SPECIAL REPORT

In September 1997, the California Environmental Protection Agency (Cal-EPA) released its final report on Health effects of exposure to environmental tobacco smoke. The report, in draft form, was reviewed in a news article by Don Shopland in the Summer 1997 issue of Tobacco Control (1997;6:87). Issuance of the final report follows an extensive process of manuscript preparation, scientific peer review, public comment, revision, and review by the Scientific Review Panel on Toxic Air Contaminants (SRP).

Described by Shopland as "the most comprehensive report to date on the topic", it contains more than 500 pages, eight chapters, two appendices, 60 tables, and 11 figures. The full report is available on the world wide web at <http://www.calepa.ca.gov/oehha/docs/finalets.htm>. The preface and executive summary of the report, and the findings of the SRP, are reproduced below. Hard copies of the full report may be obtained by contacting Ms Joyce Smylie at +1 510 540 2084 (tel), or +1 510 540 2695 (fax), or by writing to: Cal-EPA, Office of Environmental Health Hazard Assessment, 301 Capitol Mall, 2nd Floor, Sacramento, California 95814-4327, USA.

The report confirms causal relationships between environmental tobacco smoke (ETS) exposure and lung cancer, and between ETS and several conditions in children (respiratory tract infections, middle ear infections, asthma exacerbation), which have been reported previously by the US Surgeon General, the US Environmental Protection Agency, the National Research Council, and other authorities. The Cal-EPA report concludes, however, that more recent evidence allows a conclusion that ETS is causally related to fetal growth retardation, sudden infant death syndrome, asthma induction in children, nasal sinus cancer, heart disease mortality, and acute and chronic coronary heart disease morbidity. A particularly valuable contribution of the report is its quantification of the estimated annual morbidity and mortality in non-smokers associated with ETS exposure (see table ES.2 below).—ED

Health effects of exposure to environmental tobacco smoke

California Environmental Protection Agency

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 (OEHHHA) 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 OEHHHA 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.

OEHHHA, 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.

OEHHHA 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 an 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:


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."

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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 US Environmental Protection Agency (US 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.

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 US 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 also derived using information on prevalence of ETS exposure in California and the US.

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 = 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 = 1.62), and lower respiratory disease in young children (RR = 1.5 to 2). Asthma induction (RR = 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 = 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 = 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.).

**Table ES.2 Estimated annual morbidity and mortality in nonsmokers associated with ETS exposure**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Number of people or cases in the U.S.</th>
<th>Number of people or cases in California</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Developmental effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low birthweight</td>
<td>9700–18,600 cases</td>
<td>1200–2200 cases</td>
</tr>
<tr>
<td>Sudden Infant Death Syndrome (SIDS)</td>
<td>1900–2700 deaths</td>
<td>120 deaths</td>
</tr>
<tr>
<td><strong>Respiratory effects in children</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Middle ear infection</td>
<td>78,600 to 188,700</td>
<td>78,600 to 188,700</td>
</tr>
<tr>
<td>Asthma induction</td>
<td>960 to 3120 new cases</td>
<td>960 to 3120 new cases</td>
</tr>
<tr>
<td>Asthma exacerbation</td>
<td>18,000 to 120,000 children</td>
<td>18,000 to 120,000 children</td>
</tr>
<tr>
<td>Bronchitis or pneumonia in infants and toddlers (18 months and under)</td>
<td>8 to 10 million</td>
<td>8 to 10 million</td>
</tr>
<tr>
<td>and (hospitalizations)</td>
<td>136–212 deaths</td>
<td>136–212 deaths</td>
</tr>
<tr>
<td>Cancer</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lung</td>
<td>300 deaths</td>
<td>360 deaths</td>
</tr>
<tr>
<td>Nasal sinus</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td><strong>Cardiovascular effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>35,000–62,000 deaths</td>
<td>4200–7440 deaths</td>
</tr>
</tbody>
</table>

*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.

*California estimates for low birthweight, SIDS, and middle ear infection (otitis media) are provided in Chapters 3, 5, and 6, respectively. US estimates are obtained by dividing by 10, the fraction of the US population residing in California.

*Estimates of mortality in the US for lung cancer and respiratory effects, with the exception of middle ear infections (otitis media), come from US EPA (1992). US range for heart disease mortality reflects estimates reported in Wells (1988 and 1992), Glantz and Parmley (1991), Steenland (1992). California predictions are made by dividing by 10, the fraction of the US 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 heart endpoint.

