were obtained from products purchased in retail stores in California.

Costs

Comment: Data in Table 3.2 are highly misleading. In some cases cost data are given where etiology has been reasonably established. In other cases, the costs are less certain (ETS

cancer and heart disease). The estimate for VOC lung cancer is even more uncertain. It is misleading and grossly inaccurate to present the same value for "low", "average", and "high" estimates.

Table 3.3 suffers from the same problems as Table 3.2, as it mixes cost estimates of very different types. "VOCs: cancer" is not a diagnosable health end point. Suggestions are provided for a better column heading and replacement term for "VOCs: cancer".

> **Response**: The tables in Section 3 are intended to provide an overall estimate of the known costs of indoor pollution. The costs for ETS cancer and heart disease are fairly robust; they are based on estimates published in an EPA document in which all immediate medical costs are summed. The text in Sections 2 and 3, and Appendix II, describes the meaning of the word "cancer" in the VOC risk estimates. The range or confidence intervals in the health risk estimates are discussed in detail in the risk assessment for ETS by OEHHA. Those values will be finalized after the ARB Scientific Review Panel has completed their review of OEHHA's estimates. The confidence intervals for VOC carcinogens are not provided in the final Comparative Risk report; that is why the same values are shown in all columns. In the final report, we will delete the VOC estimate in the high and low columns and use a "not available" designation.

Methods to prevent and reduce, to the end

Comment: In Section 5.2 "Ventilation", ARB fails to distinguish between ASHRAE Standard 62.1 for commercial and high-rise residential spaces and 62.2 for all other residential spaces. We would like to issue caution regarding addendum "n" to 62-2001. Addendum "n" reduces the prescribed minimum ventilation rates for many spaces, based on low-emitting European buildings. We urge California to not adopt those lowered ventilation rates and potentially jeopardize the health of many Californians.

> **Response**: The two ASHRAE ventilation standards are discussed in Section 4, see Residential Guidelines and Nonresidential Guidelines.
>
> In setting ventilation standards, the California Energy Commission must consider protection of IAQ. ASHRAE standards along with other information are considered in developing these ventilation standards; stakeholder input is encouraged.
>
> Thank you for mentioning the low-emitting buildings in Europe. Such buildings highlight the fact that low-emitting alternatives are available and that other countries give indoor air quality a higher priority than the U.S.

Comment: Mitigation options should include recommendations for biological contaminants because they present the greatest health risks and costs in California. We are concerned that consumer products are included among materials requiring emissions testing and labeling.

> **Response**: Mitigation options for biological contaminants are being addressed by the Department of Health Services, federal agencies, and local agencies. Consumer products are one of several types of products that are recommended for emissions testing; however, they are in the medium, not high, priority group.

DEL ORO

Comment: Remove negative statements about the health effects of d-Limonene and citrus terpenes. These misleading conclusions will have a negative effect on the American citrus community.

> **Response**: The June 2004 draft report included some poor wording regarding the effects associated with terpenes. Language was improved in the November 2004 draft and is no longer misleading.

DURAFLAME (Cline & Duplissea)

Comment: Manufactured firelogs are not a contributor to indoor pollution. Banning fireplaces in new construction is overkill. Suggest that consideration be given to middle-ground solutions that have scant controversy and easy public acceptance. Indoor Air Quality standards would create a large, intrusive and potentially expensive bureaucracy with scant health benefits. Mandates that impact personal habits are difficult to enforce. Recommendations for ICAT should include new and existing products that are clean-burning, such as manufactured firelogs.

> **Response**: We appreciate your development of a lower-emitting product. Such middle ground options would be considered in the development of any comprehensive program, or any regulations. It is not our intent to enlarge bureaucracy, nor to impose mandates that impact people's choices. Rather, as indicated in the report, changes can be made at the manufacturing stage, or in the design and construction of buildings, that would greatly improve indoor air quality.

FLORIDA CHEMICAL COMPANY, INC.

