

Health Effects of Exposure to Environmental Tobacco Smoke

**Final Report
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C A L I F O R N I A
ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT

Preface

Environmental tobacco smoke (ETS), also called second hand tobacco smoke, can affect nonsmokers in proximity to people smoking tobacco. The scientific and medical literature contains hundreds of investigations of the association between ETS exposure and a variety of adverse health impacts, including carcinogenicity as well as cardiovascular, developmental, reproductive, and childhood respiratory effects. Although some studies have not shown an association, authoritative investigations and reviews over the past two decades have presented substantial scientific evidence linking ETS exposures to a number of adverse health outcomes.

Interest in the health effects of second hand tobacco smoke on the part of members of the Scientific Review Panel (SRP) on Toxic Air Contaminants led to a request by the SRP for a health assessment of ETS, and a collaborative agreement between the Office of Environmental Health Hazard Assessment (OEHHA) and the Air Resources Board (ARB) in February 1992 to initiate such an assessment. Although not formally entered into the State's Assembly Bill 1807 toxic air contaminant identification program, the ARB, SRP and OEHHA agreed that a thorough assessment of risk similar to that done under the AB 1807 process was warranted. This was done to ensure a comprehensive review of the scientific data, frequent public input through public comment periods and workshops, and an independent scientific review by the SRP.

This report will be presented as an informational item at a public meeting of the members of the Air Resources Board. The report along with all comments will be forwarded to the Department of Health Services (DHS) Tobacco Control Program for appropriate action under their mandate as the State's lead agency for addressing health effects related to tobacco use.

OEHHA, with the assistance of scientists from the DHS, had primary responsibility for the preparation of this assessment. ARB provided assistance with the ETS-related exposure data as well as with report reproduction, workshop organization, and mailouts.

OEHHA and ARB sponsored a workshop in October 1992 to obtain public input early in the evaluation of ETS health effects and exposure in California. At the workshop, preliminary thoughts on the direction of the ETS assessment were discussed with participants, which included individuals from local, state, and federal government agencies, universities and other research organizations, representatives of the tobacco industry, and public interest groups.

The development of the assessment involved extensive literature review, document development, public workshops, public comment and scientific peer review followed by document revision. Public release of reviews on each major area of health effects occurred as they were prepared. The first two documents (*Respiratory Health Effects of ETS* and *The Role of ETS in Cancers Other Than Lung Cancer*) were mailed in May 1994; the public comment period was May 2 to June 24, 1994, and a public workshop on these documents was held June 14, 1994. Subsequent documents were released with public comment periods (and public workshops) as follows: September 1994 release of *Cardiovascular Health Effects of Exposure to ETS*, public comment period September 2 to November 4, 1994 (workshop: October 4, 1994) (extended deadline due to late mailing of tables); March 1995 release of *Developmental and Reproductive Effects of Exposure to ETS*, public comment period March 3 to April 24, 1995 (workshop: April 4, 1995); September 1995 release of *ETS: Exposure Measurements and Prevalence*, public comment period September 1 to October 16, 1995 (workshop: October 3, 1995); and January 1996 release

of *Carcinogenic Effects of Exposure to ETS, Excerpt: ETS and Lung Cancer*, public comment period January 26 to April 1, 1996 (workshop: March 25, 1996). Following the public comment period, each document was revised to respond to comments received and updated to include critical new studies; these revised documents were compiled to form the Final Draft for Scientific, Public, and SRP Review, *Health Effects of Exposure to Environmental Tobacco Smoke*, released in February 1997. The Final Draft had a public comment period of March 7 to May 5, 1997 (public forum: April 17, 1997).

The Final Draft along with Appendices A and B, which summarize and respond to comments received during the formal comment periods, were reviewed by the SRP and discussed at its meeting on June 19, 1997. Several newly published studies were added to the final document at the request of the SRP (e.g., the full report of Kawachi *et al.*'s analysis of cardiovascular disease risk in the Nurse's Health study, published after the release of the Final Draft, in which it was reported as an abstract). The SRP's Findings as a result of its review of the Final Draft are included in Attachment I. As noted in the Findings transmittal letter from SRP Chairman Dr. James N. Pitts, "the Panel views ETS as a toxic air contaminant, and it has a major impact on public health."

Authors and Acknowledgements

This document was prepared by the Reproductive and Cancer Hazard Assessment Section (RCHAS) and the Air Toxicology and Epidemiology Section (ATES) within the Office of Environmental Health Hazard Assessment (OEHHA) of the California Environmental Protection Agency (Cal/EPA). Lauren Zeise was the project officer with overall responsibility for the contents of this report. Amy Dunn coordinated the development of the draft chapters and their revision, the public workshops, and the finalization of the full report. James Donald played a key role in the planning and development of Chapters 3, 4 and 5. Amy Dunn and Lauren Zeise were the editors of this report.

