California Environmental Protection Agency
Air Resources Board

INITIAL STATEMENT OF REASONS FOR RULEMAKING

STAFF REPORT

PROPOSED IDENTIFICATION OF ENVIRONMENTAL TOBACCO SMOKE AS A TOXIC AIR CONTAMINANT

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State of California
AIR RESOURCES BOARD

STAFF REPORT: INITIAL STATEMENT OF REASONS FOR PROPOSED RULEMAKING

PROPOSED IDENTIFICATION OF ENVIRONMENTAL TOBACCO SMOKE AS A TOXIC AIR CONTAMINANT

To be considered by the Air Resources Board on January 26, 2006, at:

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Initial Statement of Reasons
for the Proposed Identification of
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I. INTRODUCTION

In accordance with California Health and Safety Code sections 39660-39662, the Air Resources Board (ARB or Board) staff is recommending that the Board identify environmental tobacco smoke (ETS) as a toxic air contaminant (TAC). Appendix I contains the proposed regulatory amendment.

This Initial Statement of Reasons for Rulemaking for the Proposed Identification of ETS as a TAC consists of:

1) An ARB Staff Report, which summarizes the scientific basis for the proposed regulation and includes a discussion of the environmental and economic impacts of the proposal;
2) Appendix I (the Proposed Regulation Order);
3) Appendix II (the Findings of the Scientific Review Panel [SRP]); and
4) Appendix III (the SRP-approved version of the Executive Summary and the three-part report that contains the analysis of the exposure and health assessments of ETS, Parts A, B, C, as approved at the June 24, 2005 SRP meeting).

Part A, prepared by ARB staff, is an evaluation of emissions of, and exposure to, ETS. Part B, prepared by the Office of Environmental Health Hazard Assessment (OEHHA) staff, assesses the health effects of ETS. Part C consists of copies of the public comments received on the March 2005 draft report, and ARB/OEHHA staff responses.

II. BACKGROUND

A. Definition of a Toxic Air Contaminant

Section 39655 of the California Health and Safety Code defines a TAC as “an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health.” California Health and Safety Code section 39013 defines air pollutant as “any discharge, release, or other propagation into the atmosphere and includes, but is not limited to, smoke, charred paper, dust, soot, grime, carbon, fumes, gases, odors, particulate matter, acids, or any combination thereof.” In addition, the Hazardous Air Pollutants listed in section 7412 of title 42 of the United States Code have also been identified as TACs under the state’s air toxics program pursuant to section 39657(b) of the California Health and Safety Code.

B. The California Program for Identification and Control of Toxic Air Contaminants

The Toxic Air Contaminant Identification and Control Program was established by a California law, Assembly Bill 1807 (AB 1807, Tanner, Chapter 1047, statutes of 1983, Health and Safety Code section 39650 *et seq*., Food and Agriculture Code section 14021 *et seq*.). AB 1807 created a comprehensive program administered by ARB to reduce the potential adverse public health impacts caused by emissions of toxic substances to the ambient air.

AB 1807 established a two-phased process which separates risk assessment (identification) from risk management (control). During the identification phase, a report is developed which determines whether there may be potential adverse health effects from substances in consideration of their toxicity, quantities of emissions, and human exposure in California. If the Board formally identifies a substance as a TAC, the substance enters the risk management phase. In the risk management phase, ARB determines the need for and appropriate degree of controls in consideration of costs and potential health benefits. The identification phase and the control phase are open public processes in which ARB staff actively seeks industry and public participation. (Health and Safety Code section 39660-39666).

In setting priorities for substances to enter the AB 1807 identification process, ARB must consider factors relating to: 1) risk of harm to public health; 2) amount or potential amount of emissions; 3) manner of, exposure to, and usage of the substance in California; 4) persistence in the atmosphere; and 5) ambient concentrations in the community. (Health and Safety Code section 39660(f).)

Once a substance is entered into the identification process, ARB and OEHHA staffs prepare a report that serves as the basis for listing the substance as a TAC. Health and Safety Code section 39660 requires OEHHA, upon request of ARB, to evaluate the health effects of a potential TAC while ARB evaluates the exposure data associated with it.

ARB's exposure assessment is based, to the extent available, upon research and monitoring data, and information on estimated actual exposures from data on ambient and indoor air environments. (Health and Safety Code section 39660(f).)

OEHHA's health evaluation includes an assessment of the availability and quality of data on the substance's health effects, including its potency and mode of action. Where it can be established that a threshold of adverse health effects exists, OEHHA must identify a safe exposure level. If there is no threshold of significant health effects, a range of risk for exposure to the substance is determined. (Health and Safety Code section 39660(c)). In both cases, OEHHA provides a full explanation of any uncertainties associated with the data.