Comment: We would like ARB to remove a statement on page 36 regarding the reporting by Delfino of a self-reported asthma prevalence associated with several chemicals including terpenes and limonene. The statement is misleading and will lead to false conclusions. Scientific literature reports that d-limonene and other terpenes are used to alleviate asthmatic and respiratory symptoms.

> **Response**: The results of the study cited by Delfino are pertinent to the report. The report also contains a caveat that the effects seen may be subject to confounding by other causal agents.

Comment: Several arguments are made to change language in the report regarding the reactivity of terpenes with oxidants to produce formaldehyde and ultrafine PM. Generally, the comment suggests that the word "terpene" be removed from the report. It is pointed out that conditions used to demonstrate reactions have been laboratory experiments with levels greater than normally observed indoors. Wilkins et al.(2001) conducted studies on isoprene, rather than terpenes.

> **Response**: The important message is that several investigators are confirming that these chemicals with double bonds react with oxidants present in indoor air to produce irritating compounds. The paper by Long et al. (2000) clearly shows the generation of

ultrafine PM as a result of cleaning with a product containing terpenes under typical household conditions. This paper measured the production of particles from various typical indoor activities. Particle production from mopping was exceeded by particle production from frying and burning food. It is true that the Wilkins study investigated the reactivity of isoprene. However, other studies cited, in addition to Wilkins, were conducted with terpenes.

Comment: Scientific literature is not fully quoted, resulting in an inaccurate relaying of information. Test conditions should be included in the report.

Response: We believe that the report as written accurately relays the authors' findings and conclusions. The test conditions of the studies cited are typical, accepted approaches for identifying reactions and health impacts.

FORMALDEHYDE COUNCIL

Comment: The costs attributable to VOCs (2% of total indoor air costs) do not justify the emphasis on VOC reduction in indoor air. A quantitative prioritization, while apparently beyond the scope of this project, is a necessary step prior to taking action under a comprehensive program to address indoor sources.

Response: The cost estimates do not necessarily reflect the full extent of exposure and risk in California. As stated in the report, cost estimates reflect the availability of cost information and the length of time a given pollutant has been studied. We do not believe that there is an undue emphasis on reduction of indoor VOCs in the report. The high priority source categories emit a variety of pollutants, including criteria pollutants, allergens, radon, and VOCs. As stated in the previous responses to comments, a detailed prioritization based on quantified criteria would be an appropriate step prior to taking action under a comprehensive program.

Comment: The cancer risk assessment methodology developed by the CIIT Centers for Health Research should be referenced in the final report. Eleven papers have been published over the past four years on this work. It has undergone extensive review by U.S. EPA, Health Canada, and has been used by other agencies around the world. Other efforts to refine the understanding of risk associated with exposure to formaldehyde are anticipated (IRIS and NCI epidemiology study). The report should acknowledge these forthcoming developments because they may validate the CIIT approach. Recommendations in the report may not be implemented for many years, additional information may be available at that time.

Response: In November 2002, ARB and OEHHA denied a petition to formally review the CIIT formaldehyde risk assessment. The petition was based in part on the potency estimate change associated with the CIIT 1999 report. OEHHA determined that the report was a new analysis of old evidence rather than new evidence. OEHHA stated that more information is needed to evaluate the risk assessment model used by CIIT, and that it needs to be peer-reviewed and validated. In the future, if formaldehyde risk assessments are reevaluated, information stemming from this work may be considered.

Comment: ARB should reconsider mitigation strategies associated with formaldehyde; particularly the relationship between outdoor and indoor exposure levels. With outdoor levels above the guideline level, consider the feasibility of a strategy that would seek to make indoor

exposure levels lower than outdoor levels. Also consider that in schools, adequate ventilation, and uncomfortable temperature and humidity levels are also problems of concern. The draft Report gives an extremely misleading impression that the problem of elevated formaldehyde in classrooms must be corrected immediately. Outdoor formaldehyde levels also so not meet the chronic REL guideline.