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As described in the preface, the final document was preceded by a final draft and by earlier external review drafts of each topic area which were released for public review and comment. The authors wish to thank those who sought to improve the quality of this report with their comments, and are particularly grateful to the members of the Scientific Review Panel, especially the leads on ETS, Craig Byus, Gary Friedman and Stanton Glantz as well as former panel member Charles Becker, all of whom provided guidance and detailed suggestions. Special thanks to Jennifer Jinot and Steven Bayard of U.S. EPA, and Ira Tager, Kathy Hammond, Neil Benowitz, John Balmes and John Pierce. Thanks also go to James Collins, John Faust, Jeff Fowles, Andrew Salmon, Martha Sandy and David Ting, for assistance with the response to public comments.

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Table of Contents

Preface	i
Authors and Acknowledgements	iii
Executive Summary	ES-1
Attachment I: Scientific Review Panel Findings	ES-10
1 Introduction	
1.0 Impact of ETS on the Health of California	1-1
1.1 Organization of the Report	1-1
1.2 Definition of ETS	1-2
1.3 Methodology	1-2
1.4 Weight-of-Evidence Evaluations	1-6
Table	1-8
References	1-9
2 Exposure Measurement and Prevalence	
2.1 Introduction	2-1
2.2 Properties of ETS and Its Constituents	2-2
2.3 Exposure Measurement: ETS Concentrations in Indoor Environments ...2-5	
2.4 Exposure Measurement: Biological Markers2-12	
2.5 Exposure Measurement: Use of Questionnaires	2-21
2.6 Exposure Prevalence and Determinants2-24	
2.7 Chapter Summary and Conclusions	2-36
Figures	2-39
Tables	2-46
References	2-62
3 Developmental Toxicity I: Perinatal Manifestations	
3.1	
Introduction	3-1
3.2 Fetal Growth	3-1
3.3 Spontaneous Abortion and Perinatal Mortality	3-24
3.4 Congenital Malformations	3-30
3.5 Chapter Summary and Conclusions	3-35
Figures	3-37
Tables	3-39
References	3-48

4 Developmental Toxicity II: Postnatal Manifestations

4.1 Introduction	4-1
4.2 Sudden Infant Death Syndrome	4-2
4.3 Cognition and Behavior in Children	4-12
4.4 Postnatal Physical Development	4-23
4.5 Respiratory Development and Function	4-28
4.6 Chapter Summary and Conclusions	4-29
Tables	4-30
References	4-40

5 Reproductive Effects

5.1 Introduction	5-1
5.2 Female Fertility and Fecundability	5-1
5.3 Other Female Reproductive Effects	5-8
5.4 Male Reproductive Toxicity	5-10
5.5 Chapter Summary and Conclusions	5-11
Tables	5-13
References	5-15

6 Respiratory Health Effects

6.0 Introduction	6-1
6.1 Acute Health Effects	6-1
6.2 Chronic Health Effects	6-33
6.3 Susceptible Populations	6-58
6.4 Chapter Summary and Conclusions	6-66
References	6-67

7 Carcinogenic Effects

7.0 Introduction	7-1
7.1 All Cancers (combined)	7-3
7.2 ETS and Lung Cancer	7-14
7.3 ETS and Cancer Sites Other Than Lung that are Associated with Active Smoking: Nasal Sinus, Cervical and Bladder	7-31
7.4 ETS and Cancer Sites Where Evidence for the Role of Active Smoking is Equivocal: Breast, Stomach, Brain, Leukemia, Lymphomas and Non-Hodgkin's Lymphomas, Other Rare Childhood Cancers	7-41
7.5 Chapter Summary and Conclusions	7-58
Tables	7-60
References	7-84

8 Cardiovascular Effects

8.0 Introduction	8-1
8.1 Description of Epidemiologic Studies	8-4
8.2 Discussion of Epidemiologic Studies	8-23
8.3 Other Supportive Evidence	8-28
8.4 Chapter Summary and Conclusions	8-37
Tables	8-39
References	8-66

Appendix A: Summary of Public Comments and Responses	A-1
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Appendix B: Summary of Public Comments and Responses	B-1
on the February 1997 Final Draft	

List of Tables and Figures by Chapter

Executive Summary

Table ES.1	Health Effects Associated with Exposure to Environmental Tobacco Smoke
Table ES.2	Estimated Annual Morbidity and Mortality in Nonsmokers Associated with ETS Exposure

1 Introduction

Table 1.1	Estimated Annual Morbidity and Mortality in Nonsmokers Associated with ETS Exposure
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2 Exposure Measurement and Prevalence

List of Figures:

