State of California
AIR RESOURCES BOARD

APPENDIX III

PROPOSED IDENTIFICATION OF ENVIRONMENTAL TOBACCO SMOKE AS A TOXIC AIR CONTAMINANT

PART A – EXPOSURE ASSESSMENT

As Approved by the Scientific Review Panel
On June 24, 2005

The SRP approved Part A is a supporting technical document which is incorporated by reference in the Initial Statement of Reasons (Staff Report)

State of California

Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant

Part A: Exposure Assessment

As Approved by the Scientific Review Panel on June 24, 2005

California Environmental Protection Agency
Air Resources Board
Stationary Source Division
Air Quality Measures Branch

State of California
AIR RESOURCES BOARD

Technical Support Document for the "Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant"

PART A

California Environmental Protection Agency
Air Resources Board
Office of Environmental Health Hazard Assessment

This report, prepared by the staff of the Air Resources Board (ARB), contains the staff's evaluation of exposures to environmental tobacco smoke in California. This report is referred to as Part A of the technical support document, Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant. The Office of Environmental Health Hazard Assessment prepared the health assessment, or Part B, of the report.

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I.

INTRODUCTION

This report, prepared by the staff of the Air Resources Board (ARB), contains an evaluation of exposures to environmental tobacco smoke (ETS) in California. This report is referred to as Part A, "*Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant*." The Office of Environmental Health Hazard Assessment (OEHHA) has developed a comprehensive health evaluation on exposures to ETS, referred to as Part B. Together, these evaluations serve as the basis for ARB's proposal to identify ETS by regulation as a toxic air contaminant (TAC).

Under the provisions of Assembly Bill 1807 (Health and Safety Code sections 39650-39662), the ARB is mandated to administer California's TAC Program. The ARB's exposure assessment is based, to the extent available, upon research and monitoring data, emissions inventory data, and information on exposures from data on ambient and indoor air environments, as well as, an assessment of children's exposures (Health and Safety Code Sections 39650 *et seq*.). The Health and Safety Code, section 39655, also requires that each candidate TAC must meet the definition of a TAC, defined as "an air pollutant which may cause or contribute to an increase in mortality or in serious illness, or which may pose a present or potential hazard to human health."

ETS entered the identification program in June 2001. Some of the information in this report is based upon data presented in the Office of Environmental Health Hazard Assessment's (OEHHA) 1997 report: *"Health Effects of Exposure to Environmental Tobacco Smoke*" (OEHHA, 1997). Specifically, Chapter 2 (*Exposure Measurement and Prevalence*) of the OEHHA report was updated to include ETS exposure information developed subsequent to the data presented in the report (after 1995). The National Cancer Institute (NCI), acting for the U.S. Public Health Service, recognized the importance of the 1997 OEHHA report and incorporated it as part of their Smoking and Tobacco Control Monograph series (NCI, 1999).

This is the revised Scientific Review Panel (SRP) version of the report which includes the Executive Summary, Part A (exposure assessment), Part B (health effects), and Part C (responses to public comments) documents. This version of the report, along with the comments received on the public review version, will be considered by the SRP on Toxic Air Contaminants at a noticed public meeting.

The ARB's consideration of ETS as a TAC will occur following review by the SRP. If the SRP approves the report, it will be presented to the ARB at a duly noticed public hearing, after a 45-day public comment period. If the ARB approves the report at a hearing and identifies ETS as a TAC, the information contained in the report will be used in the assessment of the need for control measures. Any consideration of control

measures to reduce exposures to ETS, if identified as a TAC, will follow a separate rulemaking process, which allows for a thorough public process including workshops, and a public hearing.

REFERENCES

National Cancer Institute (NCI). (1999). *Health Effects of Exposure to Environmental Tobacco Smoke: The Report of the California Environmental Protection Agency.* Smoking and Tobacco Control Monograph No. 10, National Institutes of Health (NIH) Publication No. 99-4645. NCI, NIH, U.S. Department of Health and Human Services (USDHHS), Bethesda, MD.

Office of Environmental Health Hazard Assessment (OEHHA). (1997). *Health Effects of Exposure to Environmental Tobacco Smoke.* Final Report. California Environmental Protection Agency, Sacramento, CA.

II.

SUMMARY

This report contains the staff evaluation of environmental tobacco smoke's (ETS) physical and chemical characteristics; sources and emissions; a review of measured and modeled air concentration studies on the constituents of ETS; the results of ARB's recent ETS air monitoring study; scenario-based estimates of selected population subgroups' exposures to ETS under different smoking conditions; and the atmospheric persistence of selected ETS constituents. This report, along with the Office of Environmental Health Hazard Assessment's (OEHHA) health evaluation report (Part B), will serve as the basis for the identification of ETS as a toxic air contaminant (TAC) under the authority of California's TAC Program (Assembly Bill 1807: Health and Safety Code sections 39660-39662).

A brief summary of the information presented in the report is provided below.

Chapter III - Chemical and Physical Properties

- ETS is a complex mixture of several thousand individual gaseous and particulate compounds, many with known adverse health effects.

- ETS is produced primarily by the release of smoke from the burning tip of cigarettes and cigars between puffs (i.e., sidestream smoke) and the smoke exhaled by the smoker (i.e., mainstream smoke). Other components of ETS are the mainstream smoke emitted from the mouthpiece of cigarettes and the vapor compounds that diffuse through the wrapper.

- ETS contains several tobacco-specific nitrosamines (TSNAs). TSNA's are one of the major cancer causing agents found in tobacco smoke. N-nitrosonornicotine (NNN) and 4-(methylnitrosamino-)-1-(3,pyridyl)-1-butone (NNK) are believed to be the most potent carcinogens of this class.

- ETS particles range in size from 0.01 - 1.0 µm in sidestream smoke to 0.1 - 1.0 µm in mainstream smoke.

- Researchers have also identified at least ten polycyclic aromatic hydrocarbons (PAHs) in ETS as cancer causing toxic air contaminants. One of the most potent cancer causing PAH in ETS is benzo[a]pyrene.

Chapter IV – Production, Uses, Sources, Emissions and Smoking Trends

- Most of all tobacco is grown on the East Coast or Midwest in the United States.

- According to the most recent surveys on smoking prevalence, California sources of ETS emissions appear to originate from approximately 16% of the adult and adolescent California population.

- The California Tobacco Survey, developed by the California Department of Health Services, indicates that during the past decade, smoking prevalence among adults and adolescents has gradually decreased.

- Current smoking prevalence data was taken from the Department of Health Service's California Tobacco Survey (2002 data for adult smokers) and the California Student Tobacco Survey (2001 data for adolescent smokers) to estimate that about 16% of the California adult/adolescent population smokes.

- Since 1980, total and per capita cigarette consumption has continued to decline every year. With continuous statewide anti-smoking programs being implemented, this trend may continue.

- 2002 emission estimates of ETS from cigarettes and cigars in California and the U.S. are:

	California	U.S.
Nicotine:	40 tons/yr	647 tons/yr
Respirable Suspended Particulate:	365 tons/yr	5860 tons/yr
Carbon Monoxide:	1907 tons/yr	30,200 tons/yr

Chapter V – Exposure to Environmental Tobacco Smoke

California Activity Patterns and ETS Prevalence

- An individual's exposure is equally dependent on the air concentration of a pollutant in a given environment, and the time they spend in that environment.

- California activity pattern data suggest that a majority of a person's daily activity is spent indoors, especially at home. California adults spend about 62% of their time in their home, and children under 12 years of age spend about 76% of their time in the home, on average. Children also spend more time outdoors (10%) than adults and adolescents (6%).

- According to data from the early 1990's, on a given day, 38% of children (0 - 11 years), 56% of adults (over age 18), and 64% of adolescents (12 - 17 years) may be exposed to ETS during their daily activity.

- Recent data show that smoking prevalence continues to decline.

Monitoring ETS Constituents

- Exposure to ETS can be characterized using marker compounds that are 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 only source is tobacco smoke. Other ETS markers that have been studied include: solanesol, 3-ethenylpyridine (3-EP), carbon monoxide, iso- and anteisoalkanes (C_{29}-C_{34}), PAHs, fluorescing particulate matter, respirable suspended particles, and ultraviolet particulate matter.

- The ARB monitored nicotine concentrations at several outdoor smoking areas in California. The study gathered two 8-hour samples and six 1-hour samples per site tested. Depending on the site location and number of smokers present, the results show that the range of concentrations vary from 0.013 - 3.1 micrograms of nicotine per cubic meter of air (µg/m^3) for the 8-hour samples and 0.016 - 4.6 µg/m^3 for the 1-hour measurements. Overall, the results indicate that concentrations of nicotine correspond to the number of smokers in the smoking areas, although factors such as the size of the smoking area and wind speed affected the results.

Other ETS Ambient Air Estimation Studies

- Two Los Angeles studies estimated annual average ambient fine (2.5 microns or less) ETS particles to range from 0.21 - 0.36 µg/m^3 in 1982. Another study used personal badge monitors to measure ambient nicotine levels. This study reported a 7-day median nicotine concentration in the outdoor environment of 0.025 ug/m^3.

- The ARB has also estimated an outdoor annual average ambient ETS particle concentration for the Los Angeles air for 2003. 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 annual average fine ETS particle concentrations for Los Angeles in 2003 likely decreased to between 0.06 - 0.10 µg/m^3.

Indoor ETS Concentrations

- Current indoor concentrations of nicotine in California are estimated to range from 0.5 (low exposure) to 6.0 (high exposure) µg/m^3 in the home environment, 2 - 8 µg/m^3 in offices or public buildings where smoking is permitted, and less than 1 µg/m^3 in public buildings where smoking is prohibited.

- Certain workplaces, such as the approximately 20% of free-standing bars that are not yet compliant with California's workplace smoking ban, would likely have elevated levels of ETS based on measurements made across many studies in such locations. Concentrations in these locations could be as high as 76.0 µg/m^3 for bars and bingo parlors where smoking still occurs.

Exposure Estimates

- A scenario-based approach was used to characterize the range of the public's exposure to ETS. The scenario-based exposure method uses the results from ARB's ETS monitoring study, available indoor ETS concentration data, and scenario-based activity patterns to estimate exposures under different conditions.

- The scenario-based 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. Therefore, since exposures are localized and ETS is not monitored at ambient monitoring stations, we believe the scenario-based approach provides better and more informative estimates of public exposure to ETS.

- Current smoking practices and California regulations suggest that California children can roughly be divided into three exposure groups: children who have little or no exposure to ETS, children with smoking parents or guardians who take some measures to limit their child's exposure, and children highly exposed to ETS through smoking parents, guardians, or peer groups. Likewise, adults generally have virtually no exposure, experience regular but limited exposure in a public place, or experience substantial exposure through extensive contact with smokers.

- The results show a wide range of possible subgroup exposures. For individuals living in non-smoking homes and having only brief encounters with ETS, their average 24-hour 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 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.

- The primary, and often the only exposure for individuals that do not spend time near smokers, exposure occurs outdoors in locations over which the individual typically has little control. For non-smokers whose work or other activities bring them into contact with outdoor smokers regularly, 100% of their exposure can be attributable to proximity to outdoor smoking.

Biological Markers of ETS Exposure

- Biological markers of ETS exposure are metabolites of tobacco smoke ingredients found in physiological fluids or attached to DNA or proteins.

- Biological markers are useful in quantifying the amount of exposure to ETS. The ability to quantify exposure objectively is an important step in linking exposure to relative risk of adverse outcomes.

- Cotinine, a metabolite of nicotine, is the biological marker of choice in most epidemiological studies. Physiological fluid levels correlate very well with ETS exposure documented both by questionnaire and by personal exposure monitoring.

- Cotinine levels differ between smokers and ETS-exposed non-smokers by 2 - 3 orders of magnitude. From an epidemiological perspective, this difference is useful in that persons that misrepresent their smoking status may be excluded from study cohorts. Cotinine assays are sensitive enough that individuals without ETS exposure can be distinguished from those persons with low exposure.

- The nicotine concentration in hair is emerging as another viable biological marker of ETS exposure. In some instances, hair nicotine has been shown to better correlate with exposure than cotinine.

- The best predictor of cotinine levels, and hence exposure, in children is the number of cigarettes smoked in the home. Younger children appear to have higher exposure levels than adults. Asthmatic children may have lower clearance rates for ETS constituents than non-asthmatic children. Tobacco-specific lung carcinogens have been measured in children and correlate with ETS exposure.

Chapter VI – Atmospheric Persistence

- The combustion of cigarettes includes at least three important types of reactions, including pyrolysis, pyrosynthesis, and distillation. The result of these reactions is the production of thousands of gaseous and particle constituents. This mixture undergoes additional chemical reactions as the mix is diluted with ambient air, yielding individual compounds with their own atmospheric lifetimes.

- Gaseous chemicals that are present in ETS can react in the atmosphere with other pollutants and sunlight to form new chemical species. The ETS particles and particle-associated chemicals (those with low vapor pressure that deposit or chemically bind onto the particles) are subject to wet and dry deposition and atmospheric transformation of species adsorbed to the particles.

- Nicotine, the principal alkaloid in tobacco, is most commonly found in the gas phase in the environment. In the ambient air, nicotine may react with hydroxyl radicals to have a half-life of approximately one day.

III.

CHEMICAL AND PHYSICAL PROPERTIES OF ETS

This chapter presents the chemical and physical properties of ETS. Research shows that the combustion of tobacco products leads to the formation of thousands of particulate and gaseous constituents, each with their own physical properties. Among the various tobacco products consumed, cigarettes are the most common and therefore the main contributor to ETS (Jenkins *et al*., 2000). According to the United States Department of Agriculture (USDA), 94 percent of the tobacco leaf production in United States was used for cigarettes (USDA, 2001). The discussion below summarizes the research, which has identified the various major components of ETS. The literature cited was produced since 1972.

A. ETS AS A COMPLEX MIXTURE

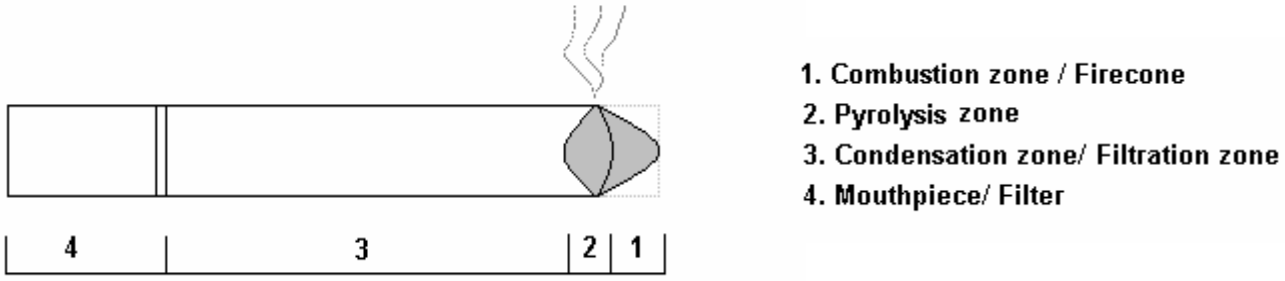
It is well established that ETS is a complex mixture of thousands of gases and particulate matter emitted by the combustion of tobacco products and from smoke exhaled by the smoker (NRC, 1986). Other minor contributors to ETS are from the smoke that escapes while the smoker inhales and some vapor-phase 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.

Of the thousands of substances that make up ETS, some are formed from combustion and some by atmospheric transformation. Appendix A includes a list of some of the compounds that have been detected in ETS.

Figure III-1 shows a cross section of a filtered cigarette, which illustrates the four zones in a burning cigarette. Cigarettes are comprised of a tobacco column (zone 3), which is housed in a paper, with a filter (zone 4) on one end. The combustion firecone (zone 1) and pyrolysis zone, where chemical decomposition occurs (zone 2), are located at the other end.

Figure III–1

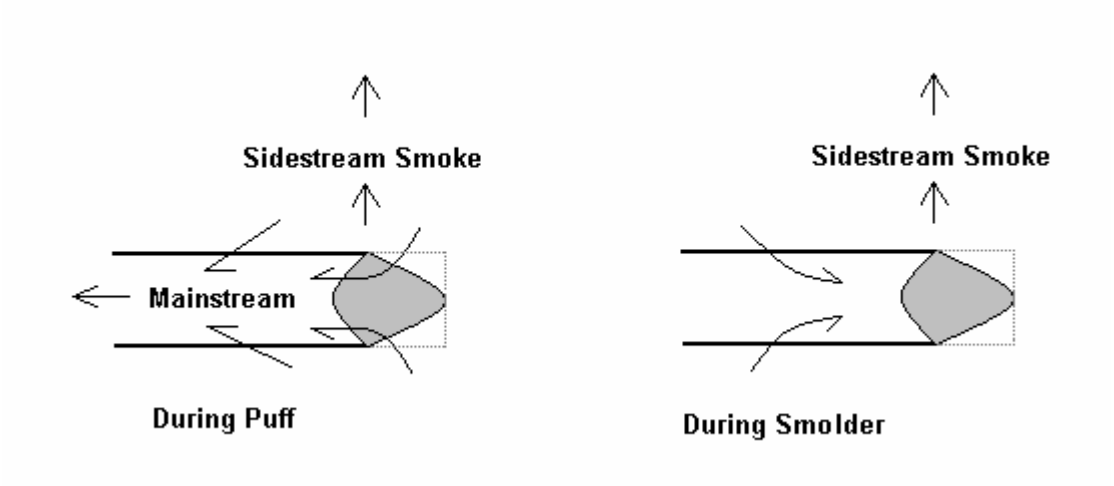
Diagram of a Filtered Cigarette



Researchers distinguish cigarette smoke as being comprised of two main components; mainstream and sidestream smoke. Figure III-2 illustrates the directions of airflow during smoking (Baker, 1980). 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.

Figure III – 2

Air Flux During Smoking



Ref: Baker, 1980

Similar chemical constituents have been found to be present in both mainstream smoke and sidestream smoke (USEPA, 1992). Differences in constituent quantities are due to variations in burning conditions, such as combustion temperature, differences in pH, and airflow rate. In general, sidestream smoke contains more ETS constituents on a per cigarette basis because more tobacco is consumed when it is smoldering between puffs, as compared to mainstream smoke.

Most tobacco crops grown in the U.S. are treated with pesticides during production. The U.S. Department of Health and Human Services has recorded the use of ethylene oxide as a common tobacco fumigant. However, pesticide residues in tobacco are likely to occur only in very low concentrations; typically as smaller, non-specific organic chemical components in ETS after decomposition during combustion (Fowles *et al.*, 2000).

1. Mainstream Smoke

Mainstream smoke is the smoke generated at the mouthpiece of a burning cigarette. More specifically, it is the exhaled smoke that was drawn in during puff and subsequently interacted with the lungs of a smoker. Modification of mainstream smoke occurs in the lungs as a result of absorption of some ETS constituents onto lung tissue, along with evaporation, particulate coagulation, and air dilution.

As a person draws in a puff from a cigarette, the airflow creates a lean burning condition with gas phase temperatures reaching 1562 ℉ (850 ℃) at the core of the firecone and solid phase temperatures reaching 1472 ℉ (800 ℃) at the firecone (Jenkins *et al*., 2000). At the firecone, core temperatures are high enough to carbonize the tobacco and thus produce an oxygen deficient combustion zone. This region of the firecone contributes to the formation of constituents produced through reductive processes (Jenkins *et al*., 2000). The gas phase and particulate matter constituents formed are cooled as the air stream passes through the tobacco column and is inhaled through the mouthpiece. The chemistry of the tobacco column changes as combustion products deposit on the remaining tobacco. The majority of ambient mainstream smoke is a result of the action of physically drawing a puff from a cigarette or cigar. However, the chemical characteristics of mainstream smoke changes as the mainstream smoke interacts in the lung, resulting in removal of some soluble organic gasses and some particulate matter.

2. Sidestream Smoke

Sidestream smoke is emitted from the burning end of a cigarette between puffs and is produced at generally lower temperatures, with a different airflow compared to mainstream smoke (Guerin *et al*., 1987). The firecone temperatures are lower for sidestream smoke at 1112 ℉ (600 ℃) (Jenkins *et al*., 2000). Because the smoldering end requires airflow, a partial vacuum is created in the tobacco column, which acts to drive the flow of air from the filter end through the firecone (Jenkins *et al*., 2000). Smoldering tobacco with lower temperatures leads to incomplete combustion, which in turn releases more quantity of compounds into the sidestream smoke as compared to mainstream smoke per cigarette (NCI, 1998).

