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 the above-mentioned 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, in which the body of evidence is examined to determine whether or not it can be concluded that 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 burning 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

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

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 U.S. population, assuming that the number affected in California would be 12 percent 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 (RR) 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 estimate corresponds to a lifetime risk of death of roughly 1 to 3 percent 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 percent of newborns of nonsmokers exposed at home or at 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 percent 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 approaching 0.1 percent for infants exposed to ETS in their homes. Lifetime risk of lung cancer death related to ETS-exposed nonsmokers may be about 0.7 percent (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 identified 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 ques-

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[b]	120 deaths[b]
Respiratory Effects in Children		
Middle ear infection	0.7 to 1.6 million physician office visits[b]	78,600 to 188,700 physician office visits[b]
Asthma induction	8,000 to 26,000 new cases[c]	960 to 3,120 new cases[c]
Asthma exacerbation	400,000 to 1,000,000 children[c]	48,000 to 120,000 children[c]
Bronchitis or pneumonia in infants and toddlers (18 months and under)	150,000 to 300,000 cases[c]	18,000 to 36,000 cases[c]
	7,500 to 15,000 hospitalizations[c]	900 to 1,800 hospitalizations[c]
	136 - 212 deaths[c]	16 - 25 deaths[c]
Cancer		
Lung	3,000 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 percent, 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 percent, 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.*

tionnaires, or more directly through the use of personal monitors and the measurement of biomarkers in saliva, urine, and blood. There are advantages and 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 that 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 stud-

ies, 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 lower 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 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 is 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 suggests 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 non-significant positive association was observed in the only cohort study con-

ducted. 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, an association which requires further investigation.

Regarding childhood cancers, it is unclear whether parental smoking increases risk, either overall or for specific cancers such as acute lymphoblastic leukemia or 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 in children found small increased risks 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—factors 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 wherein 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. 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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DEPARTMENT OF CHEMISTRY IRVINE, CALIFORNIA 92717-2025

July 18, 1997

Richard Becker, Ph.D.
Director
Office of Environmental
Health Hazard Assessment
301 Capitol Mall, Second Floor
Sacramento, California 95814

Dear Dr. Becker:

On behalf of the Scientific Review Panel (SRP/Panel) I am pleased to transmit to you our Findings as a result of our review of the Office of Environmental Health Hazard Assessment (OEHHA) final report "Health Effects of Exposure to Environmental Tobacco Smoke" (ETS).

As you will see in a review of the SRP meeting transcript, the Panel is very impressed with the quality of the report and view it as the most current and definitive statement of the science applicable to ETS. As we noted OEHHA staff scientists are to be highly commended for this successful completion.

We are also pleased that the Air Resources Board (ARB) is considering holding an "informational hearing" on the report. As you will see in the enclosed Findings, the Panel views ETS as a toxic air contaminant, and it has a major impact on public health.

If the Panel may be of further help as this health risk is addressed in California, we would be pleased to do so.

We trust our Findings and this transmittal letter will be made a part of the final report

Sincerely,

James N. Pitts, Jr. Ph.D.
Chairman
Scientific Review Panel

Enclosure

cc: John D. Dunlap, Chairman, ARB
Scientific Review Panel Members
Bill Lockett, ARB

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.

4. The 1986 *Report of the Surgeon General,* the 1986 National Research Council report *Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects*, and the 1992 U.S. EPA report *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders* have established that ETS exposure causes lung cancer. Results from recent epidemiological studies are compatible with the causal association already established.

5. Available data suggest that the prevalence of ETS exposure in California is lower than elsewhere in the U.S. 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 of SIDS.

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 48,000 to 120,000 children, 960 to 3120 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 1800 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 in toddlers and infants from bronchitis and pneumonia, approximately 360 deaths from lung cancer, and 4,200 - 7,440 deaths from ischemic heart disease. Thus, ETS has a major public health impact.

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.

I certify that the above is a true and correct copy of the findings adopted by the Scientific Review Panel on June 19, 1997

James N. Pitts, Jr., Ph.D.
Chairman, Scientific Review Panel

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