The report, together with the scientific data on which the report is based, is made available to the public and is formally reviewed by the Scientific Review Panel (SRP) pursuant to Health and Safety Code section 39661. The SRP is composed of nine members, one each with recognized scientific expertise in the field of oncology, pathology, epidemiology, atmospheric science, biostatistics, occupational medicine, toxicology, and biochemistry (or molecular biology), and one member with relevant scientific experience who is experienced in the operation of scientific review or advisory bodies. The SRP reviews the scientific procedures and methods used to support the data, the data itself, and the conclusions and assessments on which the report is based. The SRP conducts all of its business at noticed meetings that are open to the public.

If the SRP approves the report, it adopts formal findings. If the SRP determines that the report is not based on sound scientific knowledge, methods, or practices, it will return the report for revision and resubmittal. Once the SRP has reviewed and approved a report, it transmits the report with the SRP-adopted findings to ARB. The Board conducts a public hearing to determine, based on the staff's report and the SRP's findings, if a substance should be listed as a TAC. If the Board decides to list the substance as a TAC, it is added to section 93000 of the California Code of Regulations. Health and Safety Code section 39665(a) then requires ARB to prepare a report that assesses the need for and appropriate degree of control for that substance.

III. DEVELOPMENT OF THE REPORT ON ENVIRONMENTAL TOBACCO SMOKE

Environmental tobacco smoke entered the TAC identification process in June 2001 and has undergone a thorough and extensive evaluation. ETS was entered into the process because it has potential cancer and non-cancer health effects, serious impacts on infants and children, and widespread exposure in California. ARB and OEHHA gave priority to the evaluation of ETS emissions because it met the TAC program criteria related to potential risk of harm to public health, amount of emissions, exposure and use, and persistence in the atmosphere.

Some of the information in this report is based upon data presented in OEHHA's 1997 report: *"Health Effects of Exposure to Environmental Tobacco Smoke."* The National Cancer Institute, acting for the U.S. Public Health Service, recognized the importance of the 1997 OEHHA report and incorporated it into their Smoking and Tobacco Control Monograph series.

On December 17, 2003, the first draft TAC identification report was released to the public for a 90-day comment period. On March 15, 2004, a public workshop was held to discuss the report. The SRP held a meeting on November 30, 2004, to discuss the first SRP version of the report "Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant" developed by ARB and OEHHA. Comments and responses to comments on the first draft TAC identification report were also discussed.

Several SRP meetings were to follow. On January 6, 2005, comments from the November 30, 2004, SRP meeting were addressed, such as indoor versus outdoor emissions, verification of referenced studies, and expanded discussion of ETS physical characteristics over time. On March 14, 2005, ARB and OEHHA discussed the second SRP version of the report with the SRP. Discussions involved various aspects of the Part B – Health Effects of the ETS report. A third SRP draft version of the report was prepared for the June 24, 2005, SRP meeting which addressed the remaining SRP issues. The SRP approved the draft report and agreed on its findings. In its findings, the SRP concludes that ETS should be listed as a TAC and that the report, with the revisions requested by the Panel, is based on sound scientific knowledge. The SRP further recommended that should ARB list ETS as a TAC, it also be added by OEHHA to the list of TACs that may disproportionately impact children. Appendix II contains the SRP's findings as adopted at the June 24, 2005, meeting.

IV. ENVIRONMENTAL TOBACCO SMOKE IN CALIFORNIA

ARB staff reviewed and evaluated the potential for environmental tobacco smoke exposure in California (Part A of the ETS report). ARB staff considered the estimated emissions inventory and ambient concentrations. The SRP approved ARB staff's exposure assessment at its June 24, 2005, meeting.

A. Description of ETS

ETS is a complex mixture of thousands of gases and fine particulate matter emitted by the burning of tobacco products and from smoke exhaled by the smoker. Other minor contributors to ETS are from the smoke that escapes while the smoker inhales and some vapor-phase related compounds that diffuse from the wrapper of the tobacco product. The composition will vary depending on heat of combustion, tobacco content and additives present, and type of filter material used.

Researchers distinguish cigarette smoke as being comprised of two main components: mainstream and sidestream smoke. Mainstream smoke is material that is drawn through the mouthpiece of a burning cigarette while sidestream smoke is material that is emitted from a smoldering cigarette between puffs. ETS is a combination of exhaled mainstream smoke, sidestream smoke, and compounds that diffuse through the cigarette paper.

Many of the substances found in ETS have known adverse health effects. Tables 1 and 2 list some of the gas phase and particulate matter components found in ETS with notable health effects.