Figure 2.1	Plasma Cotinine Concentrations in Self-Reported Smokers and Nonsmokers
Figure 2.2	Urinary Cotinine of Breast-Fed Infants in Relation to Maternal Cigarette Smoking
Figure 2.3	Percent of Nonsmokers in California Reporting ETS Exposure
Figure 2.4	Reported Average Daily ETS Exposure Duration in California
Figure 2.5	Relative Person-Minutes of ETS Exposure in Different Environments
Figure 2.6	Adult Smoking Prevalence: California and the United States (1965-1995)
Figure 2.7	Linear Trend in Per Capita Consumption of Cigarettes in California Before and After Proposition 99 and Taxation Program

List of Tables:

Table 2.1	Influence of Puff Volume and Filter Ventilation on Deliveries of Particulate Matter and Carbon Monoxide in Mainstream and Sidestream Smoke
Table 2.2	Chemical Constituents of Tobacco Smoke that Have Been Classified or Identified as to Their Carcinogenicity, Reproductive Toxicity or Other Health Hazard
Table 2.3	Mean Concentrations of Nicotine and Cotinine in the Saliva, Plasma, and Urine of ETS-Exposed Volunteers
Table 2.4	Comparison of Biomarkers in Unexposed and ETS-Exposed Nonsmokers, and Active Smokers
Table 2.5	Cut-Off, Sensitivity, and Specificity of Biomarkers for Discriminating True Smoking Status
Table 2.6	Studies of Cotinine Measurements in Self-Reported Nonsmokers and Criteria Used to Distinguish Smokers From Nonsmokers

Table 2.7	Concentrations of Nicotine and Cotinine in Mothers' Milk
Table 2.8	Studies with Information on ETS Exposure Prevalence in California and the U.S.: Adults and Adolescents
Table 2.9	Studies with Information on ETS Exposure Prevalence in California and the U.S.: Infants and Children

3 Developmental Toxicity I: Perinatal Manifestations

List of Figures:

Figure 3.1:	Summary of Differences in Mean Birthweight and 95% Confidence Intervals Between ETS Exposed and Unexposed Pregnancies, by Definition of ETS and Study Size
Figure 3.2:	Odds Ratio (Log Scale) and 95% Confidence Interval for the Association of Low Birthweight and ETS, by Definition of ETS and Study Size

List of Tables:

Table 3.1:	Studies of Birthweight and ETS Exposure Defined by Paternal Smoking Status
Table 3.2:	Studies of Fetal Growth and ETS Exposure at Home Defined by Paternal Smoking Status
Table 3.3:	Studies of Fetal Growth and ETS Exposure of Maternal Non-Smokers from Multiple ETS Sources
Table 3.4:	Studies of Fetal Growth and ETS Exposure Determined by Biomarkers
Table 3.5:	ETS Exposure in Relation to Spontaneous Abortion and Perinatal Death
Table 3.6:	ETS Exposure and Congenital Malformations
Table 3.7:	Animal Studies of Tobacco Smoke Exposure and Fetal Growth

4 Developmental Toxicity II: Postnatal Manifestations

Table 4.1:	Sudden Infant Death Syndrome (SIDS): Studies that Assessed Some Source of Postnatal ETS Exposure
Table 4.2:	Cognition in Children: Studies that Assessed Some Source of Postnatal ETS Exposure
Table 4.3:	Behavior in Children: Studies that Assessed Some Source of Postnatal ETS Exposure
Table 4.4:	Height Growth in Children: Studies that Assessed Some Source of Postnatal ETS Exposure

5 Reproductive Effects

Table 5.1:	ETS Exposure and Infertility or Fecundability: Adult Exposure
Table 5.2:	ETS Exposure and Infertility or Fecundability: Childhood Exposure

6 Respiratory Health Effects

List of Tables

Table 6.1:	Studies Cited by U.S. EPA (1993) as Evidence Supporting a Causal Relation Between ETS Exposure and Increased Episodes and Severity of Asthma in Children
Table 6.2:	Controlled Exposures of Asthmatic Subjects to ETS
Table 6.3:	Studies of Middle Ear Effusion (MEE) and Otitis Media (OM) vs. ETS Exposure Reviewed by the Surgeon General (1986), NRC (1986), or U.S. EPA (1992).
Table 6.4:	Studies of Middle Ear Effusion (MEE) or Otitis Media (OM) vs. ETS Exposure Not Reviewed by the Surgeon General (1986), NRC (1986), or U.S. EPA (1992).
Table 6.5	Studies Excluded from Meta-Analysis of ETS and Childhood Asthma Induction
Table 6.6	ETS Exposure Relationship with Pulmonary Function, Hospitalizations and Disease Severity in Children with Cystic Fibrosis

List of Figures

Figure 6.1:	Reported risk ratios and 95% confidence intervals for studies that used clinically recognized asthma as an outcome
Figure 6.2:	Reported risk ratios and 95% confidence intervals for studies that used “wheezing bronchitis” or “chronic wheezing/whistling in the chest” as an outcome