3. Differences in the Composition of Mainstream and Sidestream Smoke

The result of the 1986 NRC report on ETS indicates that some compounds are emitted at up to more than ten times in sidestream smoke as compared to mainstream smoke (see Table III-1). Polycyclic aromatic hydrocarbons (PAHs) emissions are one example. In addition to several studies done previously measuring selected individual chemicals of PAHs in mainstream smoke, a recent study by Lodovici *et al.*, (2004) measured total PAH emissions in sidestream and mainstream smoke from different cigarettes purchased in Italy. Lodovici *et al.,* found that the PAH content in sidestream smoke is about ten fold higher compared with mainstream smoke. This study concludes that the contribution of PAHs derived from sidestream smoke is by far the most important factor in determining the PAH exposure of smokers and non-smokers.

Table III-1 also shows that ammonia emissions measured 40 - 170 times higher in sidestream smoke than in mainstream smoke. With few exceptions (e.g., hydrogen cyanide and organic acids), sidestream smoke contains greater mass emissions as compared to mainstream smoke (Jenkins *et al*., 2000; NRC 1986). Sidestream smoke is quantitatively the major contributor to ETS since more cigarette is burned in between puffs as it smolders. The available data indicate that tobacco combustion results in the emissions of a large number of known toxic compounds and that many of these will be released at rates that are higher in sidestream than in mainstream smoke. Sidestream smoke may be more toxic per unit mass as compared to mainstream smoke (U.S. EPA, 1992).

Studies indicate that sidestream smoke mass emissions are relatively constant across various cigarette types, including filter, nonfilter, full flavor or low tar cigarettes (U.S. EPA, 1992; Jenkins *et al*., 2000; Lodovici *et al.,* 2004; Leaderer and Hammond, 1991). Constituents of sidestream smoke are especially subject to phase changes because they are rapidly cooled and extensively diluted with ambient air (Jenkins *et al*., 2000). Chapter VI contains a more detailed analysis of atmospheric persistence.

Table III - 1

Distribution of Constituents in Fresh, Undiluted Mainstream Smoke (MS) and Diluted Sidestream Smoke (SS) from Nonfiltered Cigarettes

Constituents	Amount in MS per Cigarette	SS/MS Ratio
Carbon monoxide	12 - 23 mg	2.5 - 4.7
Carbon dioxide	20 - 40 mg	8 - 11
Carbonyl sulfide	18 - 42 μg	0.03 - 0.13
Benzene	12 - 48 μg	5 - 10
Toluene	100 - 200 μg	5.6 - 8.3
Formaldehyde	70 - 100 μg	0.1 - ~50
Acrolein	60 - 100 μg	8 - 15
Acetone	100 - 250 μg	2 - 5

Constituents (cont.)	Amount in MS per Cigarette	SS/MS Ratio
Pyridine	16 - 40 μg	6.5 - 20
3-Methylpyridine	12 - 36 μg	3 - 13
3-Vinylpyridine	11 - 30 μg	20 - 40
Hydrogen cyanide	400 - 500 μg	0.1 - 0.25
Hydrazine	32 ng	3
Ammonia	50 - 130 μg	40 - 170
Methylamine	11.5 - 28.7 μg	4.2 - 6.4
Dimethylamine	7.8 - 10 μg	3.7 - 5.1
Nitrogen oxides	100 - 600 μg	4 - 10
N-Nitrosodimethylamine	10 - 40 ng	20 - 100
N-Nitrosodiethylamine	ND - 25 ng	< 40
N-Nitrosopyrrolidine	6 - 30 ng	6 - 30
Formic acid	210 - 490 μg	1.4 - 1.6
Acetic acid	330 - 810 μg	1.9 - 3.6
Methyl chloride	150 - 600 μg	1.7 - 3.3
Particulate matter	15 - 40 mg	1.3 - 1.9
Nicotine	1 - 2.5 mg	2.6 - 3.3
Anatabine	2 - 20 μg	< 0.1 - 0.5
Phenol	60 - 140 μg	1.6 - 3.0
Catechol	100 - 360 μg	0.6 - 0.9
Hydroquinone	110 - 300 μg	0.7 - 0.9
Aniline	360 ng	30
2-Toluidine	160 ng	19
2-Naphthylamine	1.7 ng	30
4-Aminobiphenyl	4.6 ng	31
Benz[a]anthracene	20 - 70 ng	2 - 4
Benzo[a]pyrene	20 - 40 ng	2.5 - 3.5
Cholesterol	22 μg	0.9
γ-Butyrolactone	10 - 22 μg	3.6 - 5.0
Quinoline	0.5 - 2 μg	8 - 11
Harman	1.7 - 3.1 μg	0.7 - 1.7
N'-Nitrosonronicotine	200 - 3000 ng	0.5 - 3
NNK	100 - 1000 ng	1 - 4
N-Nitrosodiethanolamine	20 - 70 ng	1.2
Cadmium	100 ng	7.2
Nickel	20 - 80 ng	13 - 30
Zinc	60 ng	6.7
Polonium-210	0.04 - 0.1 pCi	1.0 - 4.0
Benzoic acid	14 - 28 μg	0.67 - 0.95
Lactic aid	63 - 174 μg	0.5 - 0.7
Glycolic acid	37 - 126 μg	0.6 - 0.95
Succinic acid	110 - 140 μg	0.43 - 0.62

Source: NRC (1986).

Note: A ratio greater than 1 means that more of a substance is released in SS than in MS.

B. GAS PHASE COMPONENTS IN ETS

Experimental studies have found that cigarette smoke constituents are distributed between the particle phase and gas phase. The proportion of particle to gas components depends on the environmental conditions that affect the individual chemical constituent's volatility and solubility. This proportion could also be affected by conditions at the time of the sample collection and on the approach used for sampling and analysis. According to Pritchard *et al*. (1988), about 70 percent of particulate ETS evaporates into the gas phase as smoke is diluted and aged in the air. Although it is difficult to quantify because of differences in individual breathing and smoking pattern, some amount of gas phase ETS is deposited in the lung due to diffusion of gas (Pritchard *et al*., 1988; Hiller *et al*., 1982).

Some gas phase constituents are formed during tobacco combustion and are deposited downstream of the combustion zone in the tobacco column by filtration and condensation. Those components become part of the fuel for subsequent puffs as the firecone region advances along the tobacco column (Guerin *et al*., 1987; Jenkins *et al*., 2000). These processes result in the generation of some chemical constituents found in tobacco smoke that were not originally present in the tobacco plant (Ogden and Jenkins, 1999). Table III-2 shows some of the gas phase constituents, which have been detected in ETS and have known health impacts. There are other gaseous components of ETS that exhibit health impacts not categorized in Table III-2, such as carbon monoxide and nitrogen oxides that have effect on respiratory function and further contribute to tobacco related respiratory disease.

Table III - 2

Gas Phase Components in ETS with Known Health Effects

Constituent	TAC[1/]	Prop 65[2/]	IARC Class[3/]	U.S. EPA Class[4/]	Non-Cancer Health Effects[5/]
1,3-Butadiene	Yes	Yes		B2	irritant[6/], neurological effects
Acetaldehyde	Yes	Yes	2B	B2	irritant, dermatitis
Acetone				D	irritant, dizziness
Acetonitrile	Yes			D	irritant, cause vomiting
Acrolein	Yes		3	C	irritant, pulmonary edema
Benzene	Yes	Yes	1	A	CNS[7/] depressant, nausea
Carbon monoxide		Yes			headache, dizziness
Carbonyl sulfide	Yes				irritant, CNS depressant
Ethyl benzene	Yes			D	irritant, CNS depressant
Formaldehyde	Yes	Yes	2A	B1	irritant, induce asthma
Hydrazine	Yes	Yes			hepatotoxic, dermatitis
Methanol	Yes				neurotoxicant, irritant
Methyl chloride	Yes	Yes		D	CNS depressant, fatigue
N-Nitrosodiethylamine		Yes	2A	B2	
N-Nitrosodimethylamine	Yes	Yes	2A	B2	causes liver damage
N-Nitrosopyrrolidine	Yes	Yes	2B	B2	
Pyridine		Yes			irritant, dizziness
Styrene	Yes		2B		CNS depressant, irritant
Toluene	Yes	Yes		D	CNS depressant, irritant

Sources: NRC (1986); OEHHA (1997); CARB (1997).

Notes: 1/ Substances identified as Toxic Air Contaminants by California Health and Safety Code section 39655.
2/ Chemicals listed under Proposition 65 are known to the State to cause cancer or reproductive toxicity (California Health and Safety Code section 25249.5 *et seq.*).
3/ International Agency for Research on Cancer (IARC) Classification: 1-carcinogenic to humans; 2A-probably carcinogenic to humans with sufficient animal and inadequate or no human evidence; 2B-possible carcinogenic to humans with limited animal and no human evidence; 3-not classifiable as to its carcinogenicity to humans.
4/ U.S. EPA classification: A-human carcinogen; B1-probable human carcinogen with sufficient animal and limited human evidence; B2-probable human carcinogen with sufficient animal and inadequate or no human evidence; C-possible human carcinogen; D-not classifiable as to human carcinogenicity.
5/ Non-cancer health effects information from the Toxic Air Contaminant Identification Summaries List – September 1997 (CARB, 1997)
6/ "Irritant" may be classified as an eye, respiratory, and/or skin irritant
7/ CNS – central nervous system

C. PARTICULATE MATTER COMPONENTS IN ETS

ETS particles have been measured under various conditions and techniques by many researchers in the past. The relevance of particle size and composition to toxicological and epidemiological studies has prompted researchers to devote much attention to ETS particulate matter. ETS particles have been generally found to fall in the range of particles 2.5 µm or less.

Among the various studies reviewed by staff, it was apparent that ETS particle measurement is significantly affected by the test method used. For example, Jenkins *et al*. (2000) reported a particle size distribution, collected on standard Cambridge glass fiber filters, with a particle size of 0.2 µm or larger. In comparison, NRC (1986) measured a particle size of 0.1 µm or larger. The portion of the smoke that passed through a glass fiber filter that traps particles with a diameter of 0.1 µm or larger, was designated as the gas phase. Hence, the qualitative and quantitative composition of particulate phase to gas phase may vary depending on the specific sample condition, trapping systems, and analytic methods applied to characterize the mixture of ETS constituents (NRC, 1986; Ogden and Jenkins, 1999).

In general, highly concentrated mainstream smoke has constituents preferentially distributed in the particle phase region (Jenkins *et al*., 2000). However, as the smoke ages and becomes diluted in ambient air, a large mass fraction of smoke particles evaporate to the vapor phase (Pritchard *et al*., 1988). Table III-3 lists the particulate phase components found in ETS with known health effects. Besides the information presented in Table III-3, there are other adverse health effects associated with short and long term exposure to $PM_{2.5}$ and ultrafine particles, such as asthma and other respiratory diseases.

Table III - 3

Components found in ETS Particulate Matter with Known Health Effects

Constituent	TAC[1/]	Prop 65[2/]	IARC Class[3/]	U.S. EPA Class[4/]	Non-Cancer Health Effects [8/]
2-Naphthylamine		Yes	1		irritant[9/], dizziness
2-Toluidine	Yes	Yes	2B		CNS[10/] depressant
4-Aminobiphenyl	Yes	Yes	1		hematuria, lethargy
Aniline	Yes	Yes	3	B2	methemoglobinemia
Arsenic (inorganic)	Yes	Yes	1	A	hemolysis, neuropathy
Benz[a]anthracene	Yes	Yes	2A	B2	
Benzo[a]pyrene	Yes	Yes	2A	B2	dermatitis, irritant
Cadmium	Yes	Yes	2A	B1	bronchiolitis, irritant
Catechol	Yes		2B		methemoglobinemia
Chromium VI	Yes	Yes	1	A	renal toxicity, hemolysis
Dibenzo[a,i]pyrene	Yes	Yes	2B		
Dibenzo[a,l]pyrene	Yes	Yes	2B		
Hydroquinone	Yes		3		CNS excitation, tinnitus
Lead	Yes	Yes	2B/3[5/]	B2	affects CNS, depression
N'-Nitrosonornicotine		Yes	2B		
Nickel	Yes	Yes	1	A	immune alterations, irritant
Nicotine [6/]		Yes			
N-Nitrosodiethanolamine		Yes	2B	B2	
NNK [7/]		Yes	2B		
Phenol	Yes		3	D	cardiac arrthythmias
Quinoline	Yes	Yes		B2	irritant, nausea, coma

Ref: NRC (1986); OEHHA (1997); CARB (1997).

Notes: 1/ Substances identified as Toxic Air Contaminants by California Health and Safety Code section 39655.
2/ Chemicals listed under Proposition 65, known to cause cancer or reproductive toxicity (California Health and Safety Code section 25249.5 *et seq.*).
3/ International Agency for Research on Cancer (IARC) Classification: 1-carcinogenic to humans; 2A-probably carcinogenic to humans; 2B-possible carcinogenic to humans; 3-not classifiable as to its carcinogenicity to humans.
4/ U.S. EPA classification: A-human carcinogen; B1 probable human carcinogen with sufficient animal and limited human evidence; B2-probable human carcinogen with sufficient animal and inadequate or no human evidence; C-possible human carcinogen; D-not classifiable as to human carcinogenicity.
5/ Inorganic lead – 2B; organolead - 3
6/ Also found in gaseous form.
7/ NNK: 4-(N-methyl-N-nitrosamino)-1-(3-pyridyl)-1-butanone.
8/ Non-cancer health effects information from the Toxic Air Contaminant Identification Summaries List – September 1997 (CARB, 1997)
9/ "Irritant" may be classified as an eye, respiratory, and/or skin irritant
10/ CNS – central nervous system

1. ETS Particle Size and Distribution

Virtually all ETS particulate is comprised of respirable suspended particles (RSP). Various researchers define RSP differently. For example, occupational researchers define RSP as PM_4 or less. Likewise, more conservative researchers have defined RSP as PM_{10} or PM_{15}. However, for purpose of this report, we consider most ETS particles to fall under $PM_{2.5}$, which are typically defined as particles 2.5 µm or less in diameter (NRC, 1986). RSP is also referred to as "fine" particles and can be inhaled into lungs posing health concerns (USEPA, 1992).

Of toxicological importance is the size fraction of ETS that could be deposited onto the lung. In general, particle sizes less than 0.1 µm in diameter have a high predicted deposition efficiency in the lungs (Chalupa *et al.*, 2004). Deposition efficiency of particles in the range of 0.5 µm is low because at this size, particles are too large to deposit to any great extent by diffusion and are too small to deposit by sedimentation or impaction (Hiller *et al.,*1982). Particle deposition onto the lung is greatly dependent upon size. However, other factors also play an important role such as puff frequency, volume of air inhaled and duration of the pause between inhalation and exhalation. Therefore, a longer pause in the breathing cycle between inhalation and exhalation increases the deposition of particles for all size ranges from 0.1 to 10 µm (Hiller *et al.*, 1982; Hinds, 1998).

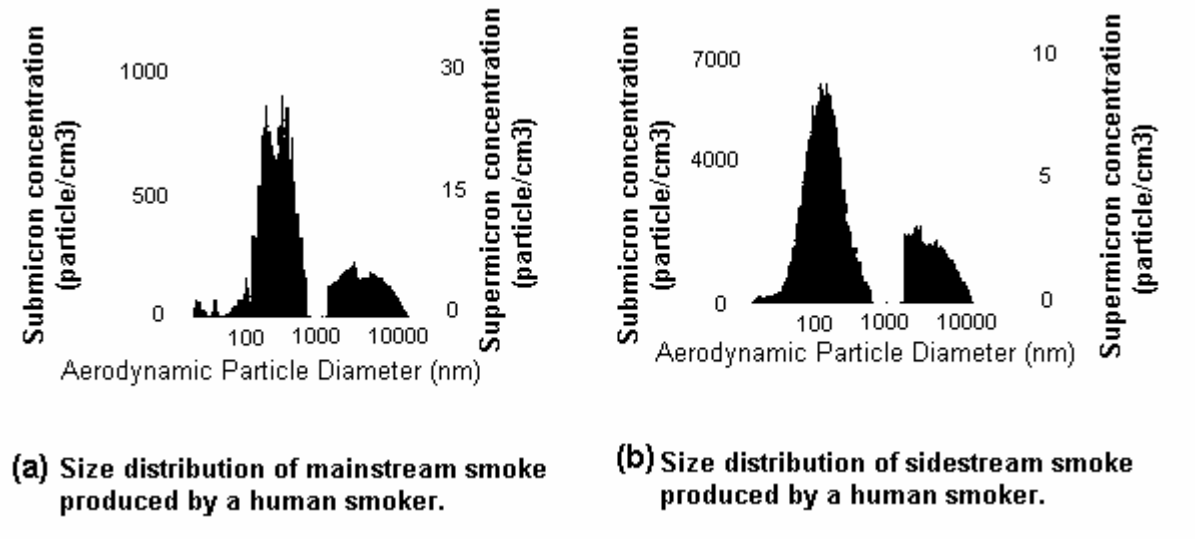
ETS particle size distribution and temporal effects have been investigated by several researchers under various controlled conditions. We found from the scientific literature that depending on the test conditions and the way ETS is generated, particle size distribution results vary. Researchers commonly report particle mass, diameter, length and particle number counts. Measured values are utilized to characterize overall ETS particle size distributions. ETS particle size distribution studies show that ETS exhibits a normal particle size distribution. Researchers typically report the mean and median peaks as measures of central tendency. The mean represents the average of particle size range, whereas the median represents the number at which half the number of particles fall above and below the value. The commonly used size distribution measurement techniques involve condensation nucleus counters, optical particle counters, aerosol electrometers and the cascade impactor.

Figure III-3 shows the distribution of ETS particle sizes in (a) mainstream and (b) sidestream smoke. Since ETS undergoes rapid chemical changes in the ambient environment, a chamber is generally used as a means to study ETS under controlled conditions. Morawska *et al*., (1997) studied the distribution of ETS particles in the diameter range of 0.01 - 30 µm. A Scanning Mobility Particle Sizer (SMPS) and an Aerodynamic Particle Sizer (APS) were used to detect submicron particle levels ranging from of 0.01 - 0.9 µm, and supermicron levels ranging from 0.5 - 30 µm, respectively. The measurements demonstrate that the distribution of ETS particles in both mainstream and sidestream smoke is bimodal. The vast majority of ETS particles were detected in the submicron range and an insignificant amount of ETS particles were in the supermicron range.

Figure III-3 shows the size distribution of ETS particles about 10 minutes after generation by a human smoker. The median diameter of the submicron peak of human-generated mainstream smoke was 0.238 µm (238 nm) with the geometric standard deviation 1.65. The median diameter of human-generated sidestream smoke was 0.136 µm (136 nm) with geometric standard deviation of 1.77.

Figure III - 3

Size Distribution of ETS Particles



(a) **Size distribution of mainstream smoke produced by a human smoker.**

(b) **Size distribution of sidestream smoke produced by a human smoker.**

Source: Morawska *et al.*, 1997
The measurements were performed independently by the SMPS (Scanning Mobility Particle Sizer) and the APS (Aerodynamic Particle Sizer) (1 µm= 1000nm).

Table III-4 lists some more notable particle measurement studies conducted by various researchers. Typically, sidestream smoke particles are in the broader size range 0.01 - 1.0 µm compared to mainstream smoke particles, which are in the range of size 0.1 - 1.0 µm (USEPA, 1992).

Table III-4

Reported ETS Particle Sizes

	Mainstream Smoke	Sidestream Smoke	Reference
Range in particle size	0.1-1.0 µm	0.01-0.8 µm	Carter and Hasegawa (1975); Hiller *et al*. (1982)
	0.1-1.0 µm	0.01-1.0 µm	U.S.EPA (1992)
Particle mean diameter [1/]	0.141µm	0.098 µm	Nelson *et al*. (1998)
	0.18 µm	0.1 µm	Guerin *et al*. (1987)
	0.41 µm	0.32 µm	Carter and Hasegawa (1975); Hiller *et al*. (1982)
Particle median diameter [2/]	0.21µm	0.185 µm	Nelson *et al*. (1998)
		0.2 µm	Ogden and Jenkins (1999)
	0.23 µm	0.14 µm	Morawska *et al*. (1997)
		0.16 µm	Ueno and Peters (1986)
		0.24 µm	Porstendorfer and Schraub (1972)
		0.52-0.67 µm	McCusker et al. (1980)
	0.235 µm		Chang et al. (1985)
	0.44-0.43 µm		McCusker et al. (1982)

Source: Morawska et al. (1997).