Table 1. Gas Phase Components in ETS with Known Health Effects

Constituent	Non-Cancer Health Effects	Constituent	Non-Cancer Health Effects
1,3-Butadiene	irritant, neurological effects	Hydrazine	hepatotoxic, dermatitis
Acetaldehyde	irritant, dermatitis	Methanol	neurotoxicant, irritant
Acetone	irritant, dizziness	Methyl chloride	CNS depressant, fatigue
Acetonitrile	irritant, cause vomiting	N-Nitrosodiethylamine	
Acrolein	irritant, pulmonary edema	N-Nitrosodimethylamine	causes liver damage
Benzene	CNS depressant, nausea	N-Nitrosopyrrolidine	
Carbon monoxide	headache, dizziness	Pyridine	irritant, dizziness
Carbonyl sulfide	irritant, CNS depressant	Styrene	CNS depressant, irritant
Ethyl benzene	irritant, CNS depressant	Toluene	CNS depressant, irritant
Formaldehyde	irritant, induce asthma		

Table 2. Particulate Matter Components in ETS with Known Health Effects

Constituent	Non-Cancer Health Effects	Constituent	Non-Cancer Health Effects
2-Naphthylamine	irritant, dizziness	Dibenzo[a,l]pyrene	
2-Toluidine	CNS depressant	Hydroquinone	CNS excitation, tinnitus
4-Aminobiphenyl	hematuria, lethargy	Lead	affects CNS, depression
Aniline	methemoglobinemia	N'-Nitrosonornicotine	
Arsenic (inorganic)	hemolysis, neuropathy	Nickel	immune alterations, irritant
Benz[a]anthracene		Nicotine	
Benzo[a]pyrene	dermatitis, irritant	N-Nitrosodiethanolamine	
Cadmium	bronchiolitis, irritant	NNK	
Catechol	methemoglobinemia	Phenol	cardiac arrthythmias
Chromium VI	renal toxicity, hemolysis	Quinoline	irritant, nausea, coma
Dibenzo[a,i]pyrene			

Typical ETS particles range from 0.01 to about 1 micrometer (µm). Freshly produced ETS undergoes complex atmospheric changes such as coagulation, evaporation, dilution, and condensation. However, ETS fine particles essentially remain below 1 µm in size.

B. ETS Emissions and Smoking Trends

ETS emission estimations were determined through cigarette sales in California, smoking prevalence, and emission factors. ETS emissions were characterized using the most commonly measured components of ETS, such as nicotine, respirable suspended particulates (RSP), and carbon monoxide (CO). In 2002, over 420 billion cigarettes, 6.3 billion large and small cigars, and 9.3 million pounds of smoking tobacco (pipe and "roll your own" cigarettes) were consumed nationwide. However, cigarettes comprised 85% of tobacco products and are the main contributor to ETS.

Total statewide ETS emissions for nicotine, RSP, and CO were estimated from cigarettes and cigars for 2002 (Table 3).

Table 3. 2002 California Statewide ETS Emissions (Tons/Year)

	Cigarettes	Cigars	Total
Nicotine	36	4	40
RSP	335	30	365
CO	1475	432	1907

The amount of ETS emitted into the outdoor environment depends in large part on the smoking public's behavior. Outdoor ETS emissions include direct emissions from outdoor smoking, plus ETS emissions generated indoors which eventually ventilate outside. In California, with the enactment of Assembly Bill 13 (AB 13) in 1998, the majority of all workplaces and other public venues, such as bars and restaurants, prohibit indoor smoking. Furthermore, according to the 2002 California Adult Tobacco Survey, half of California smoker residences have indoor smoking bans. Therefore, we assume that most physical smoking occurs outdoors. For ETS generated indoors, building ventilation studies show that 50 – 80% of ETS (including ETS constituents) is exchanged with outdoor air over a given time period. From all of the available information, ARB staff estimates that at least 80% of total ETS emissions (including those directly emitted outdoors and emissions ventilated from indoors) are emitted to the outdoor environment.

The California Department of Health Services (CDHS) conducts surveys regarding smoking and tobacco use through the implementation of Proposition 99, the Tobacco Tax and Health Protection Act of 1988, and other California laws which reauthorized provisions of Proposition 99. Researchers have measured data on smoking prevalence, attitudes, behaviors, and exposure for years through the use of detailed questionnaire surveys. Data is compiled for various subpopulations according to age, ethnicity, educational background, and several other categories. The CDHS gathers important information about smoking behavior through the California Tobacco Surveys (CTS) and the California Student Tobacco Surveys (CSTS). The CTS are random-participation telephone surveys targeting various groups, including adolescents (12 – 17 years) and adults (18+ years). The CSTS is an in-school student survey of tobacco use which collects data from both middle (grades 6 – 8) and high school (grades 9 – 12) students.

The most recent CTS and CSTS surveys show that both the adult (2002 data) and adolescent (2001 data) smoking prevalence is about 16%. Below is Table 4 of some survey categories for smoking prevalence.

Table 4. Current Adult and Adolescent Smoking Prevalence (%)

Category	Adult	Adolescent
Male	19.5	16.2
Female	13.0	15.7
African-American	19.0	8.2
Asian/PI	12.1	13.6
Hispanic	13.4	14.0
Caucasian	17.3	19.9
Overall	**16.2**	**16.0**

In 2002, California had a low smoking adult prevalence (16.2%) rate compared to the overall United States (U.S.) prevalence (23.0%). In fact, the U.S. per capita cigarette consumption (74.6 packs per fiscal year) is over twice as high as California's (35.8 packs per fiscal year). This explains why California only contributed a small percentage (≈ 6.0%) of the total ETS emissions.