> **Response**: Indoor formaldehyde levels are several times greater than outdoor levels; thus, there is a great deal of room for improvement in indoor formaldehyde concentrations. Reduction of formaldehyde levels in schools with elevated indoor concentrations is indeed considered a very high priority by ARB, because in some classrooms the levels are many times the levels considered acceptable even for prevention of acute effects, and the occupants are children. If regulations are developed to reduce formaldehyde levels in indoor air, the ambient air concentration will certainly be taken into account: we would not expect indoor levels to be lower than outdoor levels. However, we do not agree with the rationale suggested, which is that nothing should be done indoors because outdoor levels also exceed guidelines (to a much lessor degree). The majority of the exposure and risk is from the elevated indoor concentrations, which are several times higher than outdoor levels.

Comment: Misleading statements about formaldehyde levels in indoor air abound. Two examples are provided. Include statements (caveats) about the feasibility of meeting health guidelines based on outdoor levels.

> **Response**: The statements you quoted from the report are correct as written. Formaldehyde levels nearly always do exceed chronic health-based guideline levels and acceptable cancer risk levels. It is also true that it is generally not feasible to achieve levels below these guideline levels because outdoor levels average about 3-5 ppb. If regulations are developed to reduce formaldehyde levels in indoor air, the ambient air concentration will certainly be taken into account. No further discussion is needed regarding ambient formaldehyde concentrations.

FRAGRANCED PRODUCTS INFORMATION NEWORK

Comment: Fragrances are intentionally added to numerous products such as cosmetics, detergents, candles, cleaners, etc. By definition, fragrances are volatile organic chemicals. The fragrance industry is primarily self-regulated with little oversight. Of 54 fragrance materials that are used at 1 million pounds or more annually only 2 have a full set of basic toxicity data. Scientific literature includes citations that fragrances can be a sensory irritant, a lower airway irritant, and trigger for asthma.

There is a limited awareness about the fragrance issue in the general population or the medical and scientific communities. Thus far, the industry has not adequately addressed concerns regarding the impact of multiple sources of fragrance on indoor air quality and health. There is an immediate need for regulatory agencies to educate on the impact of scented consumer products on sensitive populations. Research is needed to determine mechanisms by which fragrance negatively impacts respiratory health. There is a need for a cooperative process with industry to determine problematic materials and find suitable substitutes.

> **Response**: We appreciate the information provided by the Network. We share the Network's concern regarding fragranced products, and agree that further research in this

area is needed. Some information was added to the report, and we will include fragranced products in the list of area of research needs in the final report.

GAS APPLIANCES MANUFACTURERS ASSOCIATION (GAMA)

Comment: On page 150, vented and unvented combustion appliances are combined into one category without distinction. Vented products meet national performance standards that do not allow combustion products to be emitted into the living space. Readers need to know this piece of information.

Response: The text will be revised at a later date to reflect the report's preceding discussions of the risks from unvented or poorly vented combustion appliances. As discussed above (under AHAM comments), vented products other than direct-vent models can emit pollutants into the living space.

Comment: Page 122- SCAQMD emission limits for gas water heaters in homes will be postponed until 2006, not 2005 as stated in the report.

Response: Thank you, this will be updated in the final version of the report.

Comment: The statement that design changes for appliances can be made with minor increased costs are not correct. Industry expenditures to develop water heater systems that meet new NOx limits total many millions of dollars, ultimately to be borne by the consumer. The discussion of the porous insert technology does not include costs to incorporate this feature into a complete gas appliance system (gas stove).

Response: Costs for design changes for appliance vary by industry, by facility, by appliance, and by other factors, such as whether the changes can be implemented as part of other planned facility modernization or appliance changes. In the ARB regulatory framework, ARB conducts economic analyses for proposed regulations. A full analysis would be conducted if regulatory changes were implemented. It should be noted that, in a review of the estimated vs. actual costs of implementing mobile source rules, US EPA found that the actual costs of changes required by rules were much less than those estimated by the relevant stakeholders, and even less than those estimated by EPA (JF Anderson and T Sherwood, 2002, Comparison of EPA and Other Estimates of Mobile Source Rule Costs to Actual Price Changes, Presentation at the SAE Government Industry Meeting, Washington, DC, May 14, 2002.)