[1/] Mean diameter: average diameter of all particle spectrum. (µm = 10^{-6} meter)
[2/] Median diameter : equal number of particles counted in terms of diameter above and below this size.

Studies consistently show that sidestream smoke is comprised of smaller size particles as compared to mainstream smoke under the same test conditions (Ueno and Peters, 1986; Guerin *et al*., 1987; Carter and Hasegawa, 1975; Hiller *et al*., 1982; Jenkins *et al*., 2000).

a. Aging Process of ETS

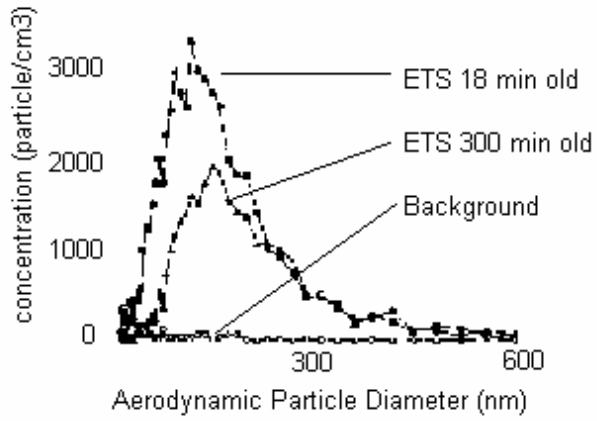
ETS undergoes a very dynamic aging process with several reactions observed such as coagulation, hygroscopic growth, evaporation, and condensation, among others.

Temporal Effect

The lifetime of ETS in ambient air depends mainly on dilution rates and environmental conditions. Yet, for indoor environments, ETS can be detected in the contained indoor environment long after it is first generated. Morawska *et al.* (1997) demonstrated the temporal effect on ETS particle size and concentration over time. As shown in Figure III-4, ETS concentrations are still well above background levels 300 minutes after the initial ETS generation. While particle concentration decreases, the particle mean and median diameter increased slightly. In chamber studies, decreases in ETS particle concentrations over time were mainly due to ventilation, wall deposition, coagulation, and evaporation of ETS particles (Morawska *et al*., 1997; Pritchard *et al*., 1988).

Figure III - 4

ETS Particle Concentration over Time



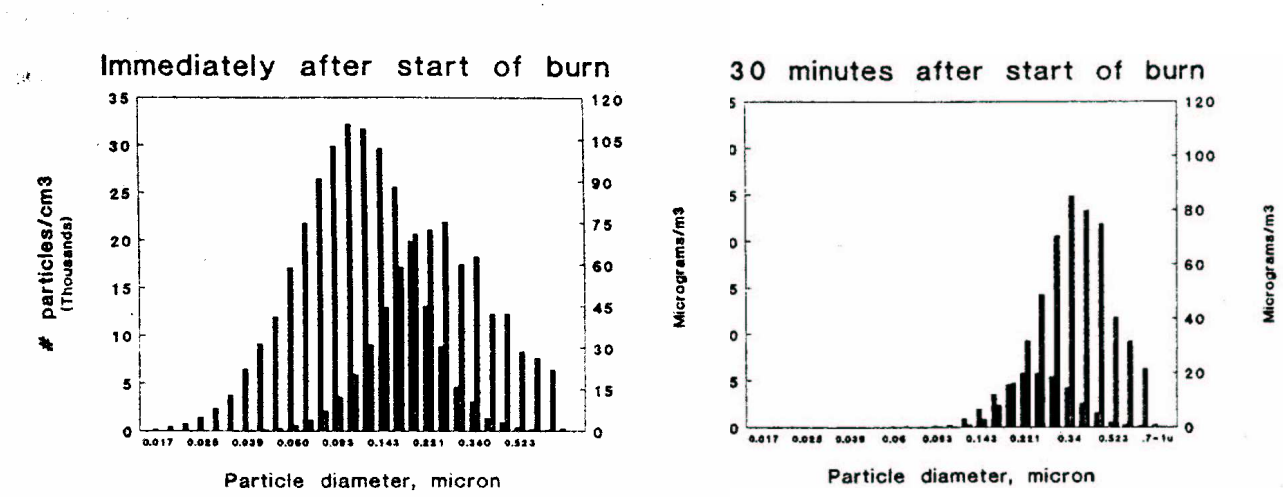
ETS size distribution at the beginning and at the end of the measurement period.

Source: Morawska *et al*. (1997).

Benner *et al*. (1989) also confirmed the prolonged-existence of ETS particles in the environment. ETS was generated in a 30-m^3 Teflon® chamber and observed for four-hours. Observed results show that the number median diameter increased from 0.11 to 0.22 µm over the four-hour experimental period while the mass-median diameter increased from 0.26 to 0.34 µm. As shown in Figure III-5, the particle distributions remain normal over time while total concentration decrease. It is difficult to measure ETS removal rates in outdoor settings since outdoor conditions are highly variable and change rapidly.

Figure III - 5

ETS Particle Distribution Temporal Effect (0.015 - 0.75 µm size range)



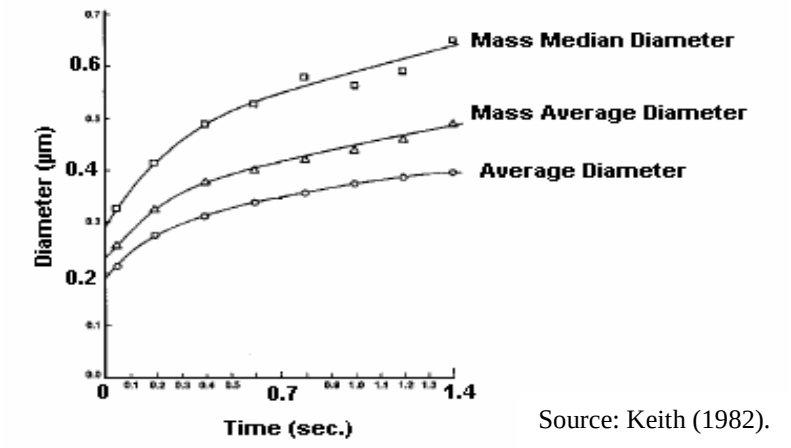
Source: Benner *et al*. (1989).

Coagulation

Coagulation of particles occurs when small particles collide into each other to form larger particles. Keith (1982) reported a doubling of particle diameter when undiluted smoke ages for 1.4 seconds (Figure III-6), consistent with coagulation theory. Coagulation mainly occurs in the lung during an active puff, as well as in indoor settings, where ambient ETS concentrations are elevated. For cigarette smoke under highly concentrated conditions (e.g., 10^9 particles per ml), coagulation of 0.1 - 1.0 µm diameter particles can occur in a fraction of a second (Keith 1982).

Fig III-6

Effect of Aging on Particle Size - Coagulation



Source: Keith (1982).

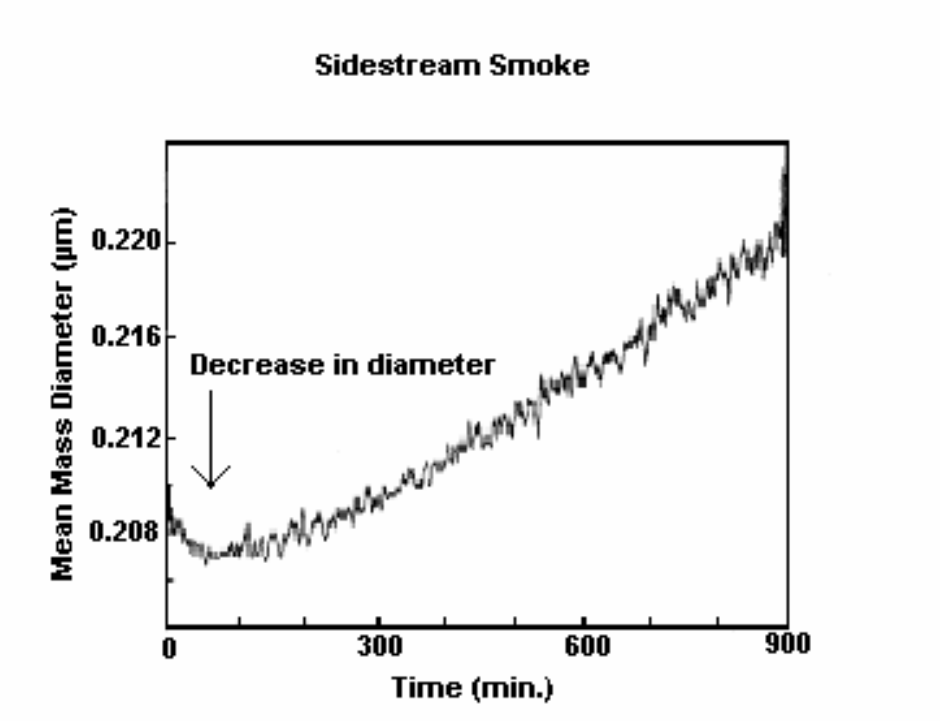
Hygroscopic Growth and Evaporation

In test chambers, Morawska *et al.* (1997) examined the effects of relative humidity on ETS particle growth. At high humidity (95 percent), total particle growth of up to 175 percent was observed, and postulated to result from hygroscopic growth (i.e., the hydration of dry particles). Studies have found that under high humidity, hygroscopic particles (such as those in ETS) can increase to the size of haze particles due to hydration (Seinfeld and Pandis, 1998). Coagulation and hygroscopic growth results in fine particle loss and faster settling of larger particles in the environment. Subsequently, the overall particle size distribution of ETS can be affected.

By contrast, a decrease in particle size has also been reported in other studies due to evaporation (Chang *et al.*, 1985; Ingebrethsen and Sears, 1989). In the work by Ingebrethsen and Sears (1989), sidestream smoke was diluted into a 0.45 m^3 stainless-steel tank under controlled conditions of smoke concentration, air exchange and mixing rate. In the first 75-minutes, there was a strong indication of particle removal by evaporation. Figure III-7 shows the initial decrease in mass-mean diameter, indicating that evaporation had taken place. Elimination of smaller particles by evaporation and higher surface removal efficiencies may explain the increase in average diameter over time.

Figure III - 7

Evaporation of Particles in Sidestream Tobacco Smoke



Source: Ingebrethsen and Sears (1989).

Dilution

Of the physical reactions that occur to ETS, the most important is dilution. Certainly for ETS generated outdoors, dilution plays an important role in determining the actual ETS concentrations to which the public is exposed. As seen by our own testing, even modest winds reduced our measured nicotine concentrations (See Chapter V).

In a study by Chang *et al.* (1985), decreases in particle size were reported when machine-generated mainstream smoke was diluted. Machine-generated mainstream smoke was immediately diluted with laboratory air (humidity 45 - 75 percent) at dilution ratios of 6, 10, and 18 and introduced into the Cascade Impactor at 12.6, 5.1 and 4.4 seconds, respectively. The same dilution ratios were introduced to the Electrical Aerosol Size Analyzer (EAA) and Condensation Nuclei Counter (CNC) analyzers, at 23.3, 12.3, and 8.4 seconds. Table III-5 shows the decreases in particle size and number count resulting from the dilution of mainstream smoke with air. As ETS ages and mixes in air, water, volatile and semi-volatile components evaporate from the particles. Evaporation results in decreases in average particle size, and can shift overall particle size distribution curve and particle concentrations.

Table III - 5

Effects of Primary Dilution Ratio on the Number Concentration and Particle Size Distribution of Mainstream Cigarette Smoke

	Case 6	Case10	Case18
Primary Dilution Ratio	6	10	18
Mean Diameter (µm)	0.302	0.259	0.262
Standard Deviation of the Mean	1.27	1.18	1.26
Mean number conc. (particle/cm^3) [a]	4.2×10^9	3.6×10^9	7×10^8
Mean number conc. (particle/cm^3) [b]	2.4×10^9	2.1×10^9	4×10^8

[a] Results from the Electrical Aerosol Size Analyzer (EAA).
[b] Results from the Anderson Cascade Impactor.

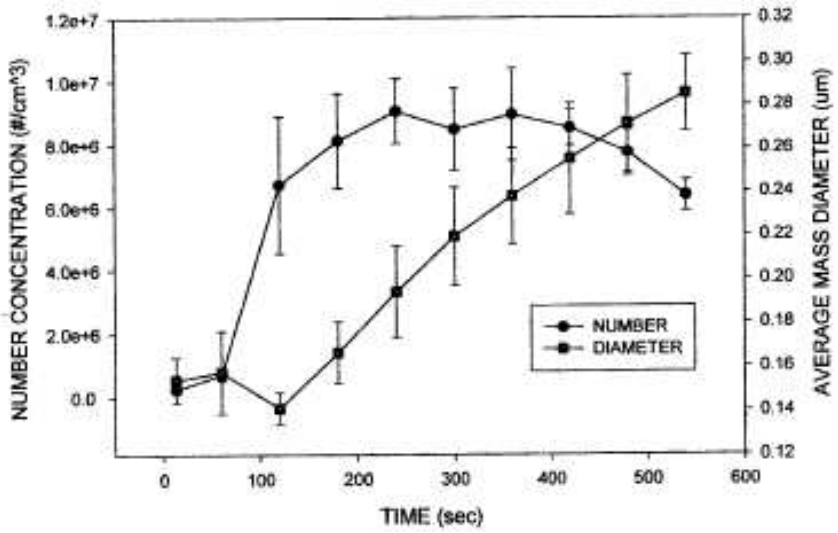
Particle Formation

Besides particle growth and shrinkage, particle formation and generation also affect ETS particle size distribution. Aerosol particle formation and growth has been observed in aging mainstream smoke from initially particle-free smoke vapor (Ingebrethsen and Lyman, 2002). In Ingebrethsen and Lyman (2002), 50 ml of particle-free filtered smoke was drawn from a cigarette attached to a filter holder. Particle formation and growth were measured using a light-scattering detection method as smoke aged over 500 seconds. Figure III-8 presents particle concentration and average particle size measurements over time on a log scale. The optical particle counter (OPC) detected particle formation and growth in average mass diameter in the first 500-seconds, most

likely due to condensation and coagulation. Beyond 500-seconds, particle number concentration began to decline from peak levels, but average mass diameter continued to increase. These results provide evidence that some fraction of filtered, particle-free mainstream smoke does not remain in the gas-phase for long before undergoing varying degrees of particle formation.

Figure III – 8

Particle Formation in Cigarette Smoke Gases



Source: Ingebrethsen and Lyman (2002).
Error bars are ± one standard deviation.

Particles in the diameter range of 0.005 - 0.05 µm can be formed by condensation of hot vapor during the combustion process and by droplet formation of atmospheric species, and contain most of the toxic compounds in ETS. In comparison, particles in the diameter range of 0.05 - 2 µm are among the most stable, and are formed by gas-to-particle conversion, chemical reaction, condensation and coagulation (Hinds, 1998).

In conclusion, the particle-size composition of ETS changes dynamically. Changes result from growth and shrinkage of particles by coagulation, hygroscopic growth, evaporation, condensation and formation among others. From a toxicological perspective, it should be noted that even after ETS undergoes complex reactions, the majority of ETS particles are still in the fine particulate range between 0.1 and 1.0 µm diameter.

D. SEMI-VOLATILE COMPONENTS IN ETS - NICOTINE

In addition to gas and particle phases, ETS also has constituents that are detected as in both phases to the degree determined by their volatility and the environmental conditions. These compounds are referred to as being "semi-volatile," and include substances such as nicotine, 3-ethenylpyridine, alkanes, and selected PAHs and PCBs.

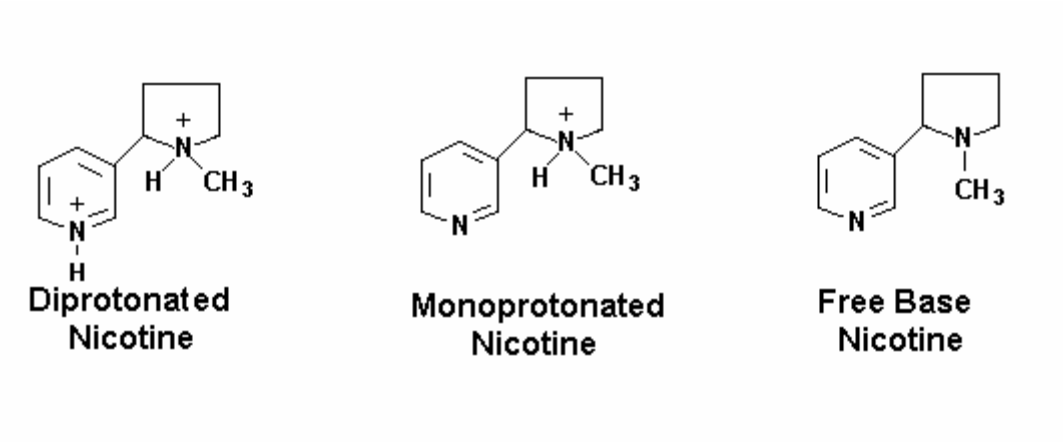
Semi-volatile compounds with lower vapor pressure may be adsorbed to the surrounding surfaces and may reenter the gas phase through desorption (Van Loy *et al*., 2001). This dynamic behavior of semi-volatile compounds prolongs its availability in the environment, particularly in the indoor environment. Therefore, one may be exposed to semi-volatile constituents, such as nicotine, for a longer period after the active smoking has ceased.

Of the various semi-volatile components in ETS, nicotine deserves some discussion because of its use as a marker in the ARB's monitoring study and because of its use as a surrogate for exposure (See Chapter V, Section E). As mentioned earlier, nicotine exists mainly in the particle phase in mainstream smoke, but exists primarily in the gas phase in sidestream smoke (Jenkins *et al*., 2000; Van Loy *et al*., 2001). Nicotine is one of the most commonly used indicators to detect ETS in the environment because it is unique to tobacco smoke (Ogden and Jenkins, 1999).

To enhance the generation of gas phase nicotine during smoking, ammonia-forming compounds are sometimes added to the tobacco. The presence of ammonia promotes nicotine existence in the gas phase rather than adsorbed to particles (Pankow *et al*., 1997). Figure III-9 shows three forms of nicotine -- mono, diprotonated, and free-base nicotine. The diprotonated and monoprotonated forms of nicotine do not exist in the gas phase and reside essentially in the particle phase. In contrast, free-base nicotine can exist in both the particle and gas phases. Unlike the protonated forms of nicotine, the free-base nicotine particles can be converted to the gas phase, and readily absorbed into the lung and into the blood stream.

Figure III-9

Three Forms of Nicotine



The semi-volatile constituents of ETS exhibit different dynamic behaviors depending on temperature, dilution and other environmental conditions. For example, some components of fine particles and volatile aerosols also may exhibit semi-volatile behavior under controlled conditions. As the volatile aerosols on the outer layer of a particle evaporate, either partially or entirely (Kunh *et al.,* 2004), particle diameters may decrease until the non-volatile core is reached.

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IV.

PRODUCTION, USES, SOURCES, EMISSIONS, AND SMOKING TRENDS

In this chapter, we discuss tobacco production, sources of ETS emissions, adult and adolescent smoking prevalences, which was determined through the California Department of Health Services surveys during the 1990s, estimated ETS emissions in California, and smoking trends. ETS emission estimations were determined through cigarette sales in California, smoking prevalence, and emission factors for nicotine, respirable suspended particulates, and carbon monoxide. Literature published between 1992 and 2003 was used to develop this chapter.

A. PRODUCTION

Although no tobacco production occurs in California, there is a significant amount of use by the public. In 2002, over 25.4 billion cigarettes were consumed in California (CBOE, 2003). In 2002, the estimated consumption of large and small cigars in California was 247 million and 135 million, respectively (USDA, 2003b).