7 Carcinogenic Effects

List of Tables

Table 7.1	Exposure to Spouse's Smoking and Relative Risk (RR) of All Cancers in Adults
Table 7.2A	Hair Concentrations of Nicotine and Cotinine in Women and Their Newborn Infants
Table 7.2B	4-Aminobiphenyl Hemoglobin Adduct Concentrations in Pregnant Women and Fetuses by Exposure to Tobacco Smoke
Table 7.2C	Cotinine and PAH-Albumin Levels in Mothers and Their Preschool Children
Table 7.3	Maternal Smoking During Index Pregnancy and Risk of All Childhood Cancers Combined
Table 7.4 Published	Study Characteristics of the Four U.S. Case-Control Studies of Lung Cancer and ETS Since 1991
Table 7.5	Association Between Risk of Lung Cancer in Lifetime Nonsmoking Females and Exposure to Spousal Smoking

Table 7.5B	Risk of Lung Cancer in Nonsmoking Women and Men: A Cohort Analysis
Table 7.6	Association Between Risk of Lung Cancer and ETS Exposures From Parents and Other Household Members
Table 7.7	Studies on ETS Exposure at the Workplace and Lung Cancer Among Lifetime Nonsmoking Subjects
Table 7.8	Association Between Passive Smoke Exposure and Risk of Nasal Sinus Cancer in Nonsmokers
Table 7.9	Relationship Between Active and Passive Smoke Exposure and Risk of Cervical Cancer
Table 7.10	Nicotine and Cotinine Measured in the Cervical Mucus of Smokers, Passive Smokers, and Nonsmokers
Table 7.11	Passive Smoking and Bladder Cancer Among Nonsmokers
Table 7.12	Mean Levels of Hemoglobin Adducts of 4- and 3-Aminobiphenyls in Nonsmokers
Table 7.13	Mean Levels of 4-ABP Hemoglobin Adducts (PG/G of Hemoglobin) Among Smokers and Nonsmokers by Acetylator Phenotype
Table 7.14	Brain Tumors in Children and Exposure to Parent's Smoking
Table 7.15	Maternal or Parental Smoking During Pregnancy and Childhood Leukemia
Table 7.16	Association Between Exposure to Passive Smoking and Risk of Non-Hodgkins Lymphoma and Lymphoma in Children

8 Cardiovascular Effects

List of Tables

Table 8.1	Cohort Studies on ETS Exposure and Heart Disease
Table 8.2	Case control Studies on ETS Exposure and Heart Disease
Table 8.3	Risks of Heart Disease and Active Smoking in Women
Table 8.4	Risks of Heart Disease and Active Smoking in Women By Age
Table 8.5	Effect of Exposure to ETS on Exercise Tolerance
Table 8.6	Effect of Exposure to ETS on Lipid Profile in Children
Table 8.7	Platelet Sensitivity to Antiaggregatory Prostaglandins Before and After Exposure to ETS
Table 8.8	Measures of Platelet Function in Relation to Exposure to Active Smoking and Passive Smoking

Table 8.9	Carotid Artery Intimal-Medial Thickness (IMT) as Measured by B-mode Ultrasound in Current Smokers, Exsmokers, Never Smokers
Table 8.10	Endothelium-Dependent Arterial Dilatation in Active Smokers, Never Smokers exposed to ETS, and Never Smokers Not Exposed

Executive Summary

Exposure to environmental tobacco smoke (ETS) has been linked to a variety of adverse health outcomes. Many Californians are exposed at home, at work and in public places. In the comprehensive reviews published as *Reports of the Surgeon General* and by the U.S. Environmental Protection Agency (U.S. EPA) and the National Research Council (NRC), ETS exposure has been found to be causally associated with respiratory illnesses, including lung cancer, childhood asthma and lower respiratory tract infections. Scientific knowledge about ETS-related effects has expanded considerably since the release of these reviews. The State of California has therefore undertaken a broad review of ETS, covering the major health endpoints potentially associated with ETS exposure: perinatal and postnatal manifestations of developmental toxicity, adverse impacts on male and female reproduction, respiratory disease, cancer, and cardiovascular disease. A “weight of evidence” approach has been used to describe the body of evidence to conclude whether or not ETS exposure is causally associated with a particular effect. Because the epidemiological data are extensive, they serve as the primary basis for assessment of ETS-related effects in humans. The report also presents an overview on measurements of ETS exposure, particularly as they relate to characterizations of exposure in epidemiological investigations, and on the prevalence of ETS exposure in California and nationally.

ETS, or “secondhand smoke”, is the complex mixture formed from the escaping smoke of a tobacco product, and smoke exhaled by the smoker. The characteristics of ETS change as it ages and combines with other constituents in the ambient air. Exposure to ETS is also frequently referred to as “passive smoking”, or “involuntary tobacco smoke” exposure. Although all exposures of the fetus are “passive” and “involuntary”, for the purposes of this review *in utero* exposure resulting from maternal smoking during pregnancy is not considered to be ETS exposure.