C. Exposure to ETS

An individual's exposure is dependent on the air concentration of a pollutant in a given environment, and the time they spend in that environment. An individual's total daily exposure is the sum of the many exposures they experience across their 24-hour day, including both indoor and outdoor environments. Thus, exposure may be heavily influenced by an individual's activity patterns if they routinely visit a location where smoking occurs, or if they live in a smoking household.

Smoking behavior and other factors that change smoking patterns such as smoking regulations and smoking customs may affect present and future exposure patterns. Information from several smoking behavior related surveys indicate that many of California's adults, adolescents, and children are exposed to ETS during some time of the day. According to earlier studies before AB 13, 56% of adults (over age 18), 64% of adolescents (12-17 years), and 38% of children (0-11 years), reported exposure to ETS during their daily activity. Since the enactment of AB 13 in 1998, actual incidence is assumed to be lower today due to decreases in workplace smoking and public locations such as restaurants, bars, and gaming clubs. However, up to 20% of adolescents may still be exposed to ETS in their homes.

Tobacco smoke is a complex mixture and cannot be measured directly. Due to the complex nature of ETS, it is necessary to select a surrogate measure of exposure that is representative of ETS as a whole. Several components of ETS have been studied as markers for ETS. Nicotine has been most widely studied as a potential marker because its source is primarily tobacco smoke. Nicotine has been used as a pesticide, but only in very limited locations and applications. Sampling and analysis methods are well documented for nicotine, as demonstrated by several authors. Other ETS markers that have been studied include: solanesol, 3-ethenylpyridine (3-EP), carbon monoxide,

iso- and anteisoalkanes (C_{29}-C_{34}), polycyclic aromatic hydrocarbons, fluorescing particulate matter, respirable suspended particles (RSP), and ultraviolet particulate matter.

In order to provide current outdoor ETS measurements, ARB conducted ambient air monitoring at outdoor smoking areas for nicotine. Different locations in California were selected to measure air concentrations of nicotine. Monitoring was conducted during 2003 at outdoor smoking areas at the following five locations: an airport, junior college campus, public building, office complex, and amusement park. At each of the study sites, sampling was conducted for nicotine over a three-day time period during typical business hours (between 8:00 a.m. and 5:00 p.m.). Two of the days were devoted to 8-hour samples; six 1-hour samples were collected on one of the sampling days. The results of the monitoring study show a wide range of exposures depending on the locations and number of cigarettes smoked. Mean 8-hour concentrations ranged from 0.013 (local government center) to 3.1 micrograms per cubic meter ($\mu g/m^3$) (amusement park). Mean 8-hour background concentrations ranged from 0.009 (junior college) to 0.12 $\mu g/m^3$ (amusement park). Mean background 1-hour concentrations ranged from less than the estimated quantitation limit or EQL (0.029 $\mu g/m^3$ for 1-hour) (junior college and local government center) to 0.17 $\mu g/m^3$ (amusement park). Overall, the results indicate that concentrations of nicotine corresponded to the number of smokers in the smoking areas, the size of the smoking area, and meteorological conditions.

Other studies have also determined outdoor air concentrations of ETS either by direct measurements or modeled outdoor air concentrations of ETS constituents. One study estimated concentrations of fine smoke particles in the Los Angeles air using tobacco-specific iso- and anteisoalkanes. Using the measurements from these marker compounds, the annual average ambient fine (less than 2.5 microns) ETS particles in the Los Angeles air was estimated to range from 0.28 to 0.36 $\mu g/m^3$. The levels were based on annual measurement data from 1982. Another study used personal badge monitors to measure personal nicotine levels. This study reported a 7-day median nicotine concentration in the outdoor environment of 0.025 $\mu g/m^3$, based on those study participants who reported no indoor exposure. Another study used a chemical mass balance receptor model based on organic compounds to estimate source contributions to fine particle mass concentrations in the Los Angeles air. The modeled annual average concentration for the Los Angeles air was estimated to be 0.21 $\mu g/m^3$ fine ETS particulate matter in 1982.

Californians who neither smoke nor associate with many smokers will have limited ETS exposure. In this case, individuals will likely experience the majority of their lifetime ETS exposure from background levels of ETS which result from occasional or steady state near-source emissions. Since most Californians live and work in urban areas, ARB staff has estimated an outdoor annual average ambient ETS particle concentration for the Los Angeles air for 2003. The staff used two Los Angeles studies as a basis for this estimate. The staff applied an adjustment factor to the 1982 fine particle estimates presented in the two Los Angeles studies to reflect reductions in cigarette sales and cigarette emission rates that have occurred since 1982. The results show that

estimated annual average fine ETS particle concentrations in Los Angeles in 2003 likely decreased to between 0.06 to 0.10 $\mu g/m^3$.

Several studies have estimated ETS levels in different indoor environments using nicotine and RSP, and other markers for ETS exposure. Current typical indoor concentrations of nicotine in California are estimated to range from near zero to about 6.0 $\mu g/m^3$ in the home environment. Because of California's workplace smoking ban, California office buildings will generally have very low smoking concentrations. However, certain workplaces, such as the small (but documented) percentage of free-standing bars that still do not comply with California's workplace smoking ban, would likely have higher levels of ETS. Based on measurements from several studies, average nicotine levels could be as high as 76.0 $\mu g/m^3$ for bars and bingo parlors where smoking still occurs.