Tobacco is grown in 21 other states, but over 65% of United States production comes from North Carolina and Kentucky (USDA, 2001*)*. Cigarettes produced for North America are predominantly produced from various varieties of tobacco plants, including Virginia bright, burley, Maryland and Turkish. Tobacco product manufacturers employ various drying methods that yield different tobacco products ranging from light to dark; each with its unique flavor (Hoffman and Hoffman, 1997). Typically, brands employ blends of the various tobaccos.

Tobacco acreage declined about 3% during 2003 and tobacco production is at its lowest since 1908 (USDA, 2003a*)*. 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 (USDA, 2003a). Tobacco can be used for cigarettes, cigars, chewing, snuff, and pipes, although cigarettes and cigars account for approximately 95% of the tobacco products produced in the United States. Cigarettes comprise 85% of tobacco products and is the main contributor to ETS (USDA, 2001).

B. USES

Staff is not aware of any industrial or commercial use of ETS. Some ETS has been used for research purposes.

C. SOURCES OF EMISSIONS

1. ETS "Point Source"

The level of ETS emissions depends in large part on the smoking public's behavior. However, at the source of ETS emissions are the combustion of individual tobacco products. The tobacco industry categorizes cigarettes and cigars according to the amount of tar and the mass of tobacco used.

Cigarette manufacturers use a number of descriptive terms in cigarette advertising, such as "light," "extra light," "medium," "mild" and "ultra light." In reality, these terms are brand descriptors (Philip Morris USA, 2003). These descriptors should not be assumed to indicate any determined amount of tar or nicotine in the cigarette.

The Federal Trade Commission (FTC) has two cigarette definitions used for advertising purposes, which is based on the amount of tar from cigarette smoke drawn in by a standardized machine and not total tar in a cigarette. The first category is "low tar", which describes machine-measured tar yields in cigarettes having a tar content of 7 - 15 milligrams (mg). The second category is "ultra low tar," which indicates the machine-measured tar amount of a cigarette to be 6 mg or less (FTC, 1997). However, these descriptors do not correspond to the actual tar and nicotine levels a smoker would inhale. Studies have revealed that light and regular cigarettes can deliver the same tar and nicotine levels (Burns and Benowitz, 2001). In 1998, nearly 82% of all cigarettes sold had a tar value of 15 mg or less (FTC, 2000).

To evaluate the effects of cigarettes on mainstream emissions, Djordjevic *et al*. (2000), compared carbon monoxide (CO) emissions from two cigarettes advertised as having either a nicotine content of 0.6 - 0.8 mg, or 0.9 - 1.2 mg per cigarette. Table IV-1 compares the yields of nicotine, tar and carbon monoxide for cigarettes tested under the FTC standard machine smoking procedure, compared to the emissions generated by an

Table IV-1

Comparison of FTC and Actual Cigarette CO Emissions

	FTC Machine		*Actual Smoker*	
	0.6-0.8 mg Nicotine	0.9-1.2 mg Nicotine	0.6-0.8 mg Nicotine	0.9-1.2 mg Nicotine
Nicotine (mg/cig.)	0.7	1.11	1.74	2.39
Tar (mg/cig.) a/	8.5	15.4	22.3	29.0
CO (mg/cig.)	**9.7**	**14.6**	**17.3**	**22.5**
Puff:				
Volume (ml)	35.0	35.0	48.6	44.1
Interval (sec)	58.0	58.0	21.3	18.5
Duration (sec)	2.0	2.0	1.5	1.5

Source: Djordjevic *et al.*, 2000

a/ Total tar particulate matter minus water and nicotine

actual smoker. The results indicated that, for the 0.6 - 0.8 mg cigarettes, smokers inhaled 1.74 mg of nicotine while the FTC machine only measured 0.7 mg of nicotine per cigarette. Similarly, smokers inhaled 22.3 mg of tar while the FTC machine measured 8.5 mg of tar. The National Cancer Institute Monograph 13 concluded that measurements of tar and nicotine yields using the FTC method do not offer smokers meaningful information on the amount of tar and nicotine that they will receive from smoking low tar and low nicotine cigarettes (Kozlowski *et al.*, 2001). As shown in Table IV-1, actual smoker mainstream smoke concentrations are greater than those levels generated by the FTC machine methodology.

Sidestream smoke is primarily related to the weight of the tobacco and paper consumed during smoldering periods (USEPA, 1992). A number of studies indicate that sidestream smoke emissions show little variability among different types of cigarettes, such as full flavor or low tar (USEPA, 1992; Jenkins *et al*., 2000; Leaderer and Hammond, 1991). Consequently, studies do not show sizeable decreases in total ETS emissions due to the marketing of low tar and low nicotine cigarettes. When comparing tar and nicotine content in cigarettes sold in the United States, the measured yields tend to be 10 - 20 times more tar than nicotine (FTC, 2000).

The FTC separates cigars into three weight categories based on the mass of 1,000 cigars. The FTC designation of “little” cigars are those that weigh less than three pounds per 1,000 cigars, while “medium” cigars weigh three to ten pounds per 1,000 cigars. FTC’s designation for “large” cigars includes the weight category of ten or more pounds per 1,000 cigars.

In 1997, the domestic market share among small, medium and large cigars was 26.6%, 35.3%, and 38.2%, respectively (FTC, 1999). Although cigar consumption is regularly reported as large cigars, consumption for small cigars can be estimated by domestic invoices (USDA, 2003b). In 1997, over 5.1 billion cigars were consumed nationwide, whereas, in 2002, cigar consumption increased by over 20% to 6.3 billion cigars (USDA, 2003b).

In a study by Repace (2001), large cigars were found to produce greater total emissions compared to cigarettes and contained most of the same toxic and carcinogenic constituents found in cigarette smoke. Emissions from one cigar have been shown to exceed those of three cigarettes, which are simultaneously consumed, and can contain up to 70 times as much nicotine as individual cigarettes (Henningfield *et al*., 1996). However, because cigars comprise such a small percentage of tobacco products consumed, cigarette consumption accounts by far for most of the ETS emissions.

2. Smoking Prevalence in California

While consumption of individual tobacco products is the origin of ETS, it is the smoking public that dictates the nature and quantity of ETS emissions the public is exposed to in the environment. To understand the segments of the population, which contribute most to ETS emissions, staff evaluated data on smoking prevalence. Simply put, prevalence measures a practice regarding whether it is widespread or universally accepted. 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 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 Assembly Bills which reauthorized provisions of Proposition 99. The CDHS conducted surveys in 1990, 1992, 1993, 1996, 1999, 2001, and 2002 (CDHS, 2003a, b). For these surveys, the CDHS contracted with the Cancer Control and Prevention Division at the University of California, San Diego and WestEd, Inc. The surveys are used as the basis for tracking the progress of the smoking cessation evaluation effort. To ensure the most accurate smoking prevalence estimates, survey methodologies occasionally alter questions or approaches over time.

The CDHS gathered important information about smoking behavior through the California Tobacco Surveys (CTS). These surveys are designed to obtain representative statewide data on the percent of the smoking population, attitudes towards smoking, perceptions regarding media coverage and use of tobacco products other than cigarettes. The CTS are random-participation telephone surveys targeting various groups, including adolescents (12 - 17 years) and adults (18+ years) (Gilpin *et al.*, 2001). Over 91,000 households were contacted among the past six CTS studies.

Another survey funded by CDHS is the California Student Tobacco Survey (CSTS). This survey is a large-scale, in-school student survey of tobacco use which collects data from both middle (grades 6 - 8) and high school (grades 9 - 12) students. This adolescent survey is considered a more accurate survey since students respond directly to solicitors and are not inhibited by the presence of their parents. The first CSTS data were weighted relative to the 2001 population of California in-school youth, by gender, grade level, and race/ethnicity. However, for the first CSTS, only high school data was available due to an insufficient sample size for middle school students.

As shown in Figure IV-1, during the past decade smoking prevalence among adults and adolescents has gradually decreased (Gilpin *et al.*, 2001). The adult smoking prevalence shown in Figure IV-1 is based on total daily smokers (smokers who now smoke everyday) and occasional smokers (smokers who now smoke some days). Beginning with the 1996 CTS, a new survey question was added to update adult smoking prevalence by capturing more “occasional” smokers. The 1996 CTS used both the “old” and “new” smoking question, which resulted in two different estimates of adult

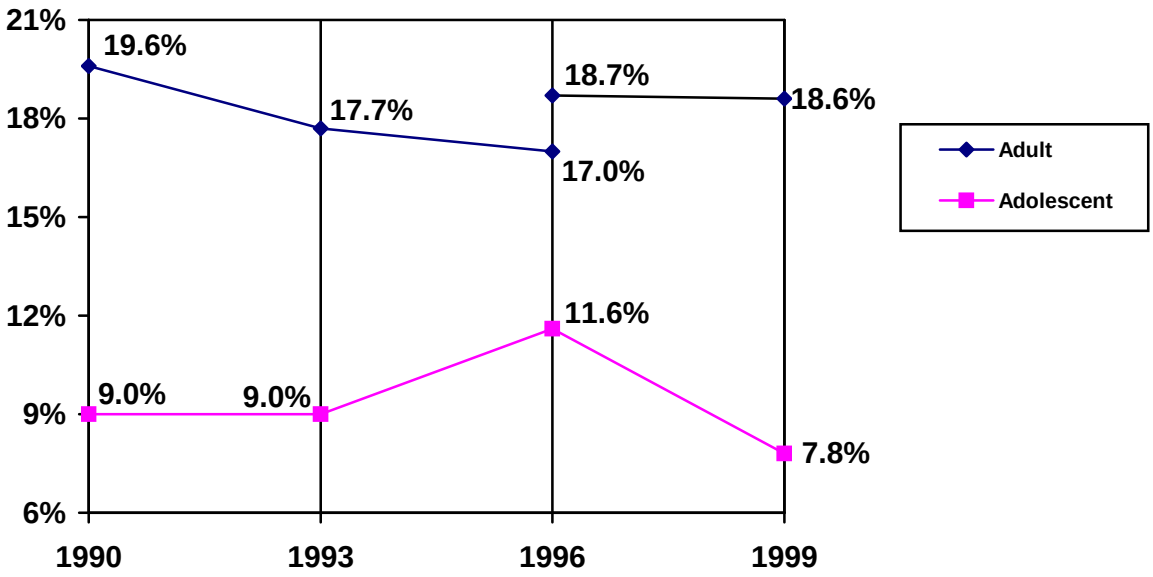
smoking prevalence. Adolescent smoking prevalence is based on criteria of any smoking within the last 30 days.

Although adolescent prevalence in California increased between 1993 and 1996, the overall smoking prevalence has decreased since 1990. In addition to overall reductions in daily adult smoker prevalence, the number of cigarettes that adults consume also appears to be decreasing as well. Heavy daily smokers (15 or more cigarettes per day) have declined considerably, while converting to occasional smoking (less than 15 cigarettes per day) (Gilpin *et al.*, 2001).

Smoking patterns among current California adult smokers have changed over time. Since the passage of Proposition 99 in 1988, the annual adult per capita cigarette consumption in California has declined by over 60%, from 126.6 packs in 1988 to 50.6 packs in 2001 (CDHS, 2003b). Adult smoking prevalence in California has decreased at a faster rate relative to the rest of the nation. However, the 18 - 24 age group has shown signs of a much smaller overall decrease. Adult male and females have remained fairly consistent in smoking prevalence rate. Non-Hispanic whites (Caucasian) show the greatest smoking prevalence, while Asians and Hispanics have the lowest smoking prevalence. African-Americans have shown the greatest decline of smoking prevalence since 1990.

Figure IV-1

[1]Adult and [2]Adolescent Smoking Prevalence in California (1990-1999)



[1] Smoking prevalence based on daily and occasional smokers
[2] Smoking prevalence based on any smoking within the last 30 days
Source: Gilpin *et al.*, 2001. The California Tobacco Control Program: A Decade of Progress, Results from the California Tobacco Survey, 1990-1999.

Table IV-2 shows the overall smoking prevalence from the current adult and adolescent surveys. In contrast to adult females, males have a higher smoking prevalence. In particular, young males between 18 - 24 years of age show no indication of reduced smoking prevalence.

From the 2001 adolescent CSTS results, adolescents that are in 9th grade showed a significantly smaller smoking prevalence than the students in 12th grade. Differences in gender smoking prevalence vary more so for adults as compared to adolescents. Adult and adolescent non-Hispanic whites are among the higher prevalence throughout the major ethnic demographic groups within California based on the new surveys.

Table IV-2

Current [1]Adult and [2]Adolescent Prevalence (%)

	Adult (%)	Adolescent (%)
Overall	16.2	16.0
Gender		
Male	19.5	16.2
Female	13.0	15.7
Age		
Grade 9		10.4
Grade 10		14.8
Grade 11		17.6
Grade 12		22.9
18-24	18.0	
25-44	18.1	
45-64	16.4	
65+	7.6	
Race/Ethnicity		
African-American	19.0	8.2
Asian/PI	12.1	13.6
Hispanic	13.4	14.0
Non Hispanic White	17.3	19.9

Source: CDHS, 2003b. The California Tobacco Control Program
[1] Adult results from the 2002 California Tobacco Survey,
[2] Adolescent results from the 2001 California Student Tobacco Survey

D. ETS EMISSIONS

As mentioned in Chapter III, ETS is a mixture containing thousands of different compounds. To estimate the total amount of ETS emissions within the State, one would have to add the amounts of all individual compounds emitted from tobacco products.

However, this is not practical since it requires the development of analytical methods to detect and measure several ETS compounds, at a very significant cost.

Therefore, to simplify the emission estimation, staff characterized ETS emissions as nicotine, respirable suspended particulate (RSP), and carbon monoxide (CO). In general, the estimate of cigarette ETS emissions was based on the following equation:

Emissions (tons/yr)= EF x N x 90% x CF;

where: EF = Average cigarette emission factor (mg/cig)
N = Number of cigarettes per year (cig/yr)
CF = Units conversion factor (tons/mg)

For purposes of this estimate, we assumed a uniform consumption rate among the population. A 90 percent adjustment factor was also applied to account for the remaining "butt" which smokers typically discard (Hildemann *et al.,* 1991). Depending on the factor used for N, number of cigarettes per year, emissions can be estimated for different geographic regions and demographic groups.

Apportioning ETS emissions as either outdoor or indoor emissions is difficult to determine due to limited information. However, other associated data can be viewed to give some insights. Outdoor ETS emissions would include direct emissions from outdoor smoking, plus ETS emissions generated indoors which eventually ventilate outside. Given the enactment of Assembly Bill 13 (AB 13) in 1998, all workplaces (including bars and restaurants) are now smoke-free in California. There are likely some workplaces that don't comply with AB 13, but we expect that a vast majority of workplaces are smoke-free. In addition, smoking behavior has changed as well. Based on the 2002 California Adult Tobacco Survey (CATS), over 80% of all California homes with children are now smoke-free. Of California smokers, 50% have reported smoking bans in their homes. Therefore, with no indoor smoking in workplaces, other public venues, and half of California smoker residences having indoor smoking bans, 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 (Rogge *et al.*, 1994). From all of the available information, the 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. Appendix B presents the calculation methodology for estimating outdoor ETS emissions.

1. ETS Emissions by Region

In the previous section regarding sources of ETS, we identified which California demographic groups contribute to ETS emissions. However, to estimate the quantity of ETS emissions, a straightforward calculation was employed that utilizes the most recent information on demographics, emission rates and cigarette consumption. For a detailed description of the emissions estimation methodology that we used, refer to Appendix B of this report.

To estimate ETS emissions, we used specific data sets including: the 2002 CDHS survey (adult prevalence), the 2001 CSTS (adolescent survey), the 2002 U.S. Census Bureau (population) and the Board of Equalization (CBOE) 2001-02 cigarette distributions in California (i.e., cigarettes consumed). We also reviewed several studies to determine representative emission factors.

Table IV-3 shows staff's estimated total statewide ETS emissions for nicotine, RSP, and CO from cigarettes and cigars. These emissions were derived from smoker population and smoking prevalence data within the different regions throughout the state. Smoking behavior was assumed to be uniform among the various demographic groups.

Estimates for CO and RSP indicate very low levels relative to total emissions. ETS emissions of CO represent less than one percent of total statewide emissions. Our RSP estimate is based on studies predominantly measuring ETS particulate less than PM_4. On this basis, ETS derived RSP contributes less than one percent to total statewide PM_{10} emissions. By comparison, diesel exhaust particulate also contributes less than one percent of total statewide PM_{10} emissions. Currently, ARB does not have an emissions inventory for nicotine. However, the estimated ETS nicotine emissions are expected to represent most of the statewide inventory, in addition to two pounds of reported pesticide use by the Department of Pesticide Regulation. While emissions may seem to be low, high exposures can result due to the generally close proximity of non-smokers to smokers (see Chapter V).

Table IV-3

2002 California Statewide ETS Emissions (Tons/Year)

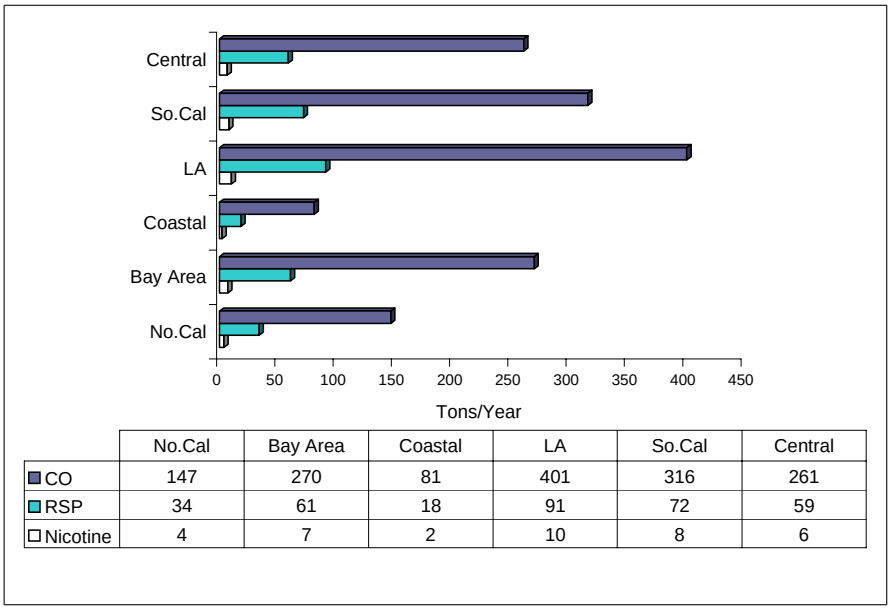
	Cigarettes	Cigars	[a]Total
Nicotine	36	4	40
RSP	335	30	365
CO	1475	432	1907

[a] Staff estimates 80-90% of total emissions reside outdoors

Figure IV-2 shows staff's calculated ETS emissions from cigarettes for various regions within the State. Appendix B (Attachment A) of this report presents the calculation methodology and estimated emissions by region within California. As expected, the highest ETS emissions correspond to areas of the highest population and population density.

Figure IV-2

Regional ETS Emissions From Cigarettes



	No.Cal	Bay Area	Coastal	LA	So.Cal	Central
CO	147	270	81	401	316	261
RSP	34	61	18	91	72	59
Nicotine	4	7	2	10	8	6

2. Comparing California and Total U.S. ETS Emissions

For the past 20 years, California cigarette consumption and ETS emissions have continued to decline. Whereas, the total U.S. cigarette consumption and ETS emissions have fluctuated. In 2002, California accounted for over 6% of the total cigarette emissions in the U.S. The quantity of ETS emissions was mainly determined using the most recent emission rate data and 2002 U.S. cigarette consumption numbers (Orzechowski and Walker, 2002). Table IV-4 shows staff's estimated total statewide and U.S. ETS emissions for nicotine, RSP, and CO from cigarettes and cigars.

Table IV-4

California vs. U.S. ETS Emissions

	Nicotine Emissions (tons)		RSP Emissions (tons)		CO Emissions (tons)	
Fiscal Year	CA	Total U.S.	CA	Total U.S.	CA	Total U.S.
2001-02	40	647	365	5,860	1,907	30,200

In 2002, California had a low smoking adult prevalence (16.2%) rate compared to the overall 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.