General Findings

ETS is an important source of exposure to toxic air contaminants indoors. There is also some exposure outdoors, in the vicinity of smokers. Despite an increasing number of restrictions on smoking and increased awareness of health impacts, exposures in the home, especially of infants and children, continue to be a public health concern. ETS exposure is causally associated with a number of health effects. Listed in Table ES.1 are the developmental, respiratory, carcinogenic and cardiovascular effects for which there is sufficient evidence of a causal relationship, including fatal outcomes such as sudden infant death syndrome and heart disease mortality, as well as serious chronic diseases such as childhood asthma. There are in addition effects for which evidence is suggestive of an association but further research is needed for confirmation. These include spontaneous abortion, cervical cancer, and exacerbation of asthma in adults (Table ES.1). Finally, it is not possible to judge on the basis of the current evidence the impact of ETS on a number of endpoints, including congenital malformations, changes in female fertility

and fecundability, male reproductive effects, rare childhood cancers and cancers of the bladder, breast, stomach, brain, hematopoietic system, and lymphatic system.

TABLE ES.1
HEALTH EFFECTS ASSOCIATED WITH EXPOSURE
TO ENVIRONMENTAL TOBACCO SMOKE

Effects Causally Associated with ETS Exposure

Developmental Effects

Fetal Growth: Low birthweight or small for gestational age
Sudden Infant Death Syndrome (SIDS)

Respiratory Effects

Acute lower respiratory tract infections in children
(*e.g.*, bronchitis and pneumonia)
Asthma induction and exacerbation in children
Chronic respiratory symptoms in children
Eye and nasal irritation in adults
Middle ear infections in children

Carcinogenic Effects

Lung Cancer
Nasal Sinus Cancer

Cardiovascular Effects

Heart disease mortality
Acute and chronic coronary heart disease morbidity

Effects with Suggestive Evidence of a Causal Association
with ETS Exposure

Developmental Effects

Spontaneous abortion
Adverse impact on cognition and behavior

Respiratory Effects

Exacerbation of cystic fibrosis
Decreased pulmonary function

Carcinogenic Effects

Cervical cancer

Many Californians are exposed to ETS, and the number of people adversely affected may be correspondingly large. Table ES.2 presents morbidity and mortality estimates for health effects causally associated with ETS exposure. For cancer, cardiovascular and some respiratory endpoints, estimates are derived from figures published for the U.S. population, assuming that the number affected in California would be 12% of the total. The estimates for middle ear infection, sudden infant death syndrome and low birthweight were derived using information on prevalence of ETS exposure in California and the U.S.

Relative risk estimates associated with some of these endpoints are small, but because the diseases are common the overall impact can be quite large. A relative risk estimate of 1.3 for heart disease mortality in nonsmokers is supported by the collective evidence; this corresponds to a lifetime risk of death of roughly 1 to 3% for exposed nonsmokers and approximately 4,000 deaths annually in California. The relative risk estimate of 1.2 to 1.4 associated with low birthweight implies that ETS may impact fetal growth of 1,200 to 2,200 newborns in California, roughly 1 to 2% of newborns of nonsmokers exposed at home or work. ETS may exacerbate asthma (RR \approx 1.6 to 2) in 48,000 to 120,000 children in California. Large impacts are associated with relative risks for respiratory effects in children such as middle ear infection (RR \approx 1.62), and lower respiratory disease in young children (RR \approx 1.5 to 2). Asthma induction (RR \approx 1.75 to 2.25) may occur in as many as 0.5 to 2% of ETS-exposed children. ETS exposure may be implicated in 120 SIDS deaths per year in California (RR \approx 3.5), with a risk of death to 0.1% of infants exposed to ETS in their homes. Lifetime risk of lung cancer death related to ETS-exposed nonsmokers may be about 0.7% (RR \approx 1.2). For nasal sinus cancers, observed relative risks have ranged from 1.7 to 3.0, but future studies are needed to confirm the magnitude of ETS-related risks.

Specific Findings and Conclusions

Exposure Measurement and Prevalence

ETS is a complex mixture of chemicals generated during the burning and smoking of tobacco products. Chemicals present in ETS include irritants and systemic toxicants such as hydrogen cyanide and sulfur dioxide, mutagens and carcinogens such as benzo(a)pyrene, formaldehyde and 4-aminobiphenyl, and the reproductive toxicants nicotine, cadmium and carbon monoxide. Many ETS constituents have been identified as hazardous by state, federal and international agencies. To date, over 50 compounds in tobacco smoke have been identified as carcinogens and six as developmental or reproductive toxicants under California's Proposition 65 (California Health and Safety Code 25249.5 *et seq.*).