*Estimates of the impact of ETS exposure on the occurrence of nasal sinus cancers are not available at this time.
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 between ETS exposure 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 between ETS exposure 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 between ETS exposure and perinatal death.

**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 no firm evidence that ETS exposure affects age at menopause. Although associations have been seen epidemiologically between active smoking and sperm parameters, conclusions cannot 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 US EPA in 1992 concluded that ETS exposure causes lung cancer. Three large US population-based studies and a smaller hospital-based case control study have been published since the completion of the US 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 US 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 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 commonly discussed. 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, Wilms tumor, bone and soft-tissue sarcomas, but not for germ cell tumors. Studies to date on these rare cancers have been limited in their ability 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.

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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.

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Findings of the Scientific Review Panel on health effects of exposure to environmental tobacco smoke as adopted at the Panel's June 19, 1997 meeting

The Scientific Review Panel (SRP/Panel) has reviewed the report “Health Effects of Exposure to Environmental Tobacco Smoke” prepared by the Office of Environmental Health Hazard Assessment (OEHHA). The Panel members also reviewed the public comments received on this report. Based on this review, the SRP makes the following findings:

1. Environmental Tobacco Smoke (ETS) is an important source of exposure to toxic air contaminants. Thus, despite an increasing number of restrictions on smoking and increased awareness of health impacts, exposures continue to be a major public health concern.

2. A causal association exists between ETS exposure from spousal smoking and coronary heart disease (CHD) mortality in nonsmokers. 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. Heart disease is the primary fatal endpoint from ETS exposure.

3. 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, mutagens and carcinogens, and reproductive and developmental toxicants. 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.) and twelve have been identified as a toxic air contaminant under AB 1807.


5. Available data suggest that the prevalence of ETS exposure in California is lower than elsewhere in the US. Nevertheless, 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.

6. 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 birth weight, is small in magnitude. If the distribution of birth weight 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 birth weight is associated with many well-recognized problems for infants and is strongly associated with perinatal mortality.

7. 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 cause.
8. 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.

9. 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.

10. The effect of chronic ETS exposure on pulmonary function in otherwise healthy adults is likely to be small. 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 (e.g. bronchitis and wheezing apart from colds).

11. 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.

12. 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.

13. 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.

14. Studies on ETS exposure and breast cancer suggest an association, but the associations were present only in select groups, or 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. However, 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.

15. In summary, ETS exposure is causally associated with a number of fatal and non-fatal health effects. Heart disease mortality, sudden infant death syndrome, and lung and nasal sinus cancer have been causally linked to ETS exposure. Serious impacts of ETS on the young include childhood asthma induction and exacerbation, bronchitis and pneumonia, middle ear infection, chronic respiratory symptoms, and low birth weight. In adults, acute and chronic heart disease morbidity is causally associated with ETS exposure. ETS also causes eye and nasal irritation and odor annoyance.

16. Effects for which evidence is suggestive of an association, but further research is needed for confirmation, include: spontaneous abortion, adverse neuropsychological development, cervical cancer, exacerbation of cystic fibrosis, and decreased pulmonary function.

17. 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.

18. Many Californians are exposed to ETS, and the number of people adversely affected is correspondingly large. Each year ETS contributes to asthma exacerbation in 18,000 to 120,000 children, 960 to 3,120 new cases of asthma in children, 78,600 to 188,700 physicians office visits due to middle ear infections in children, 18,000 to 36,000 cases and 900 to 1,800 hospitalizations from bronchitis or pneumonia in toddlers and infants, and 1,200 to 2,200 cases of low birth weight. Annual mortality estimates associated with ETS exposure in California are: approximately 120 deaths from SIDS, 16-25 deaths to toddlers and infants from bronchitis and pneumonia, approximately deaths from lung cancer, and 4,200-7,440 deaths from ischemic heart disease. Thus, ETS has a major public health impact.

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After careful review of the February 1997 draft of the OEHHA report, *Health Effects of Exposure to Environmental Tobacco Smoke*, we find the draft, with the changes specified by OEHHA in our June 19, 1997 meeting, as representing a complete and balanced assessment of current scientific understanding. Based on the available evidence we conclude ETS is a toxic air contaminant.