3. ETS Emissions by Age

In addition to regional emission estimates shown in Appendix B, staff also estimated ETS emissions amongst two age groups: adults and adolescents. These two groups comprise the majority of all California smokers. See Appendix B for a complete discussion for the methodology used by staff.

To characterize reported emissions, Table IV-5 presents the 2002 California adult and adolescent population and cigarettes consumption data.

Table IV-5

2002 California Adult and Adolescent Cigarette Consumption (millions)

	Adult (18+ years of age)	Adolescent (12 - 17 years of age)
Population	25.7	2.8
Smoker Population	4.2	0.4
Cigarettes Consumed	22,994	2,426

Population, smoking prevalence among daily and occasional smokers, and average emission factors were all considered in determining adult and adolescent emissions of nicotine, RSP, and CO, see Table IV-6.

Table IV-6

Adult vs. Adolescent Cigarette ETS Emissions (Tons/Year)

	Adult (18+)	Adolescent (12 - 17)	[a]Total
Nicotine	32.9	3.5	36.4
RSP	303	32	335
CO	1,335	141	1,476

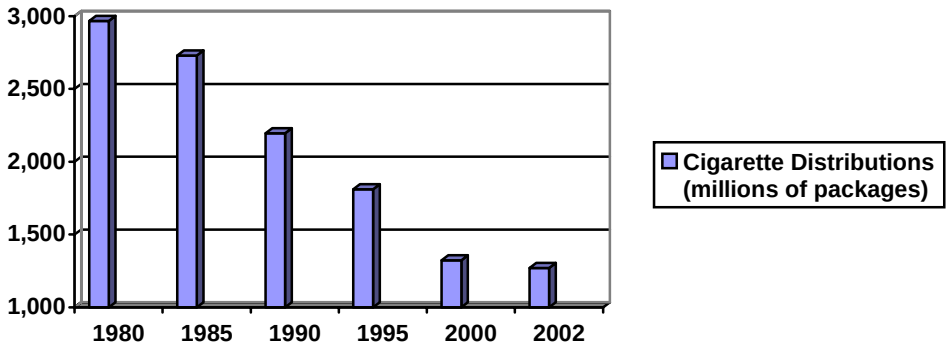
[a] Staff estimates 80-90% of total emissions reside outdoors

E. ETS EMISSIONS PROJECTION

The future trend of ETS emissions largely depends on smoking prevalence in California. Figure IV-1 shows how the adult and adolescent smoking prevalence has declined over the past several years. Likewise, Figure IV-3 indicates that since 1980 cigarette distributions (and per capita consumption) in California have decreased as well.

Figure IV-3

Cigarette Distributions in California



Source: CBOE (2003). *2001-2002 Annual Report, Table 30B – Cigarette Distributions and Per Capita Consumption, 1959-60 to 2001-02*

Current anti-smoking mandates within the California Health and Safety Code (Section 104350-104545) will ensure that California's smoking prevalence among adults and adolescents continues to decrease. In 1989, the California Legislature enacted Assembly Bill (AB) 75, which set an ambitious goal to reduce tobacco use in California by 75% by 1999. While state agencies did not meet the 75% reduction in tobacco consumption by 1999, the California Legislature found that California's anti-smoking campaign, which is overseen by the Tobacco Education and Research Oversight Committee (TEROC), was a success. Per capita cigarette consumption declined by over 50% and adult smoking prevalence was reduced by more than 25% between 1989 and 1999 (TEROC, 2000).

The TEROC was created by Health and Safety Code Section 104365 and is composed of 13 appointed members of varying backgrounds such as public health, research and education. The committee's purview includes oversight responsibilities and advising the Department of Health Services, the University of California, and the State Department of Education on policy development and evaluation of tobacco education. Under Health and Safety Code Section 104370(f), the TEROC is also mandated to develop a "master plan" to attain future reductions of smoking prevalence in California.

The TEROC policy is to continue focusing on programs that prove effective in reducing smoking prevalence and consumption. According to their January 2003 master plan, TEROC's intermediate goal is to reduce total (i.e., daily and occasional smokers) adult smoking prevalence in California to 13% and total adolescent smoking prevalence to 4% by 2005. The long-term goal is to reduce total adult smoking prevalence in California to 10% and total adolescent smoking prevalence to 2% by 2007 (TEROC, 2003).

Therefore, if TEROC's plan to achieve further reductions proves to be successful, then ETS emissions will gradually trend downwards. A quantifiable assessment is not possible, since the ultimate indicator of ETS emissions relates to the total number of cigarettes consumed (i.e., cigarette distributions) by California's smoking public.

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V.

EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE

The purpose of this chapter is to examine the available information on exposure to ETS, and to estimate exposures of various subgroups of the California population to ETS.

Information from Chapter 2 (*Exposure Measurement and Prevalence)* of the OEHHA report (OEHHA, 1997): *Health Effects of Exposure to Environmental Tobacco Smoke* was used as a starting point for the development of this chapter. Literature published subsequent to that report was then reviewed and is summarized in this section. This chapter includes a discussion of ETS exposure prevalence in California; a discussion of markers or surrogates used by researchers to estimate air concentrations of ETS; a review of measured and modeled air concentration studies on the constituents of ETS; and the results of CARB's recent ETS air monitoring study. This chapter also presents scenario-based estimates of selected population subgroups' exposures to ETS under different smoking conditions and includes an assessment of children's exposures to ETS as required pursuant to the State's adoption in 1999 of the Children's Environmental Health Protection Act (Senate Bill 25, Escutia). An assessment of the contribution of indoor exposure to total exposure is also presented in this chapter, as required by Health and Safety Code sections 39660 and 39660.5.

In Part B of this report, which describes the health effects of ETS, OEHHA estimates a range of ETS-related health effects for the California population. The range of estimated health effects is based on today's levels of ETS exposure for all members of the public and represents a range, which corresponds to the range of exposures that are present throughout the State. This report reflects the range of exposures that may be found throughout the State.

A scenario-based approach was used to characterize the range of the public's 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. 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, cigarettes and cigars, the primary sources of ETS, are smaller sources that emit pollutants near people, and ETS is not monitored at ambient monitoring stations. Therefore, because 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.

A. CALIFORNIA ACTIVITY PATTERNS AND ETS EXPOSURE

An individual's exposure is equally 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.

Californians (over 11 years old) spend an average of about 87% of their time indoors. National and California surveys show that children and adolescents spend a majority of their day indoors, especially at home (Phillips *et al*., 1991; Jenkins *et al*., 1992; Klepeis *et al*., 2001a). As shown in Table V-1 below, California adults (over 11 years old) spend about 62% of their time in their home, and children under 12 years of age spend about 76% of their time in the home, on average. Thus, if smoking occurs in an individual's home, exposure in the home typically contributes the major portion of that individual's exposure to ETS.

Table V-1

Percent of Time Californians Spend in Major Locations

AGE	PERCENT OF TIME			
	Inside the Home	Other Indoors	Outdoors	Inside a Vehicle
Children[1]				
0 – 2	85	4	7	4
3 – 5	76	9	10	5
6 – 11	71	12	13	4
All Children (0 - 11)	76	10	10	4
I. Teens 12 – 17	61	27	6	6
II. Adults 18 +	62	25	6	7
III. All Adults and Teens[2]	62	25	6	7

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

Implementation of smoking restrictions at the workplace and public places in California has greatly reduced the overall exposure of non-smokers to ETS. Other non-smokers exposure occurs in many locations, such as at bus stops; entrances to office buildings where smokers congregate; parking lots; outdoor sporting events; outside of airport

terminals; and inside homes of people who smoke. Children with smoking parents generally experience high exposures to ETS due to their proximity to their parents, with the highest exposures typically being inside homes and vehicles. Teens, college students, and elderly individuals may also experience high exposures due to activities with smoking peers and/or roommates or home residents who smoke. However, older children and some adults spend a substantial portion of their non-sleeping time outdoors. For those individuals, outdoor exposure to ETS may predominate, and may be substantial.

As discussed in the next section, data on smoking prevalence and time non-smokers are near smokers indicate that both smoking rates and the exposure of non-smokers are declining in California. By 1999, 37% of non-smoking Californians reported that they had not been near a smoker in the past six months (Gilpin *et al*., 2001). Gilpin *et al*. (2001) also reports that in 1999, 88% of children and adolescents lived in smoke-free homes. These findings and the data in Section C below on indoor concentrations in smoking and non-smoking homes suggest that levels of ETS exposure experienced by Californians range from near zero to very high levels.

B. PREVALENCE OF ETS EXPOSURE IN CALIFORNIA

This subchapter presents an overview of the past and present patterns of adults and children's exposure in California. The prevalence studies only represent the time periods covered by the study. Smoking behaviors and other factors that change smoking patterns such as smoking regulations and smoking customs may affect present and future exposure patterns. For this reason, the information presented in this section primarily focuses on the most recent smoking prevalence studies.

Burns and Pierce (1992) conducted the first of a series of California Tobacco Control Surveys on tobacco use in California since the passage of the Tobacco Tax and Health Protection Act (Proposition 99) in 1988. The survey covered the period between June 1990 and July 1991 and included a sample population of about 12,000 for children ages 0-5 years; about 13,000 children ages 6-11 years; and, about 12,000 adolescents ages 12 - 17 years. Smoking prevalence during this time among adult smokers was 22% and adolescents aged 12 - 17 years was 9.3%. The study also reported that 32% of children under 5 years of age lived in homes with one or more smokers. Similar values were reported for children 6 - 11 years of age (32%) and adolescents 12 - 17 years of age (37%).

Pierce *et al*. (1994) reviewed the progress of several California Tobacco Control Surveys conducted in 1990, 1992, and 1993. Part of the survey included an estimate of the number of women who were exposed to ETS while pregnant. Information from the surveys indicate that the proportion of non-smoking women in California of child-bearing age who are ETS-exposed is estimated to be about 22%. For childhood exposures, the 1993 survey suggests 19.6% of those age-17 and under, and 17.7% of those under age 5 may be exposed to ETS in their homes. Klepeis *et al*. (2001a) compared the data from the National and California surveys for the time children were exposed to a

smoker. The California Children Study (Jenkins *et al*., 1992) showed that children spent most of their time exposed to ETS in a residence (25% of respondents). Children spent a significant portion of their time exposed to ETS in other locations as well (outdoors-15% and in a vehicle-10%). There were not enough California children respondents in the National Human Activity Pattern Survey (NHAPS) to calculate reliable statistics for the time spent with a smoker in different locations. However, in both studies, the minutes spent per day with a smoker in all locations was close (222 minutes for NHAPS vs. 204 for the California Children Study). The percentage of children who spent time with a smoker was lower in the NHAPS (20%) than the California Children Study (38%).

Jenkins *et al*. (1992) also estimated the percentage of adults/adolescents who spent time near a smoker. On a given day, adolescent children (ages 12 - 17) spent an average of 228 minutes of potential exposure in proximity to smokers (adults average 251 minutes). However, a higher percentage of adolescents versus adults reported being near ETS at some time of the day (64% reported yes and 56% of adults reported yes) (Jenkins *et al*. 1992; Miller *et al*., 1998). Table V-2 summarizes the data for time spent near a smoker.

Miller *et al*. (1998) examined exposures of non-smoking Californians (i.e., adults, adolescents, and children) to 17 TACs known to be present in ETS. The investigators used concentration data for a variety of indoor microenvironments in combination with the CARB's activity pattern survey findings to model Californians' ETS exposures in the late 1980's and to make predictions for the late 1990's. The modeling results (for the late 1980's) indicate that of the 62% of adolescents who were exposed to ETS, 62 - 74% of total exposure was in the home, 8 - 18% occurred while in a vehicle, and 4 - 15% occurred in retail and other indoor environments (e.g., shopping malls, beauty salons, etc.). For the 33% of children (ages 7 - 11) exposed to ETS, 70 - 73% of total exposure was in the home, whereas 9 - 18% occurred in vehicles and 6 - 7% occurred in others' homes. The authors' predictions for the late 1990's showed a considerable drop in exposures: 16 - 19% of adults, 33 - 35% of adolescents, and 21 - 23% of children were expected to experience ETS exposure on any given day. Only residences, transportation, and others' residences were examined for the microenvironmental exposure simulations, due to smoking bans in workplaces and public establishments (although non-smokers may be exposed to ETS in public establishments that still allow smoking (Weber *et al*., 2003)). The results predicted that one's own home would be the major site of exposure for all age groups: 58 - 69% for adults, 58 - 66% for adolescents, and 72 - 83% for children.

In a study by Gilpin *et al*. (2001), adolescent (12 - 17 years) smoking prevalence increased between 1993 (9%) and 1996 (12%), but by 1999 had fallen to about 8%, lower than the prevalence in 1990 (9%). An increase in smokefree homes has resulted in lower exposure to ETS in the home. In 1999, 88.6% of children and adolescents lived in smoke-free homes, up from 77% in 1993. The report also suggests that parental reinforcement of strong expectations against smoking for their adolescent youth is strongly associated with low rates (11.7% overall) of adolescent smoking and is likely a key parenting practice to deter adolescent smoking throughout adolescence into adulthood.

Table V-2

Prevalence of ETS Exposure in California

Population	Percent of Non-smokers Reporting ETS Exposures	Reported Average Daily ETS Exposure Duration (minutes)	Reference
Adults	56%	251	Jenkins *et al*., 1992 Miller et al., 1998
Adolescents (12 - 17)	64% 33 - 35%	228 NA	Jenkins *et al*., 1992 Miller *et al*., 1998
Children (0 - 11)	38% 20% 21 - 23%	204 222 NA	Wiley *et al*., 1991b Klepeis *et al*., 2001a Miller *et al*., 1998

C. MONITORING FOR ETS

Tobacco smoke is composed of several thousand individual compounds (Dube and Green, 1982). Pyrolysis, pyrosynthesis, and distillation lead to the formation and emission of these compounds as a mixture in environmental tobacco smoke (Ogden and Jenkins, 1999). Since tobacco smoke is a complex mixture, it cannot be measured directly. Given the complex nature of ETS, it is necessary to select a surrogate measure of exposure that are representative of ETS as a whole. Other methods include source apportionment and modeled emissions.

1. ETS Markers

In 1986, the National Research Council listed attributes for an ideal surrogate or marker for ETS (NRC, 1986). These include uniqueness, ease of measurement, similar emission rate when compared with a variety of ETS constituents, and consistent behavior under a range of environmental conditions. No single ETS component meets all of the attributes of an ideal marker.

Several components of ETS have been studied as markers for ETS. Nicotine has been most widely studied as a potential marker because its only source is tobacco smoke (Hammond *et al.,* 1987). 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. Ninety-seven percent of indoor air nicotine has been found in the vapor phase (Ogden and Jenkins, 1999). Adsorption by nicotine on indoor surfaces complicates indoor air measurements. Adsorption should be less of a concern for outdoor measurements near sources of ETS. Other ETS markers that have been studied include: solanesol, 3-ethenylpyridine (3-EP), carbon monoxide, iso- and anteisoalkanes (C_{29}-C_{34}), PAHs, fluorescing particulate matter, respirable suspended particles (RSP), and ultraviolet particulate matter (Ogden and Jenkins, 1999; Rogge *et al*., 1994). Solanesol, a semivolatile compound adsorbed to

particulate matter, has been used as a marker for particulate matter from ETS in indoor air (Daisey, 1999). However, solanesol is thought to degrade when exposed to ultraviolet light and hence, would not be a good marker for ETS outdoors. Also, solanesol air concentrations may be too low to measure (Jenkins *et al*., 2000) and does not have a steady correlation with RSP levels nor is it consistent across different tobacco products (LaKind *et al*., 1999). 3-EP is better than nicotine as a marker for vapor phase ETS (Jenkins *et al*., 2000). However, analytical standards for 3-EP are not as readily available as for nicotine. Carbon monoxide readily dilutes to near background concentrations away from the source of the ETS (Jenkins *et al*., 2000). Analytical methods have been developed to evaluate particulate matter based on the ultraviolet absorbance and fluorescence characteristics of some particulate matter (Ogden and Jenkins, 1999). These methods provide greater sensitivity for studying these ultraviolet and fluorescing particles within tobacco smoke than simply measuring respirable particulate matter. However, these methods have interferences from non-tobacco combustion sources. Fluorescing, respirable, and ultraviolet particulate matter are not as unique to tobacco smoke as nicotine, solanesol, or 3-EP (Ogden and Jenkins, 1999). Finally, iso- and anteisoalkanes may be more stable as tracers in the outdoor urban atmosphere. Iso- and anteisoalkanes are enriched in cigarette smoke particles and show a concentration pattern characteristic of tobacco leaf surface waxes.

Although several indicators have been determined as markers for ETS, particles and nicotine have been used most widely. Whereas there are many sources of particles in the air with varying background exposures, nicotine is specific to smoking and thus makes a good marker for ETS. Consequently, the ARB study focuses on nicotine as a marker for ETS concentrations and exposures.

2. Ambient Air Monitoring Studies for ETS

Several compounds or groups of compounds have been used to measure ETS in the ambient air. One study by Rogge *et al*. (1994) estimated concentrations of fine cigarette smoke particles in the Los Angeles outdoor air based on measurements of iso- and anteisoalkanes from data collected in 1982. These compounds are associated with tobacco leaf waxes and are preserved in the atmosphere on cigarette smoke particles. Using these marker compounds, ambient fine cigarette smoke particles are estimated to be present at a concentration of 0.28 - 0.36 $\mu g/m^3$ in outdoor Los Angeles air, accounting for 1.0% - 1.3% of the fine particle mass concentration.

Jenkins *et al.* (1996) conducted personal air sampling in sixteen U.S. cities, including Fresno. The monitoring included home and workplace environments with and without exposure to ETS. Monitoring was conducted for eight ETS markers. As found in other studies, homes were found to pose the highest ETS exposure for those who live or work in smoking environments. These data are presented later in this chapter in the section on indoor air concentrations of ETS.

In another California study, Eisner *et al*. (2001) used passive badge monitors to measure personal exposures to ambient nicotine. In this study, fifty adult asthmatics were chosen based on their reported ETS exposures or potential exposures from a

survey administered from an existing asthma cohort study. Each of the study participants wore passive badge nicotine monitors over a 7-day test period and reported ETS exposures in six selected microenvironments (participant's home, another persons' home, in-vehicle, workplace, bars/nightclubs, and outdoor locations). The collected nicotine was analyzed by gas chromatography with nitrogen selective detection. The nicotine concentrations were calculated by dividing the total nicotine collected over the monitoring period, by the estimated volume of air sampled. The results show that the overall median 7-day nicotine concentration was reported to be 0.03 $\mu g/m^3$ in all microenvironments. Measured median nicotine concentrations were highest among persons who reported ETS exposures at home (0.61 $\mu g/m^3$), work (0.03 $\mu g/m^3$), and in other (outdoor) environments (0.025 $\mu g/m^3$).

3. ARB's Ambient ETS Monitoring Study

The CARB staff conducted ambient air monitoring at outdoor smoking areas for nicotine, as part of the CARB's evaluation of ETS as a potential toxic air contaminant. This study was undertaken to provide data to fill in the gaps that existed in outdoor measurements of ETS. Nicotine was used as a surrogate for ETS based on the reasons given previously regarding ETS surrogates. The purpose of this monitoring was to measure air concentrations of nicotine at different locations in California and for different durations (1 - 8 hours). The locations were selected based on potential public ETS exposures. These concentrations were then used to estimate outdoor near-source public exposures to ETS in locations representing several exposure group sub-populations. The mean and highest measured concentration were used from the sites tested to estimate a person's potential mean and high-end exposure to ETS. This was done to show that some Californians may be exposed to levels generally associated with indoor ETS concentrations.

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. A site was chosen in Sacramento as an initial test location to verify that there were no problems with the sampling and analysis methods. No problems were found. The remainder of the monitoring was conducted in southern California.

The California Department of Health Services distributes funds to counties for anti-smoking education programs. Staff in the County Health Departments in Los Angeles and Ventura Counties expressed interest to the CARB in having monitoring conducted in their counties. These two county departments provided funding to the CARB to cover monitoring expenses, in return for CARB conducting ETS monitoring in their counties.