RSP concentrations in certain entertainment venues (such as casinos and bingo parlors) are estimated to range from less than 15 $\mu g/m^3$, where smoking is prohibited, up to 350 $\mu g/m^3$, where smoking is allowed. In the home environment, short-term peak RSP levels have been found up to 300 $\mu g/m^3$, where just one cigarette was smoked. Likewise, in-vehicle ETS RSP concentrations are estimated to range from about 90 $\mu g/m^3$ to well over 1,000 $\mu g/m^3$, depending on ventilation and position of windows.

A scenario-based approach was used to characterize the range of the public's nicotine exposure to ETS in this report. The scenario-based exposure method uses the results from ARB's ETS air monitoring study, available indoor ETS concentration data, and scenario-based activity patterns to estimate exposures under different conditions. Since ETS emissions and exposure are very localized, and because only very limited data on outdoor ETS levels are available, we believe the scenario-based approach provides better and more informative estimates of public exposure to ETS. The results show a wide range of possible population subgroup daily exposures. For individuals living in non-smoking homes and having only brief encounters with ETS, their average 24-hour nicotine exposure concentrations are low, and are estimated to be less than 0.01 $\mu g/m^3$. For those living in homes with indoor smokers and experiencing in-vehicle exposures, their average nicotine exposure concentration to which they are exposed to over 24-hours can range up to 7.4 $\mu g/m^3$. Such exposures are especially of concern for developing young children because they are likely to recur daily and may result in serious health consequences.

This approach differs from previous TAC exposure assessments, which were based on California population-weighted exposures to outdoor average ambient concentrations. That approach was appropriate for TACs emitted from area-wide or region-wide sources such as motor vehicles and industrial plants. However, cigars and cigarettes, the primary source of ETS, are smaller sources that emit pollutants near people and thereby exposures to ETS are very localized.

The primary and often the only exposure for individuals that do not spend time near smokers occurs outdoors in locations over which the individual typically has little control.

For non-smokers whose work or other activities bring them into regular contact with elevated ETS concentrations during most of the day, nearly all of their exposure can be attributable to proximity to outdoor smoking.

V. HEALTH EFFECTS OF ENVIRONMENTAL TOBACCO SMOKE EXPOSURE

ETS exposure is causally associated with a number of health effects, including effects on infants and children. ETS has a number of serious impacts on children's health including sudden infant death syndrome (SIDS), induction and exacerbation of asthma, increased respiratory tract infections, and increased middle ear infections. ETS also causes developmental toxicity resulting in low birth weight, and impaired lung function growth, predisposition to SIDS (to the extent that this is a developmental effect), and other developmental impacts.

Listed in Table 5 are the developmental, respiratory, carcinogenic, and cardiovascular effects for which there is sufficient evidence of a causal relationship, including fatal outcomes such as SIDS, heart disease mortality, and lung cancer death, as well as serious chronic diseases, such as childhood asthma. There are a number of effects for which evidence is suggestive of a causal association, but further research is needed for confirmation, including spontaneous abortion, decreased lung function growth, cervical cancer, and chronic respiratory symptoms in adults. 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, adverse male reproductive effects, and rare childhood cancers.

Many Californians are exposed to ETS, and the number of people adversely affected may be correspondingly large. Table 6 presents morbidity and mortality estimates for health effects causally associated with ETS exposure. For lung cancer, where certain California-specific data are unavailable, 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 cardiovascular disease, middle ear infection, asthma episodes, SIDS, pre-term delivery, and low birth weight were derived using information on prevalence of ETS exposure in California and the U.S.

Relative risk (RR) estimates associated with some of these endpoints are small, but because the diseases are common and ETS exposure is frequent and widespread, the overall impact can be quite large. The relative risk is a measure of the relation between exposure to a substance and the incidence of a disease. A relative risk of 1.0 indicates no relationship. For ETS, a relative risk estimate of 1.2-1.7 for heart disease mortality in nonsmokers is supported by the collective evidence; this corresponds to approximately 1,700-5,500 deaths annually in California. The relative risk estimate of 1.38 associated with low birth weight implies that ETS may impact fetal growth of 1,600 newborns in California. It is estimated that at least 31,000 children in California experience one or more ETS-related asthma episodes (new onset or exacerbation) each year. Large impacts are also associated with relative risks for respiratory effects in children such as middle ear infection (RR $\approx$ 1.62) (about 50,000 children annually), and lower respiratory

infection in young children (RR ≈ 1.5 to 2) (18,000 to 36,000 children annually). ETS exposure is implicated in 21 SIDS deaths per year in California (RR ≈ 3.5). About 400 to 1,100 lung cancer deaths in California are ETS-related. For nasal sinus cancers, observed relative risks have ranged from 1.7 to 3.0. This is as high as or higher than the relative risks observed for lung cancer. Finally, for breast cancer, when evaluating younger, primarily premenopausal women at diagnosis, a pooled risk estimate of 1.68 is derived in the meta-analysis, and when restricted to the studies with better exposure assessment, an estimate of 2.20 is obtained (see Table 5). These estimates of association could represent a significant number of cases as this is a relatively common cancer in women. Adding the mid-point of the ranges for lung cancer deaths and heart disease deaths, and including the SIDS point estimate, one can attribute about 50,000 deaths per year in the U.S. and 4,000 deaths per year in California from ETS-associated disease. This does not include the estimates for other ETS-associated cancer deaths.