The text will be revised in the final report to add a caveat concerning the system design for incorporating the porous insert.

HEARTH, PATIO, AND BARBECUE ASSOCIATION

Comment: The draft report does not distinguish between conventional and certified wood stoves, and in so doing overestimates the emissions of wood stoves. Since 1992 all wood stoves have to be certified which results in a reduction of PM and PAH emissions. Additionally, they must operate with their gasketed doors firmly closed. As a result, certified stoves cannot leak pollutants into the indoor environment. It would be appropriate to recommend that conventional wood stoves be replaced with certified wood stoves.

Response: The report mentions the U.S. EPA emission standards for certified woodstoves. Those standards are for emissions to the outdoors; such stoves may have lower emissions to the indoors, but those emissions can still be substantial. The federal standards do not require door seals or the catalyst to be maintained over the life of the stove, nor do they prevent woodstoves from being operated with leaky doors. Any stove can leak into the room while the stove is being stoked.

Comment: The report fails to establish a direct link between emission to the ambient atmosphere and decreased indoor air quality for hearth products. Examples are provided of local air districts that require limited wood stove emissions to the ambient air, but their impact on indoor air quality is not stated. The report also mentions energy efficiency standards for certain hearth products and requirements for a permanently open flue damper for certain applications. The discussion assumes, without any supporting information, that a more efficient appliance will benefit indoor air quality. A strong causal connection must be made between these recommended control measures and an improvement in indoor air quality.

Response: As stated in our responses to this comment on the previous draft report, the large study in two California cities by Sheldon et al. documented the infiltration of outdoor PAHs from neighborhood woodburning on indoor PAH levels. In addition, several studies have shown that a major part of outdoor PM can penetrate into homes, as discussed in Section 2. Finally, ARB and air districts receive a number of complaints each winter from citizens who live in neighborhoods with woodburning who are bothered by woodsmoke entering their home from outdoors.

Comment: The report imprecisely concludes that fireplaces are a significant source of carbon monoxide. The report cites a study by Sheldon et al., in which "a fireplace" – presumably one unit in one home – caused a violation of a CO standard. It is important to identify if this was a wood or gas log fireplace. It would be extremely unlikely for a woodburning fireplace to cause a violation because the occupants of a home would quickly detect improper venting. Gas fireplaces are designed and tested to minimize CO generation and leakage. Such incidents are generally due to improper installation and/or operation. It appears the significance of one fireplace has been exaggerated in the report. Please remove fireplaces from Tables ES-1 and 2.1 unless a clearer and stronger discussion of fireplaces as a source of CO can be provided.

Response: Fireplace emissions of PM, PAHs, aldehydes, and other pollutants in addition to CO are also a health concern. It was not specified whether the one home with a fireplace that exceeded the CO standard had a gas or woodburning fireplace. However, Sheldon et al. also found that several homes had CO levels between 5 and 9 ppm that exceeded ambient levels, indicating a significant indoor source of CO. A smoldering fire that produces a high level of CO may not necessarily produce enough smoke to alert the occupants, especially if they are sleeping at night, which is a documented scenario for CO poisoning. Additionally, backdrafting can occur, bringing CO and other combustion pollutants back into the living area. We recognize that this is not usually die to fault design, but rather to other factors; however, it needs to be considered in assessing fireplaces (and other combustion devices) in terms of their potential impact on indoor air quality.

Comment: The report has placed wood stoves and fireplaces too high on the ranked list of pollution sources for mitigation. Hearth products when properly installed and maintained will not significantly contribute to contaminants in indoor air. Industry is working with U.S. EPA, under

the auspices of ASTM International, to develop standards for a wood-fueled fireplace that will emit fewer particles. ARB must consider the benefits of potential mitigation approaches, whether effects will be direct or indirect, how significant the improvements will be, and the size of the population that will benefit.