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. For each sampling period, two samplers were situated adjacent to the outdoor smoking area, with a third sampler located away from the smoking area as a background sampler in the expected upwind direction. Several methods have been used for collecting air samples of nicotine (Caka *et al.*, 1990). During this monitoring,

nicotine was collected on XAD-4 adsorbent resin by pulling air through sampling cartridges at a rate of 15 liters per minute. The sampling cartridges contained about 30 milliliters of XAD-4 resin. Analysis was conducted by gas chromatography with a mass selective detector. The estimated quantitation limit (EQL) was 0.029 $\mu g/m^3$ for 1-hour samples, and 0.0036 $\mu g/m^3$ for 8-hour samples. Concentrations measured below the EQLs were reported as "trace."

The CARB staff collected meteorological data including wind speed/direction and ambient temperatures at three of the study sites. They did not collect meteorological data at two of the study sites due to the physical obstacles and variable wind patterns that existed at these sites.

In addition, CARB staff counted the number of cigarettes smoked during each sampling period to determine the subsequent exposures. A summary of the monitoring results is presented in Table V-3. Overall, the results indicate that concentrations of nicotine correspond to the number of smokers in the smoking areas, although factors such as the size of the smoking area and wind speed affected the results, as illustrated by the range in results at individual study sites and between study sites. A complete description of the monitoring and results is contained in Appendix C**.**

Quality assurance samples (trip and field blanks, trip and field spikes, and collocated samples) were also collected. No nicotine was detected in the trip blanks. Some field blanks contained trace levels of nicotine, but all field blanks were below the EQLs. Trip spikes had recoveries that ranged from 72 - 89 percent. Field spikes had recoveries that ranged from 76 - 87 percent. There were two 8-hour and two 1-hour collocated sampling periods with quantifiable levels of nicotine. The comparison of collocated samples (calculated as the difference between the two collocated samples divided by the mean of the two samples) ranged from 32 - 58 percent for the 8-hour samples and was 42 - 54 percent for the 1-hour samples.

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 $\mu g/m^3$ (amusement park). Mean 8-hour background concentrations ranged from 0.009 (junior college) to 0.12 $\mu g/m^3$ (amusement park). It is important to note that the background concentrations measured in this study may not be representative of background nicotine levels throughout southern California. At most sites, the location of the background monitors, due to physical obstacles and/or meteorological conditions, were close to the smoking areas (see Appendix C for more details and the location of sampling sites). However, even at the background site locations, background concentrations were substantially lower than measured in the smoking areas. Mean background 1-hour concentrations ranged from less than the 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).

Table V-3

Results of ARB Nicotine Air Monitoring Adjacent to Outdoor Smoking Areas

Site Tested	8-hour Data	Concentration ($\mu g/m^3$)	Cigarettes Smoked (8 hours)	1-hour Data	Concentration ($\mu g/m^3$) [b]	Cigarettes Smoked (1 hour)
Airport	Mean Day 1 [a] Mean Day 2 [a] 2-Day Mean Range Mean bkgd.	0.61 0.74 0.68 0.48 - 0.99 0.021	261 326 294	Maximum Mean Range Mean bkgd.	1.5 0.72 0.36 - 1.5 0.046	61 75
Junior College [c]	Mean Day 1 Mean Day 2 2-Day Mean Range Mean bkgd.	0.035 0.018 0.027 0.013 – 0.044 0.012	30 34 32	Maximum Mean Range Mean bkgd.	0.15 0.051 0.017 - 0.15 <EQL [d]	5 4
Local Govern-ment Center [c]	Mean Day 1 Mean Day 2 2-Day Mean Range Mean bkgd.	0.066 0.055 0.061 0.042 – 0.073 0.009	59 60 60	Maximum Mean Range Mean bkgd.	0.18 0.097 0.039 - 0.18 <EQL	15 11
Office Complex [c]	Mean Day 1 Mean Day 2 2-Day Mean Range Mean bkgd.	0.12 0.14 0.13 0.11 - 0.15 0.09	261 251 256	Maximum Mean Range Mean bkgd.	0.28 0.19 0.10 - 0.28 0.06	31 29
Amuse-ment Park	Mean Day 1 Mean Day 2 2-Day Mean Range Mean bkgd.	2.6 2.8 2.7 2.4 - 3.1 0.12	653 719 686	Maximum Mean Range Mean bkgd.	4.6 2.4 0.66 - 4.6 0.17	148 91

[a] Mean concentration of samples adjacent to outdoor smoking area.
[b] Maximum, mean, range, and mean background concentration of six 1-hour sampling periods. (Means include all samples, with trace values below the EQL assigned 0.017, the midpoint between the EQL and limit of detection.)
[c] Light to moderate winds occurred on all three days of monitoring at this location.
[d] EQL for 1-hour samples = 0.029 $\mu g/m^3$; EQL for 8-hour samples = 0.0036 $\mu g/m^3$ (1 $\mu g/m^3$ nicotine = 0.15 ppbv).

4. Modeled Ambient Concentrations for ETS

Schauer *et al*. (1996) used a chemical mass balance (CMB) receptor model based on organic compounds to estimate source contributions to airborne fine particle mass concentrations in the Los Angeles air. Receptor-based CMB models use emission source chemical composition profiles to linearly extrapolate source contributions to the measured chemical composition of ambient samples (Watson, 1984). The model was applied to four air quality sites in southern California using atmospheric organic compound concentration data and source emission profile data collected specifically for

the purpose of testing this model (Gray *et al*., 1986, Hildemann *et al*., 1991; Rogge *et. al*., 1993). The contributions to fine organic aerosol of up to nine primary particle source types were identified: diesel engine exhaust, paved road dust, gasoline-powered vehicle exhaust, emissions from food cooking and wood smoke, with smaller contributions from tire dust, plant fragments, natural gas combustion aerosol, and cigarette smoke. Using the fine organic aerosol concentration data and source emission profile data, Schauer *et al*. (1996) estimated an annual average ETS fine particle mass concentration of 0.21 µg/m^3 in the Los Angeles area (average of the four sites studied). Table V-4 summarizes the results from outdoor measurement or modeled studies on the constituents of ETS.

5. Estimated Los Angeles Outdoor Annual Average Ambient ETS Air Concentrations

Although a scenario-based approach was used to characterize the range of the public's exposure to ETS in this report, 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 results from the contribution of occasional or steady state near-source emissions. Since most Californians live and work in urban areas, it would be helpful to ascertain what outdoor ambient ETS levels could exist in these areas. For comparison purposes only, CARB staff estimated an outdoor annual average ambient ETS fine particle concentration for the Los Angeles area for 2003.

This estimate is derived from data collected from studies done by Schauer *et al*. (1996) and Rogge *et al*. (1994). As discussed in previous sections of Chapter V, these studies estimated annual average ETS fine particle concentrations in Los Angeles air based on data from 1982. To calculate a 2003 Los Angeles annual average ETS fine particulate concentration, CARB staff applied an adjustment factor to the 1982 fine PM estimates presented in the Schauer *et al*. (1996) and Rogge *et al*. (1994) studies to reflect reductions in cigarette sales and cigarette emission rates since 1982. Current cigarette sales data (CBOE, 2004) and cigarette emission rate data (Nelson, 1994; Nelson *et al*., 1997; Martin *et al.*, 1997; Repace, 2004) were used for these calculations. The analysis is premised on the assumptions that the ratio of fine particle-emitting sources and fine particle ambient concentrations that existed in 1982 are similar to those that exist today. It was also assumed that the decline in emissions from cigarettes smoked in 1982 to 2003 directly correlates to a linear reduction in outdoor ambient air ETS concentrations. Refer to Appendix D for an explanation of assumptions and the method used to calculate the 2003 Los Angeles outdoor ambient ETS particle concentrations.

Using the estimated annual average ETS fine particle concentrations from two previous studies (i.e., Schauer *et al*. (1996) and Rogge *et al*. (1994)), CARB staff estimated the annual average Los Angeles ETS fine particle concentration in 2003 to range from 0.06 - 0.10 ug/m^3. In addition, and to compare with other outdoor ambient nicotine results, the fine PM concentrations were adjusted by the ratio of fine PM to nicotine (8.1:1) (Nelson, 1994; Martin *et al*., 1997) to calculate a range of Los Angeles annual average nicotine concentrations of 0.008 - 0.013 µg/m^3 (Table V-4).

Table V-4

Estimates of ETS Outdoor Ambient Concentrations

Method/Reference	Data Year	Concentrations ($\mu g/m^3$)	
		Fine $PM_{2.5}$	Nicotine
Fine PM – Source Apportionment Schauer *et al*. (1996)	1982	0.21 $\mu g/m^3$ annual average	*0.026 $\mu g/m^3$ annual average
Iso- and anteisoalkanes – measurement Rogge *et al*. (1994)	1982	0.28 – 0.36 $\mu g/m^3$ annual average	*0.035 – 0.044 $\mu g/m^3$ annual average
Nicotine – measurement Eisner *et al*. (2001)	2001	*0.20 $\mu g/m^3$ 7-day median conc.	0.025 $\mu g/m^3$ 7-day median conc.
Nicotine – measurement CARB (2003)	2003	*0.11 – 25 $\mu g/m^3$ 8-hour range *0.073 – 0.97 $\mu g/m^3$ 8-hour background	0.013 – 3.1 $\mu g/m^3$ 8-hour range 0.009 – 0.12 $\mu g/m^3$ 8-hour background
Los Angeles background – Estimate CARB (2004)	2003	0.06 – 0.10 $\mu g/m^3$ annual average	0.008 - 0.013 $\mu g/m^3$ annual average

* Calculated value using: $PM_{2.5}$/Nicotine concentration = 8.1 (see Appendix C)

D. INDOOR AND PERSONAL AIR CONCENTRATIONS OF ETS

1. Introduction

As discussed earlier in the chapter, ETS is a complex mixture and measurement of all or most of its components is not practicable. Two main approaches have been used to quantify indoor concentrations and exposure: direct methods, using personal monitors and/or measuring biomarkers, and indirect measurement methods, using ETS markers and/or mass balance modeling. Personal monitors measure ETS exposure at an individual's breathing zone. Biomarkers, which are components of ETS or their metabolites found in human physiological fluids, are the best direct means of assessing ETS exposure. However, biomarkers are difficult to obtain relative to indirect markers because they require collection of human body fluid samples, such as urine, serum, or saliva. Thus, indirect methods, primarily measurement of ETS components in indoor air, are the predominant means for quantifying indoor concentrations and exposure.

Markers of ETS should be unique to tobacco smoke, have similar emission rates across cigarette brands, and be found in similar proportions to the ETS component they propose to trace. Nicotine and RSP are the most widely used markers for the presence

and concentration of ETS in indoor environments. Nicotine particularly has been favored because it is specific to ETS and because, in its vapor phase, it is fairly simple and inexpensive to measure. However, critics of its use as a marker note that nicotine in environmental chambers has a different decay pattern than many ETS components. Within a few hours of nicotine emission, 80-90% is deposited on surfaces, whereas RSP is removed largely through building ventilation and thus may vary greatly relative to nicotine over time and with changes in ventilation rates (as reviewed by Daisey, 1999). Sorbed nicotine can be re-emitted from surfaces at significant levels compared to those emitted by active cigarettes, as determined by long-term sampling in areas where smoking occurs regularly (Daisey, 1999). Singer *et al.* (2003) tested the sorption effects of nicotine and other compounds and potential ETS exposures under habitual smoking conditions. The results indicate that indirect exposures (residual ETS when a non-smoker is present after a smoker finishes) accounted for a larger fraction of exposures for nicotine and other sorbing compounds versus non-sorbing ETS components. Indirect routes accounted for about 50 percent of potential nicotine exposures during the non-smoking periods. Despite the sorption and desorption of nicotine, it is still a very useful marker for ETS.

Respirable suspended particulates (RSP) are another commonly used marker. Different authors may refer to RSP as $PM_{2.5}$, $PM_{3.5}$, PM_4 or less in occupational settings, or some other size cut. However, for purposes of this report, most of the RSP in ETS is considered to fall under $PM_{2.5}$, which is typically defined as particles 2.5 µm or less in diameter (NRC, 1986). ETS-related particles typically are less than 1-µm in diameter, so are included in both $PM_{2.5}$ and PM_{10}. Unlike nicotine, RSP is not specific to cigarette smoke, as it is also produced by other indoor combustion sources. However, typically these sources contribute much less to indoor RSP levels than does ETS (OEHHA, 1997), although some styles of cooking may contribute notably to residential RSP levels (Fortmann *et al*., 2001).

Models based on mass balance are another means of indirectly assessing ETS exposure. Although it has been argued that predictions derived from these models are too situation-specific to be generalized to the overall population (OEHHA, 1997), several recent studies have taken steps toward designing models with greater general applicability. For example, recent studies (e.g., Klepeis *et al*., 2001a; Klepeis, 1999) have taken survey data of human activity patterns in California and combined them with models based on a mass balance equation to generalize to a larger population. The Klepeis *et al.* (2001a) study also incorporated point estimates of ETS-related $PM_{2.5}$ concentrations in various microenvironments, thereby allowing even greater ability to predict population-wide patterns. Another study (Repace *et al*., 2000) used actual measured volumes and air exchange rates for 316 California homes to generalize indoor ETS measurements to a broader population.

Three comprehensive reviews on ETS concentrations in indoor air were published in the late 1990's. The most recent review of indoor ETS concentrations, the OEHHA 1997 report: *Health Effects of Exposure to Environmental Tobacco Smoke* (later adopted by the National Cancer Institute's 1999 report entitled *Health Effects of Exposure to Environmental Tobacco Smoke: The Report of the California Environmental Protection*

Agency), includes studies conducted in California prior to 1997 with findings from two earlier major reviews (discussed below). This OEHHA report provides the basis for the pre-1997 information presented in this section.

A 1992 USEPA report, *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders,* examined studies that reported indoor concentrations of various ETS-related air contaminants, focusing primarily on nicotine and RSP. This report reviewed studies published primarily in the 1980's and early 1990's that measured contaminant levels across a broad range of different microenvironments.

An extensive compilation of measured indoor levels of ETS-related components also is presented in a book by Guerin *et al*. (1992), entitled *The Chemistry of Environmental Tobacco Smoke: Composition and Measurement*. Concentrations of nicotine, RSP, carbon monoxide, nitrogen oxide, formaldehyde, volatile organic compounds, and polycyclic aromatic hydrocarbons were compared between smoking and control areas across a wide variety of indoor environments. The data summarized were published mainly from about 1980 - 1991, and were collected both in the U.S. and abroad.

Since these reviews were published, smoking habits in California have changed. Initiation of the California Tobacco Control Program in 1988 and passage of the statewide smoke-free workplace law in 1995 have led to a reduction in smoking by the California population and eliminated smoking at most California indoor workplaces, including restaurants, bars, and gaming clubs. The proportion of California adults who were daily smokers declined from 15.9% in 1990 to 13.0% in 1999 (Gilpin *et al*., 2001). Data also indicate that those who continue to smoke are smoking fewer cigarettes than they had in the past.

Consequently, although the following discussion will reference concentrations before 1997, the emphasis has been placed on indoor ETS studies published from 1997 forward, and on data collected in California to reflect the recent reduction in smoking prevalence. There are a limited number of new studies that reflect the effects of the ban on smoking in California workplaces. In contrast to the reduction in ETS concentrations in the workplace, the levels of ETS constituents in homes are relatively similar to what they were prior to 1997.

2. Indoor Air and Personal Exposure Concentrations of ETS Based on Nicotine Measurements

a. Studies of indoor nicotine concentrations presented in the 1997 OEHHA report

The USEPA review (1992) included studies conducted in a wide variety of indoor environments in the United States. Results of those studies indicate that average indoor concentrations of nicotine prior to 1992 ranged about 100-fold, from 0.3 - 30 $\mu g/m^3$. The average concentrations in residences with one or more smokers typically ranged from 2 - 11 $\mu g/m^3$, with high values of up to approximately 14 $\mu g/m^3$. In data collected from the mid-1970's through 1991, average concentrations of nicotine in

the workplace were similar to or greater than average concentrations measured in residences, and workplace concentrations ranged to levels several times as high as those in homes. The concentrations of nicotine were found to increase as a function of the number of smokers present and the number of cigarettes consumed (USEPA, 1992: Section 3.3.1.2 and pages 3-32 to 3-33). In one study, for example, by Marbury *et al*. (1990) measured the smoking activities of parents and nicotine concentrations in the activity rooms and bedrooms of 48 children under age two. The results show that activity and bedroom concentrations of nicotine in the children's homes increased with the number of cigarettes smoked in the home by their parents. Weekly average concentrations ranged from 0.15 $\mu g/m^3$ in the activity room in a home of non-smoking parents to 12.11 $\mu g/m^3$ in the activity room of a home where both parents smoked.

In the Guerin *et al.* (1992) comprehensive survey of indoor measurements, the maximum nicotine concentrations were 30 $\mu g/m^3$ or less in over 50 percent of the studies examined, and less than 100 $\mu g/m^3$ in 90 percent of the studies. Average indoor nicotine concentrations when smoking was present ranged from about 1 - 40 $\mu g/m^3$, with maximum concentrations substantially greater.

One study reviewed in Guerin *et al*. (1992) clearly illustrates the change in nicotine concentrations when a workplace smoking ban is implemented. Vaughan and Hammond (1990) measured nicotine levels in an office building before and after implementation of smoking restrictions. Prior to the restriction, the average nicotine level at the desk of a non-smoker was 2.0 $\mu g/m^3$. Seven weeks after smoking was restricted, average nicotine measurements at non-smokers' desks ranged from 0.1 - 0.3 $\mu g/m^3$. Off-gassing from smokers' clothing and office furniture may have contributed to residual airborne nicotine levels. There was also evidence of spillover from a smoking floor to a non-smoking floor through a shared air handler. Smoking was allowed at the snack bar, which led to an increase in nicotine levels in that area from about 11 $\mu g/m^3$ before restrictions to an average of 85.4 $\mu g/m^3$ after restrictions. On one occasion, a maximum concentration of 179 $\mu g/m^3$ was measured in the snack bar area. On this floor, the non-smokers' desks had the highest non-smoker nicotine readings in the study (i.e., 0.7 $\mu g/m^3$).

Hammond *et al*. (1995) conducted an extensive workplace nicotine measurement study in Massachusetts. Investigators collected samples with a week-long averaging time to determine occupational exposures to ETS in diverse settings, including offices and production areas. They also evaluated the effectiveness of policies that restrict smoking at the workplace. Results clearly indicate that workplace nicotine concentrations decrease in magnitude from areas where smoking is allowed to areas with restricted smoking, and those where smoking is banned. Mean concentrations in open offices at non-smokers' desks were 14.0 $\mu g/m^3$, 3.4 $\mu g/m^3$, and 0.7 $\mu g/m^3$ for smoking offices, offices with restricted smoking, and offices where smoking was banned, respectively. Similar results were found at non-office workplaces (production areas and fire stations) with mean concentrations of 4.4 $\mu g/m^3$, 2.2 $\mu g/m^3$, and 0.2 $\mu g/m^3$ for smoking areas, restricted smoking, and smoking banned areas, respectively. Nicotine concentrations in offices were higher than non-office workplaces, according to the authors, presumably because they are more enclosed with lower ceilings and less ventilation.

In the OEHHA 1997 report (and 1999 National Cancer Institute Review), the new studies discussed in the review primarily reported personal nicotine concentrations, with only limited information on indoor air concentrations. Key results from those studies are highlighted in Table V-5. Detailed information such as sample size was drawn from the original articles when the information was not included in the OEHHA review.

In Jenkins *et al.* (1996), briefly mentioned in the OEHHA review, investigators used pairs of personal monitors to measure ETS exposure in sixteen U.S. cities, including Fresno, California. Study participants wore a personal monitor in the workplace (for approximately 8-hr) and another monitor away from the workplace (for approximately 16-hr). Data were collected for eight different ETS markers. Total 24-hour mean exposures to nicotine ranged from 0.055 $\mu g/m^3$ for those not exposed to smoking at either the workplace or home, to 3.27 $\mu g/m^3$ for those exposed both at work and home. However, the study population in Jenkins *et al.* (1996) differed notably from the U.S. population on several counts. The study population over-represented females by about 25%, and had nearly double the "some college" population and about 50% more college graduates relative to the U.S. population. Concomitant with the differences in education level, the study population also had a higher income level and a higher percentage in management and professional positions relative to the U.S. population, and therefore a lower percentage of participants in service jobs, production, labor, and other blue-collar positions. The population sampled is known to have a lower proportion of smokers than the population at large; thus the somewhat low levels measured are not surprising. The study sample also differed further from the California population: minority populations (African American, Hispanic) were under-represented in the study relative to the U.S. population, and California has a substantially greater percentage of minority residents relative to the U.S.