Exposure assessment is critical in epidemiological investigations of the health impacts of ETS, and in evaluating the effectiveness of strategies to reduce exposure. Exposure can be assessed through the measurement of indoor air concentrations of ETS constituents, through surveys and questionnaires, or more directly through the use of personal monitors and the measurement of biomarkers in saliva, urine and blood. There are advantages and

TABLE ES.2
ESTIMATED ANNUAL MORBIDITY AND MORTALITY IN NONSMOKERS
ASSOCIATED WITH ETS EXPOSURE

Condition	Number of People or Cases ^a	
	in the U.S.	in California
Developmental Effects		
Low birthweight	≈ 9,700 - 18,600 cases ^b	≈ 1,200 - 2,200 cases ^b
Sudden Infant Death Syndrome (SIDS)	≈ 1,900 - 2,700 deaths	≈ 120 deaths
Respiratory Effects in Children		
Middle ear infection	0.7 to 1.6 million physician office visits	78,600 to 188,700 physician office visits
Asthma induction	8,000 to 26,000 new cases	960 to 3120 new cases
Asthma exacerbation	400,000 to 1,000,000 children	48,000 to 120,000 children
Bronchitis or pneumonia in infants and toddlers (18 months and under)	150,000 to 300,000 cases 7,500 to 15,000 hospitalizations ^c 136 - 212 deaths ^c	18,000 to 36,000 cases ^c 900 to 1800 hospitalizations ^c 16 - 25 deaths ^c
Cancer		
Lung	3000 deaths ^c	360 deaths ^c
Nasal sinus	N/A ^d	N/A ^d
Cardiovascular Effects		
Ischemic heart disease	35,000 - 62,000 deaths ^c	4,200 - 7,440 deaths ^c

^a The numbers in the table are based on maximum likelihood estimates of the relative risk. As discussed in the body of the report, there are uncertainties in these estimates, so actual impacts could be somewhat higher or lower than indicated in the table. The endpoints listed are those for which there is a causal association with ETS exposure based on observations of effects in exposed human populations.

^b California estimates for low birthweight, SIDS, and middle ear infection (otitis media) are provided in Chapters 3, 4, and 6, respectively. U.S. estimates are obtained by dividing by 12%, the fraction of the U.S. population residing in California.

^c Estimates of mortality in the U.S. for lung cancer and respiratory effects, with the exception of middle ear infection (otitis media), come from U.S. EPA (1992). U.S. range for heart disease mortality reflects estimates reported in Wells (1988 and 1994), Glantz and Parmley (1991), Steenland (1992). California predictions are made by multiplying the U.S. estimate by 12%, the fraction of the U.S. population residing in the State. Because of decreases in smoking prevalence in California in recent years, the number of cases for some endpoints may be somewhat overestimated, depending on the relative impacts of current versus past ETS exposures on the health endpoint.

^d Estimates of the impact of ETS exposure on the occurrence of nasal sinus cancers are not available at this time.

disadvantages associated with the various techniques, which must be weighed in interpreting study results. One important consideration in epidemiologic studies is misclassification of exposure. Studies on the reliability of questionnaire responses indicate qualitative information obtained is generally reliable, but that quantitative information may not be. Also, individuals are often unaware of their ETS exposure, particularly outside the home. In studies using both self-reporting and biological markers, the exposure prevalence was higher when determined using biological markers.

Available data suggest that the prevalence of ETS exposure in California is lower than elsewhere in the U.S. Among adults in California, the workplace, home and other indoor locations all contribute significantly to ETS exposure. For children the most important single location is the home. Over the past decade ETS exposures in California have decreased significantly in the home, workplace and in public places. Over the same period, restrictions on smoking in enclosed worksites and public places have increased (*e.g.*, Gov. Code, Section 19994.30 and California Labor Code, Section 6404.5) and the percentage of the adults who smoke has declined. Decreases in tobacco smoke exposure may not be experienced for some population subgroups, as patterns of smoking shift with age, race, sex and socioeconomic status. For example, from 1975 to 1988, the overall smoking prevalence among 16 to 18 year olds declined, but after 1988 the trend reversed.

Perinatal Manifestations of Developmental Toxicity

ETS exposure adversely affects fetal growth, with elevated risks of low birth weight or “small for gestational age” observed in numerous epidemiological studies. The primary effect observed, reduction in mean birthweight, is small in magnitude. But if the distribution of birthweight is shifted lower with ETS exposure, as it appears to be with active smoking, infants who are already compromised may be pushed into even higher risk categories. Low birthweight is associated with many well-recognized problems for infants, and is strongly associated with perinatal mortality.

The impact of ETS on perinatal manifestations of development other than fetal growth is less clear. The few studies examining the association between ETS and perinatal death are relatively non-informative, with only two early studies showing increased risk associated with parental smoking, and with the sparse data on stillbirth not indicative of an effect. Studies on spontaneous abortion are suggestive of a role for ETS, but further work is needed, particularly as a recent report did not confirm the findings of four earlier studies. Although epidemiological studies suggest a moderate association of severe congenital malformations with paternal smoking, the findings are complicated by the use of paternal smoking status as a surrogate for ETS exposure, since a direct effect of active smoking on sperm cannot be ruled out. In general, the defects implicated differed across the studies, with the most consistent association seen for neural tube defects. At this time, it is not possible to determine whether there is a causal association between ETS exposure and this or other birth defects.