> **Response**: The category of "combustion appliances" remains in the high priority group because one or more combustion appliances are present in virtually every home, and because gas stoves and some other devices are unvented, leading to a high potential for emission and exposure. However, this does not mean that every device would warrant changes. A full analysis would be conducted if measures are to be pursued to address indoor combustion appliances. For its existing regulatory programs, ARB conducts economic analyses prior to taking action.

INSTITUTE OF MEDICINE

Comment: Several corrections or improvements were recommended, with specific language changes provided. Suggested wording changes are subtle, but make statements align perfectly with IOM reports.

> **Response**: Thank you, the suggested changes were implemented.

MANUFACTURED HOUSING RESEARCH ALLIANCE

Comment: There have been changes in manufactured homes construction that result in overall reduction in formaldehyde levels. Specifically, there has been a dramatic reduction in the use of UF-bonded wood products. This was not addressed in the revised report. The response to comments that 4 homes in Florida is representative of California is flawed.

> **Response**: Hodgson et al. (2000) state that the formaldehyde levels in manufactured homes are approximately 50 percent of what they were two decades ago. He attributes this to construction changes, such as less paneling, and reduced emissions from the products relative to 20 years ago. This is consistent with conclusions from a formaldehyde emissions study conducted by Tom Kelly at Battelle (1996). These statements are included in the report. ARB acknowledges that conditions in Florida may not be representative of conditions in California, but there are some areas of California that are similar to Florida, and there is no published data countering these results. It is important to note that there are other sources of formaldehyde besides building materials in a new home. Examples include wallpaper, draperies, furniture, and gas appliance use. We would agree that a study of new manufactured homes would provide useful information.

DAVID MORALEZ, Davis, CA

Comment: Gratitude is expressed for the long overdue report. Support is offered for the recommendations listed to make meaningful progress to protect the public from unhealthy indoor air. Ideally the recommendations would be supported with funding.

> **Response**: Thank you, we appreciate your positive comments.

NATIONAL PROPANE GAS ASSOCIATION

Comment: Statements related to low- or zero-emitting appliances are misleading for several reasons. Appliances are specifically designed to exhaust all of the combustion products to the outdoor air, therefore no exhaust can enter the living space when equipment is properly installed. The recommendation for zero-emitting appliances implies one should use electric appliances, which shifts the pollutants to another site (where the power plant is located).

> **Response**: Direct-vented (sealed-combustion) appliances are virtually zero-emitting in terms of indoor emissions. For example, direct-vent gas furnaces and gas fireplaces are very energy efficient, are resistant to backdrafting, and have been used widely for years. Direct vent gas stoves were developed by the Canadian Gas Research Institute in the 1990's but have not been marketed widely. The report will be revised to clarify this topic. The report also discussed the need for proper, effective, and well-maintained venting of combustion appliances and for cooking appliances, both electric and gas.

Comment: ARB recommends active exhaust ventilation to reduce pollutants from combustion appliances. This would have no impact on the appliance if the venting system is properly installed because all pollutants would be vented to the outside. Active exhaust ventilation is required to achieve the requisite number of air changes per hour in homes with tight new construction. Active exhaust could also lead to backdrafting problems.

> **Response**: Our intention is to recommend active *local* exhaust ventilation, with installation specifications that assure adequate flow and low noise levels. Such a provision would be provided if the operation of a range hood were linked to the operation of a gas kitchen stove. To prevent backdrafting, accepted measures for house-as-a-system design should be used, such as direct-vent gas furnaces, limits on flow rates for range hoods and other exhaust devices, and depressurization testing.

Comment: On page 23 of the report, an option is suggested to require manufacturers to have a third party conduct emissions testing on appliances. Appliances already receive testing during the certification process. Adding a label with results would confuse the general public. They would interpret it to mean the emissions that are deposited within the living space, which would not occur with a properly installed vent.

> **Response**: Our focus is on unvented appliances, such as gas stoves, which typically are not vented, and can emit pollutants directly to the indoor space. Because all products on the market may meet certain emission performance criteria, consumers and builders need labels and accessible information to identify low-emitting products.. This approach would be developed with input from the public, consumer groups, and other stakeholders.