Table 5. Health Effects Associated with Exposure to Environmental Tobacco Smoke

Effects Causally Associated with ETS Exposure

Developmental Effects

Fetal growth: Low birth weight and decrease in birth weight
Sudden Infant Death Syndrome (SIDS)
Pre-term Delivery

Respiratory Effects

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

Carcinogenic Effects

Lung cancer
Nasal sinus cancer
Breast cancer in younger, primarily premenopausal women

Cardiovascular Effects

Heart disease mortality
Acute and chronic coronary heart disease morbidity
Altered vascular properties

Effects with Suggestive Evidence of a Causal Association with ETS Exposure

Reproductive and Developmental Effects

Spontaneous abortion, Intrauterine Growth Retardation
Adverse impact on cognition and behavior
Allergic sensitization
Decreased pulmonary function growth
Adverse effects on fertility or fecundability

Cardiovascular and Hematological Effects

Elevated risk of stroke in adults

Respiratory Effects

Exacerbation of cystic fibrosis
Chronic respiratory symptoms in adults

Carcinogenic Effects

Cervical cancer
Brain cancer and lymphomas in children
Nasopharyngeal cancer
All cancers – adult and child

Table 6. Attributable Risks Associated with ETS

	Conclusion OEHHA 1997	Conclusion OEHHA 1997	Conclusion Update	Conclusion Update
Outcome	**Annual Excess # in CA**	**Annual Excess # in US**	**Annual Excess # in CA**	**Annual Excess # in US**
Pregnancy: Low birth weight Pre-term delivery	1,200-2,200	9,700-18,600	1,600 [1] 4,700 [1]	24,500 [2] 71,900 [2]
Asthma (in children): # Episodes [3]				
# New cases			31,000 [4]	202,300 [5]
#Exacerbations	960-3120 48,000-120,000	8,000-26,000 400,000-1,000,000	N/A	N/A
Lower respiratory illness	18,000-36,000	150,000-300,000	N/A	N/A
Otitis media visits	78,600-188,700	700,000-1,600,000	50,200	790,000 [6]
SIDS	120	1,900-2,700	21 [7]	430 [8]
Cardiac death (Ischemic heart disease death)	4,200-7,440	35,000-62,000	3,600 (range: 1,700-5,500) [9]	46,000 (range: 22,700-69,600) [10]
Lung cancer death	360	3000	400 [11]	3400
Breast cancer – diagnosis in younger, primarily premenopausal women			All studies: OR 1.68 (95% CI 1.31-2.15) [12] Best studies: OR 2.20 (95% CI 1.69-2.87) Approximate 68-120% increased risk	

[1] Based on California Dept Health Services (CDHS, 2000a), Table 2-6, Number and percent of live births with selected medical characteristics by race/ethnic group of mother, California 2000, and Gilpin *et al.* (2001).

[2] Based on CDC (2002b) National Vital Statistics Report. Vol 51(2) 2002. Births: Final data for 2001, and on adult females reporting exposure to ETS in NHANES III for 1995 (Pirkle *et al.*, 1996).

[3] The data to distinguish number of new cases from number of exacerbations were not available for the updated calculations; thus, OEHHA considered that these estimates were best described as number of episodes.

[4] Based on number of asthma attacks or episodes in previous 12 months for 0-17 year olds. Calculated from California Health Interview Survey for 2001.

[5] Based on number of asthma attacks or episodes in previous 12 months for 0-14 year olds in Mannino *et al.* (2002b) CDC-MMWR 51(SS01)).

[6] Based on Freid *et al.* (1998) National Center for Health Statistics Series 13 No. 137. Ambulatory Health Care Visits by Children: Principal Diagnosis and Place of Visit for yrs 1993-1995.

[7] Based on California Dept Health Services (CDHS, 2000b), Table 4-10 for yr 2000 Leading causes of infant death by race/ethnic group of child, California 2000.

[8] Based on CDC (2002a) National Center for Health Statistics (2002). www.cdc.gov/nchs/fastats/infort.htm for yr 2000.

[9] Based on California Dept Health Services (CDHS, 2000c), Table 5-7, Deaths, death rates, and age-adjusted death rates for leading causes by sex, California, 1999- 2000.

[10] Based on Anderson and Arias (2003). National Vital Statistics Report. Vol 51(9) Table 2 for yr 2000 Ischemic heart diseases including AMI.