Postnatal Manifestations of Developmental Toxicity

Numerous studies have demonstrated an increased risk of sudden infant death syndrome, or “SIDS,” in infants of mothers who smoke. Until recently it has not been possible to separate the effects of postnatal ETS exposure from those of prenatal exposure to maternal active smoking. Recent epidemiological studies now have demonstrated that postnatal ETS exposure is an independent risk factor for SIDS.

Although definitive conclusions regarding causality cannot yet be made on the basis of available epidemiological studies of cognition and behavior, there is suggestive evidence that ETS exposure may pose a hazard for neuropsychological development. With respect to physical development, while small but consistent effects of active maternal smoking during pregnancy have been observed on height growth, there is no evidence that postnatal ETS exposure has a significant impact in otherwise healthy children. As discussed in greater detail below, developmental effects of ETS exposure on the respiratory system include lung growth and development, childhood asthma exacerbation, and, in children, acute low respiratory tract illness, middle ear infection and chronic respiratory symptoms.

Female and Male Reproductive Toxicity

Though active smoking by women has been found to be associated with decreased fertility in a number of studies, and tobacco smoke appears to be anti-estrogenic, the epidemiological data on ETS exposure and fertility are not extensive and show mixed results, and it is not possible to determine whether ETS affects fecundability or fertility. Regarding other female reproductive effects, while studies indicate a possible association of ETS exposure with early menopause, the analytic methods of these studies could not be thoroughly evaluated, and therefore at present, there is not firm evidence that ETS exposure affects age at menopause. Although associations have been seen epidemiologically between active smoking and sperm parameters, conclusions can not be made regarding ETS exposure and male reproduction, as there is very limited information available on this topic.

Respiratory Effects

ETS exposure produces a variety of acute effects involving the upper and lower respiratory tract. In children, ETS exposure can exacerbate asthma, and increases the risk of lower respiratory tract illness, and acute and chronic middle ear infection. Eye and nasal irritation are the most commonly reported symptoms among adult nonsmokers exposed to ETS. Odor annoyance has been demonstrated in several studies.

Regarding chronic health effects, there is compelling evidence that ETS is a risk factor for induction of new cases of asthma as well as for increasing the severity of disease among children with established asthma. In addition, chronic respiratory symptoms in children, such as cough, phlegm, and wheezing, are associated with parental smoking.

While the results from all studies are not wholly consistent, there is evidence that childhood exposure to ETS affects lung growth and development, as measured by small, but statistically significant decrements in pulmonary function tests; associated reductions may persist into adulthood. The effect of chronic ETS exposure on pulmonary function in otherwise healthy adults is likely to be small, and unlikely by itself to result in clinically significant chronic disease. However, in combination with other insults (*e.g.*, prior smoking history, exposure to occupational irritants or ambient air pollutants), ETS exposure could contribute to chronic respiratory impairment in adults. In addition, regular ETS exposure in adults has been reported to increase the risk of occurrence of a variety of lower respiratory symptoms.

Children are especially sensitive to the respiratory effects of ETS exposure. Children with cystic fibrosis are likely to be more sensitive than healthy individuals. Several studies of patients with cystic fibrosis, a disease characterized by recurrent and chronic pulmonary infections, suggest that ETS can exacerbate the condition. Several studies have shown an increased risk of atopy (a predisposition to develop IgE antibodies against common allergens, which can then be manifested as a variety of allergic conditions) in children of smoking mothers, though the evidence regarding this issue is mixed.

Carcinogenic Effects

The role of ETS in the etiology of cancers in nonsmokers was explored, as smoking is an established cause of a number of cancers (lung, larynx, oral cavity, esophagus and bladder), and a probable cause of several others (cervical, kidney, pancreas, and stomach). Also, ETS contains a number of constituents which have been identified as carcinogens.

Reviews published in the 1986 *Report of the Surgeon General*, by the National Research Council in 1986, and by the U.S. EPA in 1992 concluded that ETS exposure causes lung cancer. Three large U.S. population-based studies and a smaller hospital-based case control study have been published since the completion of the U.S. EPA review. The population-based studies were designed to and have successfully addressed many of the weaknesses for which the previous studies on ETS and lung cancer have been criticized. Results from these studies are compatible with the causal association between ETS exposure and lung cancer already reported by the U.S. EPA, Surgeon General, and National Research Council. Of the studies examining the effect of ETS exposure on nasal sinus cancers, all three show consistent associations, presenting strong evidence that ETS exposure increases the risk of nasal sinus cancers in nonsmoking adults. Further study is needed to characterize the magnitude of the risk of nasal sinus cancer from ETS exposure.