PEACE RIVER CITRUS PRODUCTS

Comment: Remove negative statements about the health effects of d-Limonene and citrus terpenes. These misleading conclusions will have a negative effect on the American citrus community.

Response: The June 2004 draft report included a misstatement regarding the health effects associated with terpenes. Language was revised in the November 2004 draft, and information from recent scientific studies and related information was included. We believe that the information we provided in the November 2004 report is correct.

REGAL AIR QUALITY, INC.

Comment: We need a vehicle to identify the population that is currently at risk but don't know it and provide proper education and information for them. The report should include a recommendation that seniors be provided with monetary assistance to implement procedures to retrofit their homes for improved indoor air quality. Specifically, HVAC duct insulation can emit formaldehyde in areas with high temperatures such as Arizona. Improvements to the HVAC system and/or insulation can improve the occupant's health, but monetary assistance is sometimes needed.

Response: We agree that there is increased education needed. In California, our approach to address cost issues might be to recommend low-emitting formaldehyde duct insulation in state and utility weatherization programs for low-income households. We are not yet certain, however, whether the duct insulation used by California builders also emits formaldehyde as does the type used in Arizona. Accordingly, we have included limited measurements to be made in residential ventilation systems in a study of new homes in California, which we expect to start later this year.

RESILIENT FLOOR COVERING INSTITUTE

Comment: In October, 2004, the DHS determined that the Section 01350 limit for acetaldehyde should be something other than one-half the OEHHA chronic REL. This should be reflected in the report. Alternate wording is provided for a couple of places in report.

Response: We did not concur with DHS on this matter, but will add the information to the final report if it is accurate at that time. Our concern is that it would allow a single product to potentially contribute sufficient amounts of the chemical to exceed OEHHA's chronic REL guideline value (which for acetaldehyde is 9 ug/m^3).

SEMPRA ENERGY

Comment: They expect their comments to be shared with the scientific review panel.

Response: All of the comments received from the public on the June 2004 draft report were provided to the peer review panel when they reviewed the November 2004 document, so that they could see the comments received and how we responded to them. The peer review panel has completed their review, so will not be sent the current comments. However, if any peer review member requests them, we certainly would provide them.

Comment: Banning gas stove use would have a profound economic and social impact on the state. A study would probably show that electrocution death rates from electric cooking appliances are nearly equal to death rates from CO poisoning from gas cooking appliances.

Response: The report does not recommend banning gas stoves from homes, but rather to use alternatives such as direct vent appliances, or to improve their local exhaust and reduce their emissions. In addition to concerns regarding CO poisoning, ARB is concerned about NO2 emissions and other emissions from combustion appliances. A revision to the building standards would require a detailed analysis and public review.

Comment: An attached report (2000) conducted by the CPSC with assistance from NIST looked at CO emissions from the misuse of natural gas ranges. The CPSC declined to take any further action on setting new CO emission standards based on their studies.

Response: While the standards may be sufficient for safety purposes (prevention of life-threatening effects or death), we believe they are not sufficient to adequately protect health. The referenced modeling studies address indoor CO, and, in fact, show that the ARB's IAQ guideline and state ambient air quality standard would be exceeded. For example, the 1-hour and 8-hour guideline level would be exceeded in the baking scenarios with smaller kitchen volumes and lower air exchange rates. The medium and lower air exchange rates used in the modeling are not as low as those are for some new or weatherized homes in California. This modeling estimate does not include the contributions of outdoor CO levels, which can be substantial at times. Further, these studies do not address what is typically the limiting factor for combustion appliances: indoor NO_2.

Comment: The foods cooked and specific type of cooking activity are more important to IAQ than the energy source of the cooking appliance. For example, PAHs are emitted during cooking with electricity or gas.

Response: That is true for most PM emissions, although PM emissions (especially ulftrafine PM) are generally higher when using a gas stove. In addition, NO_2, CO, and aldehydes are always higher when using a gas stove.