[11] Assuming California exposure and death rates are similar to national rates and California population is 12% of national population.

[12] OEHHA is unable at this time to calculate an attributable risk as it is not possible to account accurately for the portion attributable to other known risk factors. The OR for all studies is based on our meta-analysis of all studies with risk estimates for younger primarily premenopausal women. The OR for best studies is based on the OR for studies which evaluated younger primarily premenopausal women and which did a better job of ascertaining exposure – see Part B Section 7.4.1.3.2 and Table 7.4.1l.

N/A = data not available.

Citations for documents cited in above table appear in Part B Chapter 1 references.

A. Developmental Toxicity – Perinatal Manifestations of Prenatal ETS Exposure

ETS causes 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 birth weight, is small in magnitude. But 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. ETS is also associated with pre-term delivery. Premature babies are also at higher risk for a number of health problems.

The impact of ETS on perinatal manifestations of development other than fetal growth and pre-term delivery is less clear. The few studies examining the association between ETS and perinatal death are relatively non-informative. Studies on spontaneous abortion are suggestive of a role for ETS, but further work is needed. Although epidemiological studies suggest an 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.

B. Developmental Toxicity – Postnatal Manifestations of Pre- and/or Post-natal ETS Exposure

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, and many of these studies demonstrated a dose-response gradient.

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 on growth in otherwise healthy children. As discussed in greater detail below, developmental effects of ETS exposure on the respiratory system include childhood asthma induction and possibly adverse effects on lung growth and development.

C. Female and Male Reproductive Toxicity

Active smoking by women has been found to be associated with decreased fertility in a number of studies, and active tobacco smoking appears to be anti-estrogenic. The

epidemiological data on ETS exposure, though not conclusive, are suggestive of adverse effects on fecundability and fertility, and possibly on menstrual cycle disorders, although not many studies are available on this endpoint. 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.

D. 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 (in children and adolescents/adults) as well as for increasing the severity of disease among children and adults 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.

E. Carcinogenic Effects

The role of ETS in the etiology of cancers in nonsmokers was explored, because active smoking, has been recognized as an established cause of cancers in a number of organs including: lung, larynx, oral cavity, naso-, oro-,and hypo-pharynx, nasal cavity and sinuses, esophagus, kidney, urinary bladder and ureter, uterine cervix, pancreas,

liver, bone marrow (myeloid leukemia), and stomach (IARC, 2004). Also, ETS contains a number of constituents that have been identified as carcinogens in animals and humans.

Reviews published in the 1986 *Report of the Surgeon General* (U.S. DHHS, 1986), by the National Research Council (NRC, 1986g), and by the United States Environmental Protection Agency (U.S. EPA) (1992i), as well as the original OEHHA report (Cal/EPA, 1997) concluded that ETS exposure causes lung cancer. Since the previous OEHHA review (Cal/EPA, 1997), numerous epidemiological studies and several meta-analyses have examined the association between passive smoking and lung cancer. 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. The studies examining the effect of ETS exposure on nasal sinus cancers consistently (though not uniformly) show statistically significant associations, presenting strong evidence that ETS exposure increases the risk of nasal sinus cancers in non-smoking adults. Finally, studies suggest an association between ETS exposure and elevated risks of nasopharyngeal cancers.

Many population-based case-control studies (as well as three cohort studies), controlling for several important reproductive, dietary, and other potential confounding factors, have identified elevated breast cancer risks for residential and occupational exposure overall or in individual strata. Higher risks were noted in several studies for breast cancer diagnosed in women under age fifty (primarily premenopausal), or with long duration or high intensity exposure. The toxicological data on carcinogenicity of tobacco smoke constituents strongly support that the risk associated with ETS exposure is highly plausible. Overall, the weight of evidence (including toxicology of ETS constituents, epidemiological studies, and breast biology) is consistent with a causal association between ETS exposure and breast cancer in younger, primarily premenopausal women. In contrast to the findings in younger women, in studies which reported statistics for women diagnosed with breast cancer after menopause, risk estimates cluster around a null association (see Figure 7.4.4). There are, however, elevated risk estimates in some studies for postmenopausal women either overall or in specific strata. The evidence to date for older/postmenopausal women is, therefore, considered inconclusive. Further research indicating a positive association would be necessary prior to altering this finding.

The epidemiological and biochemical evidence suggest that exposure to ETS may increase the risk of cervical cancer. Positive associations were observed in three of four case-control studies and a statistically nonsignificant positive association was observed in the only cohort study conducted. A new population-based cross-sectional study found statistically significant elevated risks for cervical cancer. Findings of DNA adducts in the cervical epithelium as well as nicotine and cotinine in the cervical mucus of ETS-exposed nonsmokers supports biological plausibility.