The epidemiological and biochemical evidence suggest that exposure to ETS may increase the risk of cervical cancer. Positive associations were observed in two of three case-control studies and a statistically nonsignificant positive association was observed in the only cohort study conducted. Findings of DNA adducts in the cervical epithelium as

well as nicotine and cotinine in the cervical mucus of ETS-exposed nonsmokers provides biological plausibility.

For other cancer sites in adults, there has been limited ETS-related epidemiological research in general: there is currently insufficient evidence to draw any conclusion regarding the relationship between ETS exposure and the risk of occurrence. A review of the available literature clearly indicates the need for more research. For example, although compounds established as important in the etiology of stomach cancer are present in tobacco smoke, only a single cohort study has been performed for this site. Precursors of endogenously formed N-nitroso compounds suspected of causing brain tumors are present in high concentrations in ETS, and the one cohort and two case-control studies available suggest a positive association, but the results are based on small numbers and may be confounded by active smoking. In biochemical studies of nonsmokers, higher levels of hemoglobin adducts of the established bladder carcinogen, 4-aminobiphenyl, have been found in those exposed to ETS. However, no significant increases in bladder cancer were seen in the two epidemiological studies (case-control) conducted to date, although both studies were limited in their ability to detect an effect. Several compounds in tobacco smoke are associated with increased risk of leukemia, but only one small case-control study in adults, reporting an increased risk with ETS exposure during childhood, has been performed. Finally, all four studies on ETS exposure and breast cancer suggest an association, but in two of the studies the associations were present only in select groups, and in three studies there is either no association between active smoking and the risk of breast cancer or the association for active smoking is weaker than for passive smoking. Moreover, there is no indication of increasing risk with increasing intensity of ETS exposure. Still, results from a recent study suggest that tobacco smoke may influence the risk of breast cancer in certain susceptible groups of women, and this requires further investigation.

Regarding childhood cancers, it is unclear whether parental smoking increases risk overall, or for specific cancers such as acute lymphoblastic leukemia and brain tumors, the two most common cancers in children. The lack of clarity is due to the conflicting results reported and the limitations of studies finding no association. The epidemiological data on ETS exposure and rare childhood cancers also provide an inadequate foundation for making conclusions regarding causality. Some studies found small increased risks in children in relation to parental smoking for neuroblastoma, Wilm's tumor, bone and soft-tissue sarcomas, but not for germ cell tumors. Studies to date on these rare cancers have been limited in their power to detect effects. The impact of ETS exposure on childhood cancer would benefit from far greater attention than it has received to date.

Cardiovascular Effects

The epidemiological data, from prospective and case-control studies conducted in diverse populations, in males and females and in western and eastern countries, are supportive of a causal association between ETS exposure from spousal smoking and coronary heart

disease (CHD) mortality in nonsmokers. To the extent possible, estimates of risk were determined with adjustment for demographic factors, and often for other factors related to heart disease, such as blood pressure, serum cholesterol level and obesity index. Risks associated with ETS exposure were almost always strengthened by adjustment for other cofactors. For nonsmokers exposed to spousal ETS compared to nonsmokers not exposed, the risk of CHD mortality is increased by a factor of 1.3. The association between CHD and risk is stronger for mortality than for non-fatal outcomes, including angina.

Data from clinical studies suggest various mechanisms by which ETS causes heart disease. In a number of studies in which nonsmokers were exposed to ETS, carotid wall thickening and compromise of endothelial function were similar to, but less extensive than those experienced by active smokers. Other effects observed include impaired exercise performance, altered lipoprotein profiles, enhanced platelet aggregation, and increased endothelial cell counts. These findings may account for both the short- and long-term effects of ETS exposure on the heart.

Attachment I

Review of the OEHHA Assessment of Environmental Tobacco Smoke by the Scientific Review Panel (SRP)

Interest in the health effects of second hand tobacco smoke on the part of members of the Scientific Review Panel (SRP) on Toxic Air Contaminants led to a request by the SRP for a health assessment of environmental tobacco smoke, and a collaborative agreement between the Office of Environmental Health Hazard Assessment (OEHHA) and the Air Resources Board (ARB) to initiate such an assessment. SRP members reviewed the drafts as they were developed and participated in each of the workshops held as the document underwent public review (see Preface for details). The Final Draft reflected the input of SRP members, as well as that of other reviewers.

Specific changes made at the request of the SRP following its review of the Final Draft include the addition of new studies (e.g., the results of Kawachi *et al.*'s analysis of cardiovascular disease risk in the Nurse's Health study, published after the release of the Final Draft, in which it was reported as an abstract), a discussion of issues related to misclassification of smoking status and cancer risk, and clarifying language in the presentation of attributable risk estimates; minor editorial changes were also requested and made. The SRP discussed the assessment and made findings on the health effects of exposure to environmental tobacco smoke as a result of its review; these findings are included in this Attachment.