SOAP AND DETERGENT ASSOCIATION

Comment: Add references to the Executive Summary for the production of formaldehyde and ultrafine PM from the reaction of terpenes and oxidants (page 5). Also, add a reference to the paragraph about VOCs on page 8.

Response: Numerous facts are presented in the Executive Summary, most of them widely accepted in the field of indoor air quality. Please refer to the body of the report to find extended discussion with references.

Comment: Household and institutional cleaners are listed as a source of several VOCs in Tables ES-3.2 (and 6.2). However, these ingredients are not intentionally added to water soluble, surfactant-based products. Therefore, these chemicals should be absent or at *de minimus* levels in the water soluble, surfactant-based products.

Response: Thank you for this information. We do not feel clarification is needed in the text because footnotes indicate all of the pollutant sources may not emit all of the pollutants listed in the corresponding box. However, such information would clearly be important in developing mitigation approaches for specific products.

Comment: The paragraph describing the types and uses of mercury does not detail the products with inorganic mercury. We recommend their inclusion.

Response: The sources of inorganic mercury are listed in Table 2.13; this is sufficient.

Comment: According to the U.S. EPA report, "Reducing Mercury Use in Healthcare: Promoting a Healthier Environment", mercury can be a contaminant in detergents and cleansers when the mercury-cell process is used to manufacture some cleaning product ingredients. This should be statedThe products mentioned in the U.S. EPA report are down-the-drain products. Recognizing this, one would not expect any mercury that may be in a product to become air borne. Moreover, there are no validated methodologies for testing cleaning product for mercury. Therefore, reported test findings may not be actual mercury concentrations.

Response: *Our report adequately states the findings from the 1999 EPA report. In fact,* if we consider the statement from the EPA report that "many forms of mercury circulate in the environment, moving from land or water to air and back again," the comment to not expect any mercury in a [down-the-drain] product to become airborne does not necessarily hold true. More importantly, any mercury in products used indoors that are released to the indoor environment, even as splash droplets, will persist. Also, it may be the case that there are no validated methodologies for testing cleaning products, but the concentrations in the AB 1173 report are not being reported for cleaning products.

Comment: Add a description of the DGS EPP task force to page 120-121.

Response: The Environmentally Preferable Purchasing (EPP) Task
Force is discussed in Section 4.3.3.2 under the subheading "Sustainable Buildings and Section 01350". EPP is also discussed in the same section under the subheading "Environmentally Preferable Products for School Construction".

SOUTHERN GARDENS CITRUS (two commentors: Liddle and Chapman)

Comment (same comment from both commentors): Remove negative statements about the health effects of d-Limonene and citrus terpenes. These misleading conclusions will have a negative effect on the American citrus community.

Response: The June 2004 draft report included a misstatement regarding the health effects associated with terpenes. Language was revised in the November 2004 draft, and information from recent scientific studies and related information was included. The information we provided in the November 2004 report is correct.

TREATT

Comment: Remove negative statements about the health effects of d-limonene and citrus terpenes. Technical arguments will be made by others in the citrus industry.

Response: The June 2004 draft report included a misstatement regarding the health effects associated with terpenes. Language was revised in the November 2004 draft,

and information from recent scientific studies and related information was included. We believe that the information we provided in the November 2004 report is correct.

UNITED STATES DEPARTMENT OF AGRICULTURE

Comment: The report states a major objection to the use of d-limonene and other terpenes in cleaning products is due to the reaction products formed between terpenes and oxidants in the air. These compounds cause irritation without any terpenes being present. Before banning terpenes, the actual levels of formed irritants should be determined and alternatives for safe cleaning chemicals investigated. Numerous agencies do not categorize terpenes as posing health risks.

Response: We do not suggest banning terpenes: rather, we indicate that further research is needed. ARB certainly acknowledges that formaldehyde and PM are irritants, regardless of their source. However, if terpenes introduced to indoor air in cleaning products lead to significant increased levels of these irritants in indoor air, then mitigation would be warranted. Several investigators around the country and the world are studying the reaction of terpenes and oxidants to determine potential impacts on indoor air quality.

THE END

No written material available at time of electronic book creation.