In adults, the epidemiological evidence for an association between ETS exposure and risk of brain tumor remains weak and inadequately researched. More recent studies have focused on the potential association between ETS and childhood brain tumors. In children, recent studies or others not previously reviewed by OEHHA, provide no substantial evidence for an association between maternal smoking and childhood brain tumors, with risk estimates generally near the null. Several studies indicated a slightly stronger association with paternal smoking and brain cancer, although the association is still somewhat weak. Overall, the generally positive, but inconsistent, associations reported between paternal smoking and childhood brain tumors, in combination with biological plausibility, provide suggestive evidence of an association between ETS and brain cancer in children. Similarly, suggestive evidence of an association between exposure to ETS and childhood cancer is noted for lymphomas and acute lymphocytic leukemia (children of paternal smokers). These observed associations may reflect an effect of pre-conceptional paternal smoking on sperm, rather than an effect of ETS exposure.

For other cancer sites in adults, there has been limited ETS-related epidemiological research in general. The evidence to date regarding the relationship between ETS exposure and the risk of occurrence of cancer in sites other than lung, nasal cavity, breast, and possibly brain and lymphoma and leukemia, is inconclusive. 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 well designed population based study provided minimal evidence that ETS exposure may increase the risk of stomach cancer, particularly cancer of the cardia. 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 case-control studies and one cohort study conducted to date, although both studies were limited in their ability to detect an effect.

The epidemiological data are insufficient to assess potential associations between ETS exposure and rare childhood cancers. 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.

F. 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, support a conclusion that there is 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 confounders. The association

between CHD and risk is stronger for mortality than for non-fatal outcomes, including angina. It is also evident that these effects exacerbate or are exacerbated by underlying conditions, and individuals with other chronic conditions such as diabetes, vascular disease, or hypertension comprise a susceptible population at even greater risk from ETS exposure.

Data from clinical and animal 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, lesion formation, aortic distensibility and reactivity, 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. The data reviewed also suggests that the effects of ETS may also contribute to stroke, the etiology of which includes atherosclerosis of the carotid and large arteries of the brain, and degeneration of intracerebral arteries.

VI. EVALUATION OF THE NEED FOR CONTROL OF OUTDOOR ETS EMISSIONS

Following Board adoption to formally identify environmental tobacco smoke as a TAC, the Health and Safety Code section 39665(a) requires staff to prepare a report on the need of control (the "needs assessment") for ETS.

ARB regulatory authority is generally limited to outdoor exposures and most public places already have laws and/or local ordinances restricting smoking activity. However, a review of the existing laws and input from stakeholders will be important as the needs assessment is developed.

Furthermore, in its findings, the SRP recommended that ETS be added to the list of TACs that may disproportionately impact children, pursuant to Health and Safety Code section 39669.5(c). See Appendix II for the SRP findings. If ETS is added to this list, ARB is required to prepare a report on the need for ETS regulation within three years. ARB is further required to adopt within that same three-year timeframe, as appropriate, any new control measures to reduce exposure to protect public health, particularly infants and children.

VII. ENVIRONMENTAL IMPACT ASSESSMENT

The identification of ETS as a TAC is not expected to result in any adverse impact on the environment. The Board's identification of ETS as a TAC and the subsequent analysis of the need to further control outdoor emissions may result in the adoption of control measures pursuant to Health and Safety Code sections 39665 and 39666. When considering the adoption of control measures, ARB will consider all potential impacts of the measures on human health, as well as the potential benefits to public health by reducing ETS emissions. Therefore, the identification of ETS as a TAC may ultimately result in control measures that will result in environmental benefits. Adverse

environmental impacts identified with respect to specific control measures will be included in the consideration of such control measures pursuant to Health and Safety Code sections 39665 and 39666. Furthermore, ARB is committed to integrating environmental justice in all of its activities. Environmental justice is defined as the "fair treatment of people of all races, cultures, and incomes with respect to the development, adoption, implementation, and enforcement of environmental laws, regulations, and policies." Any proposed airborne toxic control measure (ATCM) for ETS must reduce health risks in all communities, including low-income and minority communities. Environmental justice will be fully assessed if, and when, an ATCM for ETS is proposed.

VIII. ECONOMIC IMPACT ASSESSMENT

The identification of ETS as a TAC will not directly have any economic impact on sources of ETS because the act of identifying a TAC does not mandate any specific risk management action. This proposed action would not require any private person or business to incur any cost in reasonable compliance with the proposed action. Once a substance is identified, ARB will assess the need and appropriate degree of control for that substance. Potential control measures will be assessed and developed in a public forum, in which the impact of these measures on businesses would be fully assessed.

IX. STAFF RECOMMENDATION AND PLAIN ENGLISH SUMMARY OF THE PROPOSED REGULATION

Based on the information available on ETS-induced non-cancer and cancer health effects and the results of the risk assessment, and the findings of the SRP, we conclude that ETS meets the definition of a TAC which is an air pollutant "which may cause or contribute to an increase in mortality and serious illness, or which may pose a present or potential hazard to human health" (Health and Safety Code section 39655). Therefore, staff recommends that the Board adopt the proposed regulation shown in Appendix I identifying ETS as a TAC with no identified level of exposure below which no health effects are anticipated.