In the Particle Total Exposure Assessment Methodology (PTEAM) study (Özkaynak *et al*., 1996), conducted in the early 1990's, sponsored by the USEPA and the CARB, investigators collected exposure data from 178 non-smokers in Riverside, California using indoor and personal monitors with pumps for PM_{10}. They collected vapor-phase nicotine on a filter treated with citric acid. Additional data analyses since 1996 indicate that for participants who reported ETS exposure, personal and indoor nicotine measurements were about 1 $\mu g/m^3$ while those with no reported exposure had concentrations below the limit of detection (0.15 µg/filter, approximately 0.5 $\mu g/m^3$). When Özkaynak *et al*. (1996) performed a stepwise regression on indoor nicotine concentrations (considering air exchange rates, house volume, and number of cigarettes smoked), they concluded that nicotine levels increased by approximately 0.2 - 0.3 $\mu g/m^3$ for each cigarette smoked ($R^2 = 0.35$, n = 227). A regression on personal levels of nicotine, based on minutes of exposure to cigarette smoke, showed that personal exposure increased approximately 0.013 $\mu g/m^3$ for each minute of exposure ($R^2 = 0.37$, n = 334). This study was also included in the OEHHA review.

Table V-5 summarizes the nicotine concentrations measured in smoking environments before 1997, as reported in these review documents.

Table V-5

Summary of Indoor and Personal Nicotine Concentrations[1] in Smoking Environments Before 1997

Source	Range of Concentrations ($\mu g/m^3$)	Mean Concentration ($\mu g/m^3$)	Location
U.S. Environmental Protection Agency (1992)	~0-~14	~2-~11	Residences
	~0-35	~1-~12	Offices
	~0-70	~6-~18	Restaurants
	~0-83	<1-47	Transportation
	~0-25	<1-~13	Other indoor locations
Guerin *et al*. (1992)	0-292	1.6-21	Residences
	0-292	2.0-21	Residences overall[3]
	0.7-69.7	3.8-36.6	Offices
	(0.7-199)	(3.8-75)	(Offices, incl. Cigars)
	0-71.5	1.1-36.6	Offices overall[3]
	(0-199)	(1.1-75)	(Offices overall, including cigars)[3]
	<1.6-43.7	14-15	Restaurants
	0-84.5	2.3-34	Restaurants overall[3]
	≤0.03-112.4	7.1-41	Transportation
	≤0.03-112.4	0.4-1,010[2]	Transportation overall[3]
	0.9-167	11.7-37	Other indoor
	0-167	0.6-106	Other indoor overall[3]
Hammond *et al.*, 1995	<0.1 - > 40		Open office, non-smoker's desk
		14	Smoking allowed
		3.4	Smoking restricted
		0.7	Smoking banned
	<0.1 - >20		Non-office workplace (production and fire station)
		4.4	Smoking allowed
		2.2	Smoking restricted
		0.2	Smoking banned
		~ 1- 2	Smoking homes (prior study, for comparison)

Table V-5 (cont.)

Source	Number of samples, (Averaging time)	Location	Concentration (ug/m3)			Comments
			Personal 95th %ile	Personal Mean	Indoor Mean	
OEHHA (1997)[4]						
Jenkins *et al*. (1996)	122	16 U.S. Cities	9.08	3.27		Exposure at work & away from work
	149		4.39	1.41		Exposure away from work, no exposure at work
	154		2.10	0.686		Exposure at work, no exposure away from work
	555		0.173	0.055		No Exposure to ETS
	("at work" & "away from work" samples, total of 24 hours)					
Özkaynak *et al.* (1996)	334 personal samples	Riverside CA		0.013	0.2 – 0.3	Increase per cigarette smoked
	~ 178 homes for indoor samples			~1	~1	Exposure reported
				ND[5]	ND	No exposure reported
	(12-hr.day & night samples)					

1. Includes all averaging times.
2. Value falls outside of specified range because ranges and means not reported for all studies.
3. May include nonsmoking values (not specified in review).
4. Only selected new studies that were not included in the USEPA and Guerin reviews are reported here.
5. Not detected.

b. Studies of Indoor and Personal Exposure Nicotine Concentrations Since the OEHHA 1997 Report

i) Studies of nicotine conducted in California

In recent studies, investigators have used passive badges to measure personal exposure to nicotine. The passive badges are convenient to use and provide sufficiently sensitive results. In one study, fifty adult asthmatics living in northern California who had reported exposure to ETS were invited to participate in a study to measure their exposure to ETS (Eisner *et al*., 2001). The individuals wore passive nicotine badges for one week. At the end of the week, subjects estimated the time they had spent in different microenvironments containing ETS while they were wearing the passive badge. The subjects' self-reported exposure times were compared to actual measured levels. The median personal nicotine level for the week was determined to be 0.05 $\mu g/m^3$ (range: 0 – 3.69 $\mu g/m^3$) for those participants reporting any indoor exposure to ETS. Based on personal ETS concentrations and time spent in various locations, the investigators estimate the following nicotine concentrations for each microenvironment: home concentrations, 0.61 $\mu g/m^3$; outdoor work concentrations, 0.03 $\mu g/m^3$; and other outdoor concentrations, 0.025 $\mu g/m^3$ (Eisner *et al*., 2001).

ii) Studies of nicotine conducted outside of California

Siegel and Skeer (2003) reviewed existing indoor data on exposure to ETS in free-standing bars, bowling alleys, billiard halls, betting establishments, and bingo parlors (5 B's) as determined by nicotine air concentration levels and compared them to levels of exposure in offices, homes, and restaurants. Studies were included in the review if they reported a mean concentration of nicotine measured in at least one of the 5 B's. A weighted-average of the mean nicotine concentrations reported in each of the studies was calculated for each of the 5 B's. From this data, it was determined that nicotine concentrations in the 5 B's ranged from 9.8 - 76 $\mu g/m^3$ and were 2.4 - 18.5 times higher in than in offices or residences, and 1.5 - 11.7 times higher than in restaurants.

Jenkins *et al.* (2000) reviewed more than 50 separate studies in which nicotine levels were measured in over 125 different environments. However, the data presented in Table V-6 are limited to studies conducted in the U.S. and added since the publication of Guerin *et al*. (1992). As expected, nicotine concentrations in environments without ETS are considerably lower than environments with ETS. For example, the mean of measurements in nonsmoking homes was 0.072 $\mu g/m^3$, while that for homes with smoking ranged from 2.2 - 2.7 $\mu g/m^3$. Mean concentrations in offices for the studies reviewed ranged from 0.7 - 6.1 $\mu g/m^3$. Other workplaces had varying levels of nicotine. Those where smoking was banned had a mean nicotine concentration of 0.86 $\mu g/m^3$, while workplaces with unrestricted smoking had mean nicotine concentrations ranging from 3.4 - 9.4 $\mu g/m^3$. A reported nicotine level for a workplace designated smoking area was 0.30 $\mu g/m^3$. Restaurants and bars had the highest reported nicotine concentrations with means ranging from 5.8 - 14.4 $\mu g/m^3$. Data from the different studies may not be

directly comparable due to differing collection methods (active and passive) and analysis methods of the many studies reviewed.

Graves *et al*. (2000) used data from Jenkins *et al.* (1996) to further examine ETS-associated nicotine levels encountered by non-smokers at reportedly non-smoking workplaces. The authors compared subjects from non-smoking workplaces/non-smoking households to those from non-smoking workplaces/smoking households. Graves *et al*. (2000) found that in smoking households, median and mean personal breathing zone concentrations of nicotine were 0.06 µg/m^3 and 0.24 µg/m^3, respectively, when ETS exposure was reported in the home (*n* = 235), versus 0.02 µg/m^3 and 0.08 µg/m^3 for non-smoking homes (*n* = 813). Thus, nicotine exposures were significantly higher for individuals from self-reported smoking homes as opposed to those who reported no ETS exposure at home. The results from Graves *et al*. (2000) are somewhat low relative to those reported by Jenkins *et al*. (1996) in their earlier papers; this was attributed to the deletion of some data points due to misclassification, apparatus failure, and other data clean-up procedures.

Maskarinec and colleagues (2000) examined ETS exposure in restaurant and tavern workers in the vicinity of Knoxville, Tennessee. The authors collected area samples of nicotine in 32 non-bar areas and 53 bar areas and obtained average concentrations of 6.01 µg/m^3 and 14.4 µg/m^3, respectively.

Nicotine concentrations have been compared in many smoking and non-smoking environments. Hammond (1999) conducted a review of the available literature to assess levels of ETS in a wide variety of workplaces in the United States. The author focused on studies from 1984 - 1999 that used nicotine as an ETS tracer. Comparison among work sites that allowed, restricted, or banned smoking, showed that locations with smoking bans had the lowest exposure levels; typically nicotine concentrations were less than 1 µg/m^3. Conversely, higher mean levels were found in locations where smoking was allowed; generally 2 - 6 µg/m^3 in offices, 3 - 8 µg/m^3 in restaurants, 1 - 3 µg/m^3 in blue-collar workplaces, and 10 – 40 µg/m^3 in bars. In the homes of smokers, mean nicotine values ranged from 1.5 – 5.8 µg/m^3 and median values ranged from 1.0 - 3.3 µg/m^3.

In another study, investigators used passive nicotine badges in a study of homes for a weeklong period to correlate the reported number of cigarettes smoked with measured nicotine levels (Glasgow *et al.*, 1998*)*. This study had 39 participants who lived in homes where smoking occurred; 87 percent were smokers. An average of 148 cigarettes was smoked in each home during the week. The mean measured nicotine value was 5.4 µg/m^3 and ranged from 0.02 - 29.2 µg/m^3. Households that reported no indoor smoking during the monitoring period had significantly lower nicotine levels than those that reported smoking (0.10 µg/m^3 vs. 6.3 µg/m^3, respectively). For households reporting 50 or fewer cigarettes per week, nicotine concentrations were below 3 µg/m^3.

Trout *et al.* (1998) investigated the effects of ETS exposure on employees at a casino in Atlantic City, New Jersey. As part of this study, ten general area air samples were tested for nicotine vapor. On a Thursday evening, the area time weighted average for

nicotine had a geometric mean of 8 µg/m^3 and a range of 6 - 12 µg/m^3; on a Friday evening, the mean and range were 11 µg/m^3 and 8 - 16 µg/m^3, respectively.

In another study (conducted in Texas), nicotine concentrations in 50 homes with infants ranged from 0 - 16.55 µg/m^3, with a median of 0.40 µg/m^3. Investigators mailed a passive nicotine monitor to each home then instructed the participants over the telephone on how to place the monitor in their home. The results indicate that 68% of the women in these homes reported that they smoked, while 32% reported that only their partners smoked (Hudmon *et al*., 1997).

Nicotine concentrations from studies published after 1996 are summarized in Table V-6.

Table V-6

Summary of Indoor Nicotine Concentrations in Smoking Environments After 1996

Reference	Number of samples, (Averaging time)	Location	Concentration (µg/m^3)			Comments
			Personal		Indoor Mean	
			Range	Mean		
Siegel and Skeer, 2003	940[1] 91 402 4 6 3 27 3 (Variable averaging times)	Offices Residences Restaurants Betting establishments Bowling Alleys Billiard halls Bars Bingo Parlors			4.1 4.3 6.5 9.8 10.5 13.0 31.1 76.0	Weighted mean concentrations were reported from all studies in each of the study locations
Eisner *et al*. 2001	20 people 7 people 12 people (1 week passive)	Places visited by asthmatic adults, CA	0-3.69 (25th-75th quartile)	0.05 0.03[2] 0.61[2] 0[2]		Subjects reporting indoor exposure (12 with outdoor exposure also) Outdoor Work Subjects reporting home exposure Subjects reporting no exposure

Table V-6 (cont.)

Reference	Number of samples, (Averaging time)	Location	Concentration (µg/m^3)			Comments
			Personal		Indoor Mean	
			Range	Mean		
Jenkins *et al*. review 2000[3]	Sample size 47 – 899	Homes			0.072 (95% = 0.19)	Nonsmoking homes
					2.2-2.7 (range = 0.1-9.4)	Smoking homes
	Largest study had 16 offices	Offices			0.7-6.1 (range = 0.2-16.7)	
	703 workers	Workplace	0.21 (95th %)	0.086		Smoking banned
	52 workers		2.21 (95th %)	0.30		Designated smoking areas
	134 workers		15.0 (95th %ile)	3.4		Unrestricted smoking
	18 casino workers		4-14 (range)	9.4		
	4 to 9-hr shifts, 162 workers across 4 studies (Various averaging times)	Restaurants and bars	29-44 (95th % ile)	5.9-14.1	5.8-14.4 (95th % ile-36-50)	
Graves *et al.* 2000	235 people	16 U.S. cities		0.24 0.06[2]		Home exposure reported
	813 people			0.08 0.02[2]		No home exposure reported

Table V-6 (cont.)

Reference	Number of samples, (Averaging time)	Location	Concentration (µg/m^3)			Comments
			Personal		Indoor Mean	
			Range	Mean		
Maskarinec *et al.* 2000		49 establish-ments Knoxville, TN				Smoking Permitted
	32 area	Non-bar Area			6.01 (0-49.3) range	
	53 area	Bar Area			14.4 (0-61.3) range	
	80 personal	Bartenders	0-116	14.1		
	83 personal	Waiters	0-67.9	5.83		
	(4 to 8 hour samples)					
Hammond, 1999 (review)	Sample size varies across studies	Indoor workplaces			<1	Smoking ban
		Offices		1.8-48.35	1.7-21.95 (34.4; 71.5)	Smoking permitted (90^{th} %ile; max.)
					0.27-7.87	Smoking restricted
					0.1-2.83	Smoking prohibited
		Restaurants Cafeterias		4.5-43	3.4-8.4 <1-14	Smoking permitted
		Bars/ Nightclubs			10-40 7.36-65.5	Smoking permitted
		Blue-collar workplaces			<1-6	Smoking permitted
		Other non-office		0.8-24.8	0.6-5.83	Smoking permitted
					0.17-5.85	Smoking restricted
					<1	Smoking prohibited
	5 homes (1 hour or greater)	Smokers' homes			1.5-5.8	

Table V-6 (cont.)

Reference	Number of samples, (Averaging time)	Location	Concentration (µg/m^3)			Comments
			Personal		Indoor	
			Range	Mean	Mean	
Glasgow *et al*. 1998	39 homes, 1 week (passive)	Homes w/ 1 or 2 Smokers			5.4	Overall mean (mean of 148 cigarettes smoked/ week)
					0.02-29.2	Overall range (mean of 148 cigarettes smoked/ week)
					0.10	Homes with no smoking during monitoring period
					6.3	Homes with smoking during monitoring period
					<3	Actual concentration smoking homes, ≤50 cigarettes smoked/ week
Trout *et al.* 1998	Approx. 8 hr	Casino Atlantic City, NJ	6-12	8		TWA range Geometric mean. Thursday
			4-15	10	8-16 11	TWA range Geometric mean. Friday
Hudmon *et al*. 1997	50 homes (2 weeks passive)	Homes with infants			0.40[2] (0-16.55)	Smoking homes

1. Number of establishments sampled
2. Median value.
3. Data presented for this entry are only from studies published since 1996 and summarized in Appendix 2 of Jenkins *et al*. 2000. For additional details, refer to original articles.

3. Indoor Air and Personal Exposure Concentrations Based on ETS-Associated Respirable Particulate Matter

a. Studies of Indoor and Personal RSP Concentrations Presented in the 1997 OEHHA Report

Measurements of ETS-associated RSP were summarized in the USEPA document (1992: Figures 3-5, 3-8, and 3-10). An extensive compilation of RSP measurements is also provided in Guerin *et al*. (1992). The 1997 OEHHA report, *Health Effects of Exposure to Environmental Tobacco Smoke*, summarized additional studies that were relevant to California and published by 1996. As with nicotine, these studies may not be representative of current ETS-associated RSP concentrations due to the decrease of smoking in California, particularly at the workplace.

According to USEPA (1992), measured concentrations of ETS-associated RSP ranged about 100-fold, from 5 - 500 $\mu g/m^3$ over a wide variety of indoor environments. In residences with one or more smokers, average daily or weekly concentrations of ETS-associated RSP were increased about 20 - 100 $\mu g/m^3$ over concentrations in similar non-smoking environments. Somewhat lower levels are reported in the workplace (offices), with average concentrations ranging from 2 to 60 $\mu g/m^3$ over concentrations in similar non-smoking environments. Both the maximum reported concentrations (1,370 $\mu g/m^3$) measured in any environment and the highest range of average concentrations (35 - 986 $\mu g/m^3$) were measured in restaurants (USEPA, 1992: Figure 3-8).

Variable measurement methods make it difficult to compare RSP results from different studies. Guerin *et al*. (1992) concluded that most RSP levels are less than 100 $\mu g/m^3$ in control or non-smoking environments. However, he noted exceptions to this statement. When smokers are present, RSP levels range from a small increase over background to as much as three-times the background concentration, or more. Guerin *et al.* (1992) reported a high concentration range for RSP of 100 – 300 $\mu g/m^3$, and concentrations above 300 $\mu g/m^3$ as extreme.

Studies discussed in OEHHA (1997) reported RSP concentrations are consistent with other reviews. The OEHHA 1997 review reported particle data from the PTEAM study conducted in Riverside, California (incorrectly cited as Pellizzari *et al* (1992) instead of from Clayton *et al*. (1993)). In that study, 12-hour daytime residential PM_{10} concentrations were consistently higher in homes with smokers, than in homes without smokers. The average PM_{10} residential concentration was 125.6 $\mu g/m^3$ in homes with smokers and 87.8 $\mu g/m^3$ in homes without smokers. A similar difference was observed for nighttime PM_{10} measurements: both daytime and nighttime smoking vs. nonsmoking differences were statistically significant.

Jenkins *et al*. (1996) measured RSP ($PM_{3.5}$) in the “16 Cities Study” previously discussed in the nicotine section of this chapter. Investigators measured personal concentrations while subjects were at work, yielding about an 8-hour sample, and then had subjects wear another sampler while they were away from work, yielding about a 16-hour “away from work” sample. The mean personal concentration for those in a

smoking environment both at work and away from work was 47.0 µg/m^3. The mean concentration for those not exposed to smokers in any location was 18.1 µg/m^3. Workplace exposure levels for those exposed only at work averaged 28.7 µg/m^3, and personal exposures for those exposed only when away from work averaged 33 µg/m^3. As discussed in the nicotine section above, this study suffered from bias due to selection of a less exposed population.

In another study reported in the OEHHA (1997), Ott *et al.* (1996) repeatedly measured RSP concentrations in a sports tavern in California before and after a smoking prohibition took effect. The investigators measured $PM_{3.5}$ inside and just outside of the tavern; average readings were taken approximately every 2 minutes. During pre-ban visits, it was determined that on average, 1.17 cigarettes were active at any given time. Results from this study indicate that the average indoor RSP concentration was 56.8 µg/m^3 above outdoor levels before the ban, and 5.9 µg/m^3 above outdoor levels (a 90% decrease) in the first two months following the ban. In subsequent months, indoor RSP concentrations were 12.9 µg/m^3 above outdoor levels (77% decrease compared to the smoking period). Ott *et al*. (1996) also determined RSP concentrations produced by four cigars smoked in the center of the tavern. No customers were present during this experiment, but ventilation sources (e.g., cooking grill ventilation, windows, and doors) were adjusted to typical positions during business hours. Indoor RSP concentrations reached a maximum of nearly 800 µg/m^3 before the cigars were extinguished; these concentrations decayed to initial levels after approximately 20 minutes.

Concentrations of RSP (and PM_{10}) reported in studies published before 1996 are summarized in Table V-7.

Table V-7

Summary of Indoor Particulate Matter Concentrations[1] in Smoking Environments Before 1997

Source	Concentrations (µg/m^3)				Location/ Comments
	Smoking		Background/Controls		
	Range	Mean	Range	Mean	
U.S. Environmental Protection Agency (1992)	~5 – 560	~15 - ~95			Residences
	~2.5 - 90	~2.5 - ~60			Offices
	~12 – 1,370	~35 – 986			Restaurants
	~0 – 850	~0 - ~100			Transportation
	~0 – 1,140	~0 – 295			Other indoor
Guerin *et al*. (1992)	0.7-3,150	36-700	0-2,050	0.7-300	Residences
	0-1,088	27-720	4-208	6-300	Offices
	0-685[2]	26-690[2]	15-57[3]	24-400	Restaurants
	0-4,980	18-1,000	3-1,830	15-500	Transportation
	<5-6,220	29-1,947	0-2,200	9.1-520	Other indoor

Table 7 (cont.)

Source	Number of Samples, (Averaging Time)	Location	Concentration (µg/m^3)		Comments
			Smoking	Nonsmoking	
OEHHA (1997)[3]					
Clayton *et al*., (1993)			125.6[3]	87.8[3]	Daytime 12-hr home, w/smokers, 12-hr home w/out smokers
Jenkins *et al*. (1996) (nonsmokers; personal exposures)	122	16 U.S. Cities. Measured PM3.5	47.0 (95%=117)		Mean personal conc., exposed at work & away from work
	149		33.0 (95%=76.3)		Mean personal conc., exposed away from work only
	154		28.7 (95%=75)		Mean personal conc., exposed at work only
	555 At work and away from work samples over 24 hr			18.1 (95%=41.5)	Mean personal conc., no exposure to ETS
Ott *et al.* (1996)	~2 min intervals, up to 2 hr duration 26 dates	1 Sports tavern, Menlo Park, CA	56.8[1]		Mean increase over outdoor levels before smoking ban
	10-second intervals, 40 min total			5.9	Mean increase over outdoor levels, first two months after smoking ban
				12.9	Mean increase over outdoor levels, more than two months after smoking ban
			~800		Maximum from 4 cigars (using piezobalances)

1. Covers a range of averaging times and methods. Studies conducted outside of the United States were excluded from this table when this information could be deduced from the review articles.
2. Mean exceeds maximum value of the range because means and ranges were not reported for all studies.
3. Only selected new studies that were not included in the USEPA and Guerin reviews are reported here.

b. Studies of Indoor RSP Concentrations Since OEHHA 1997 Report

i) *Studies of RSP conducted in California*

Several studies examining indoor RSP from smoking in California have been completed since 1996. Most of the California studies used real-time monitors, yielding valuable information regarding peak and short-term concentrations and exposures in specific locations, such as bars and bingo parlors. Some of these studies clearly illustrate the benefits of smoking restrictions and bans on reducing airborne concentrations of respirable particles in these locations. While these studies do not necessarily contribute to knowledge bases of long-term population exposures, they do provide useful information for assessing the peak exposures experienced by patrons of entertainment establishments, which often include senior citizens and others who may be especially sensitive to the adverse effects of cigarette smoke. In Table V-8, several studies listed provide short-term measurements of this type. The other studies in Table V-8 provide longer duration measurements that are more useful for estimates of general, long-term population exposure.

Switzer *et al.* (2001) measured ETS pollutants at one-minute intervals in a variety of Northern California public locations, some before and some after smoking was banned. Measurements at a church-sponsored bingo game, where smoking was permitted, found indoor RSP levels that were 87 - 348 $\mu g/m^3$ above outdoor levels. When the church banned smoking at its bingo games, measured RSP levels in the same building (on 11 subsequent visits) were at most, 15 $\mu g/m^3$ above outdoor levels. In general, statistical analysis of the pollutant data, in combination with active cigarette counts, showed that RSP levels increased by about 32 $\mu g/m^3$ for each additional active cigarette. Based on 1992 - 1994 activity data, and using statistical modeling techniques, the investigators estimated that 1.5 - 3.5% of Californians would receive a 24-hour ETS-particle exposure exceeding 20 $\mu g/m^3$.

Klepeis (1999) measured RSP and carbon monoxide (CO) in a San Francisco, California, restaurant/ bar. Over a two-hour period there was on average, one smoker actively smoking at a time. This resulted in an average RSP concentration of 68 $\mu g/m^3$ (range = 36 – 116 $\mu g/m^3$) above background levels (measured just outside of the bar) for an approximately 800 m^3 room.

In another study conducted in San Francisco, California, Klepeis *et al*. (1999) examined the contributions of cigar and cigarette smoke to $PM_{3.5}$ levels in a residence. When a single cigar was smoked in the parlor, a mean $PM_{3.5}$ concentration of 160 $\mu g/m^3$ and a peak of 350 $\mu g/m^3$ were recorded. In contrast, one cigarette smoked in the same room produced mean and peak values of 65 $\mu g/m^3$ and 160 $\mu g/m^3$, respectively. $PM_{3.5}$ emission rates also were calculated in this study: the emission rate for a cigar smoked for 90 minutes was 0.98 mg/min, whereas the cigarette's emission rate was 1.9 mg/min. However, due to the much larger mass and resulting longer duration of the cigar, the total RSP emissions of the cigar were about five times higher than for the cigarette (88 vs. 17 mg).

Klepeis *et al*. (2001b) used a total human exposure model to estimate particulate ETS ($PM_{2.5}$) exposures to children. Concentration data from six locations were used along with activity pattern data (Wiley *et al.*, 1991a) to estimate ETS $PM_{2.5}$ concentrations. In all locations examined, it was estimated that 66% of children experience no exposures to ETS. Of those exposed to ETS, 21% were exposed to concentrations of 0 - 10 $\mu g/m^3$; 8% to 10 - 65 $\mu g/m^3$; and 5% greater than 65 $\mu g/m^3$ (i.e., the 24-hour average National Ambient Air Quality Standard for $PM_{2.5}$). The results indicate that although most children are not exposed, a significant percentage are exposed to ETS at concentrations which compare to elevated levels found indoors with smokers present.

In another study, Ott *et al.* (2005) (not shown in Table V-8) measured short-term peak RSP levels as part of a project to validate a multi-compartment model. Two-minute real-time measurements of CO, RSP, particle-bound polycyclic aromatic hydrocarbons (PAH), and $PM_{3.5}$ emitted by cigarettes and cigars were measured in a one-bedroom home in Redwood City, California. When an individual smoked one cigarette in the bedroom, $PM_{3.5}$ levels rose to about 300 $\mu g/m^3$ in 20 minutes, followed by a gradual two-hour decay to background levels. The smoking of three Kentucky reference cigarettes (No. 2R1), one after the other, in the bedroom of a home in Menlo Park, California caused extremely high RSP concentrations, with a peak of 5,500 $\mu g/m^3$. Measurements were taken simultaneously in the adjacent living room (with the door between the rooms remaining open). Despite the fact that the cigarettes were being smoked in the bedroom, RSP concentrations equilibrated at approximately 2,000 $\mu g/m^3$ between the living room and bedroom after 45 minutes. These results illustrate that short-term peak concentrations of RSP can be extremely high in homes where smoking occurs, including in rooms other than those where the smoker is smoking.

ii) *Studies of RSP Studies Conducted Outside of California*

Investigators outside of California are also measuring the effects that smoking bans have on RSP levels. To assess the effects of a smoking ban on indoor air quality in Delaware, eight hospitality venues (a casino, a pool hall, and six bars) were sampled for respirable suspended particulates ($PM_{3.5}$) before and two months after the ban (Repace, 2004). Prior to the ban, the average RSP level was 231 $\mu g/m^3$ (about twenty times the average outdoor background level of 11 $\mu g/m^3$). The average RSP measurement at each venue ranged from 44 - 686 $\mu g/m^3$. ETS contributed 90 - 95% of these indoor RSP levels. For comparison, the annual average National Ambient Air Quality Standard (NAAQS) for $PM_{2.5}$ is 15 $\mu g/m^3$, and $PM_{3.5}$ (which was examined in this study) is closely related to $PM_{2.5}$. On average, 5% of the patrons at these establishments were actively smoking at any given time. Following the ban, the average RSP concentration was reduced to only 9.4% of the pre-ban value (range of averages for each venue: 2.5 - 119 $\mu g/m^3$), which, with the exception of one venue, was very similar to outdoor levels. Measurements from each venue were collected for approximately 30 minutes using a pump-driven real-time aerosol monitor.

Using previously published personal monitoring data collected from sixteen U.S. cities, Graves *et al*. (2000) examined ETS-associated ultraviolet-absorbing particulate matter (UVPM) levels encountered by non-smokers at “nonsmoking” workplaces (i.e., smoking

typically did not occur within 100 feet of the subjects' personal workspaces.) The authors compared subjects from nonsmoking workplaces/nonsmoking households with those from nonsmoking workplaces/smoking households. Median levels of UVPM were 1.07 µg/m^3 for subjects from smoking homes (n = 235) and 0.82 µg/m^3 for those from nonsmoking homes (n = 813) (mean values were 3.27 µg/m^3 vs. 1.54 µg/m^3, respectively). These UVPM exposures were significantly higher for individuals from self-reported smoking homes as opposed to those who reported no home ETS exposure**.** As discussed earlier, these results are lower than other studies that measured PM-related ETS exposures.

In an ETS study conducted in a casino in Atlantic City, New Jersey, Trout *et al.* (1998) measured respirable dust concentrations ranging from undetectable (i.e., below the detection limit of 20 - 30 µg/m^3) to 90 µg/m^3.

In a study of restaurant and tavern employees in Knoxville, Tennessee, mean RSP levels of 73 µg/m^3 in non-bar areas and 135 µg/m^3 in bar areas were measured (Maskarinec *et al*., 2000). These researchers also measured UVPM concentrations in the two aforementioned settings, and found mean levels of 29.4 µg/m^3 in non-bar areas and 95.0 µg/m^3 in bar areas.

Table V-8 summarizes indoor particulate matter concentrations in smoking environments reported in studies published after 1996.

Table V-8

Summary of Indoor Particulate Matter Concentrations in Smoking Environments After 1996

Reference	Number of samples, (Averaging time)	Location	Measured	Concentration (µg/m^3)		Comments
				Smoking	Non-smoking	
Repace (2004)	Each venue sampled once before ban and once following ban (30 minute real-time)	8 Hospitality venues, DE	$PM_{3.5}$	231		Mean before smoking ban
				44-686	2.5-119	Range of means across venues, before and after smoking ban
		1 Casino		205	9.4	
		6 bars/ restaurants		44 – 337	2.5 – 24	
		1 pool hall		686	119	
Offermann *et al*. (2002)	Real-time samples (1 second interval: approx 18 minutes total) During smoking of 1 low-tar cigarette	1 Minivan, CA	$PM_{5.0}$	92		Mean, windows open, vents closed
(Discussed in Section 5 of this chapter)				693		Mean, windows closed, vents open
				1,195		Mean, windows and vents closed
						Outdoor RSP ranged from 4 - 7 µg/m^3

Table V-8 (cont.)

Reference	Number of samples, (Averaging time)	Location	Measured	Concentration ($\mu g/m^3$)		Comments
				Smoking	Non-smoking	
Switzer *et al*. (2001)	23 visits total, up to 2 hr duration (1 min sampling intervals)	Church bingo games, 1 building northern CA	$PM_{3.5}$	87-348		Increase over outdoor levels before smoking ban
					≤15	Increase over outdoor levels after smoking ban
Graves *et al*. (2000)	235 people	16 U.S. cities	UVPM	3.27	1.54	Mean, home exposure reported
	813 people			1.07	0.82	Median, no home exposure reported
Maskarinec *et al.* (2000)		49 establishments, Knoxville, TN				
	32 area samples	Non-bar area	RSP	73 (0-233)		Smoking Permitted
	53 area samples	Bar area	RSP	135 (0-768)		
	80 Bartenders		RSP	151 (0-511)		
	83 Waiters (4-8 hours)		RSP	110 (0-474)		

Table V-8 (cont.)

Reference	Number of samples, (Averaging time)	Location	Measured	Concentration (µg/m^3)				Comments
				Smoking		Non-smoking		
				Range	Mean	Range	Mean	
Jenkins *et al*. (2000)[2] Review	28-899 homes 15-hr samples:	Homes	RSP	12-825	44-89	8-100	20-28	
	1-25 offices 4- and 8-hr samples:	Office	RSP	12-392	27-99	18-35	2-25	
	Sample size variable 4-, 6-, 8-hr samples: one study 0.4-2 hrs.	Restaur-ant, nightclub, tavern	RSP	11-428	57-190	0-66	38-62	Personal exposure for bartenders and waiters fall within the ranges given
	703 workers per study 8- and 9-hr samples: 28-	Workplace	RSP	18-181	62-67	0-98	17-30	Value of 181 at high end of range for smokers is 95th percentile
	2 lounges 1-3-hr samples:	2 Airport smoking lounges	RSP	65-177	114	5-23	13	

Table V-8 (cont.)

Reference	Number of samples, (Averaging time)	Location	Measured	Concentration (µg/m^3)		Comments
				Smoking	Non-smoking	
Klepeis (1999)	2 hr duration	1 Smoking restaurant/ bar, San Francisco, CA		68 36-116		Mean increase over background levels (just outside bar); 1 active smoker on average Range of increases over background levels
Klepeis *et al*. (1999)	2-min averages 4.75 hr duration 2.75 hr duration	1 Home, San Francisco, CA	PM 3.5	160 350 65 160		Mean concentration in parlor, 1 cigar smoked Maximum concentration in parlor, 1 cigar smoked Mean concentration in parlor, 1 cigarette smoked Maximum concentration in parlor, 1 cigarette smoked
Trout *et al.* 1998	9 samples (8 hour duration)	1 Casino, Atlantic City, NJ	RSP	<20 – 90	--	Range

1. 1.17 active cigarettes, on average.
2. Data included in this table are based on studies published since 1996 and summarized in Appendix 1 of Jenkins *et al.* (2000). For additional details, refer to original articles.

4. Indoor Air Concentrations Based on Measurement of Other ETS Constituents

a. Studies of Other ETS Constituents Presented in the 1997 OEHHA Report

Environmental tobacco smoke contains numerous hazardous air pollutants (HAPs) and toxic air contaminants (TACs). Concentration data for select constituents of public health concern, including *N*-nitrosamines, benzene, benzo[a]pyrene and total polycyclic aromatic hydrocarbons (PAHs), carbon monoxide, formaldehyde, and toluene are presented in USEPA (1992: Table 3-3 and Figure 3-3), as are references to the literature (USEPA, 1992: Section 3.3.1). An extensive compilation of data from measurements of a variety of ETS-derived constituents is also given in Guerin *et al*. (1992).

b. Studies of Other ETS Constituents Since OEHHA 1997 Report

i) Studies Conducted in California

Several studies have been published since 1995 that report concentrations of other ETS constituents in ETS environments, including several conducted in California. Particle-bound PAHs were measured in a multiple pollutant study conducted in California by Ott *et al*. (2005). When one cigarette was smoked in the bedroom of a small home in Redwood City, concentrations of PAHs peaked in the bedroom at approximately 0.07 $\mu g/m^3$ (door was closed) after 20 minutes before slowly decaying over a 2-hour time period. When three cigarettes were smoked, one after the other, in another home in Menlo Park, the PAH level peaked at about 1 $\mu g/m^3$ in the bedroom, this time with the door open to the rest of the house.

Carbon monoxide (CO) is another constituent of tobacco smoke. In contrast to the large database available on pollutant concentrations from cigarette smoking, much less is known about the levels of pollutants due to cigar smoke. Consequently, Klepeis *et al*. (1999) measured concentrations at a cigar social in a well-ventilated private club in suburban San Francisco attended by about 50 people. The average CO concentration was about 6 ppm (range = 5 - 11 ppm), with the highest concentration measured on a balcony in the main hall where 18 individuals were smoking. Corrected for ambient CO levels, the authors estimated that the active smokers contributed 4.5 ppm of CO, which was about the same concentration that was measured in freeway rush-hour traffic en route to the event. At a second event, held at a restaurant in downtown San Francisco and attended by 40 people, CO levels were 13 - 17 ppm with about 24 active smokers. The CO concentration was 10 ppm (9 ppm over ambient levels) averaged over the entire 3.3 hour visit, during which over 100 cigars were smoked. If the social event had lasted for 8 hours, it could have exceeded the USEPA's NAAQS of 9 ppm over an 8-hour period.

Additionally, Klepeis *et al*. (1999) investigated the contributions of cigar smoke to indoor levels of CO and particle-bound ($PM_{2.5}$) PAHs in various locations around San Francisco. Cigars were machine-smoked in an office for an average of 19 minutes each (range = 7 - 40 minutes), resulting in peak CO concentrations ranging from 3 - 19 ppm. One-hour time-averaged concentrations exceeded 8 ppm for six out of seven cigars when the air exchange rate was below two air changes per hour (ach). Average CO emissions for the cigars ranged from 14 - 140 mg/min, with total emissions ranging from 630 – 1,200 mg/cigar. These values are substantially higher than the total CO emissions of 40 - 70 mg typically reported for cigarettes (Klepeis *et al.*, 1999).

Emission rates for PAHs were compared in a study conducted in a residence. The PAH emission rate for a cigar smoked for 90 minutes was 0.0042 mg/min, whereas the cigarette's emission rate was 0.015 mg/min. However, total PAH emissions from the cigar were about three-times higher than that of the cigarette (0.38 vs. 0.14 mg, respectively) due to the much larger mass and smoking duration of the cigar (Klepeis *et al*., 1999).

In another study examining cigar emissions, Ott *et al*. (1996) measured CO concentrations resulting from cigar smoke in a sports tavern in Menlo Park using Langan L16 monitors. Four cigars were smoked in the center of the tavern when no customers were present, with all ventilation sources (e.g., cooking grill ventilation, windows, and doors) adjusted to simulate “typical” conditions during business hours. At three different locations in the tavern, indoor CO levels reached peaks of 4.5 - 6.0 ppm after 10 - 15 minutes.

While emissions from cigars and cigarettes vary in magnitude, the variability in emissions between brands of cigarettes is relatively low. Daisey *et al*. (1998) conducted a chamber study testing six of the most popular commercial brands in California and one reference cigarette for emissions of 21 different air toxics and other airborne compounds, including volatile organic compounds (VOCs), nicotine, aldehydes, and airborne particulate matter (estimated to be $PM_{2.5}$). Diluted sidestream smoke (produced by a smoking machine that smoked three cigarettes sequentially) was used to approximate ETS aging in a room-sized chamber, and a mass-balance model was used to generate estimates of indoor concentrations. Among the VOCs, acetaldehyde and formaldehyde displayed the highest emission factors (average emission factors were 3,340 ng/mg tobacco and 2,040 ng/mg tobacco, respectively), and PM had an emission factor of 12,400 ng/mg. These results suggest that ETS has a substantial influence on indoor concentrations of these compounds.

ii) Studies of Other ETS Constituents Conducted Outside of California

Two noteworthy studies measuring PAH concentrations were recently conducted in the eastern United States. Repace (2004) measured particulate polycyclic aromatic hydrocarbons (PPAH) at eight hospitality venues in Delaware, before and two months after effecting a smoking ban. Prior to the ban, the average PPAH concentration was 134 ng/m^3 (averages for each venue ranged from 44 - 249 ng/m^3), about five times the

outdoor background level of 27 ng/m^3. ETS was responsible for 85 - 95% of these PPAH levels. Following the ban, the average PPAH level was 4.7% of the pre-ban value (range of average values = 1.3 - 11 ng/m^3), which was basically indistinguishable from outdoor levels. Measurements were collected for approximately 30 minutes using a pump-driven real-time particle-bound polycyclic aromatic hydrocarbon monitor.

Chuang *et al*. (1999) investigated PAH exposures to children in low-income rural and inner-city areas in North Carolina. The researchers determined that potentially carcinogenic PAH concentrations in smokers' homes were significantly higher than in nonsmoking homes (geometric mean = 6.14 ng/m^3 vs. 1.38 ng/m^3, respectively)

In the previously discussed sixteen Cities Study (Jenkins *et al.*, 1996), a number of ETS constituents were measured as indicators of ETS. In addition to nicotine and RSP, these included 3-ethenyl pyridine, myosmine, ultraviolet absorbing PM (UVPM), fluorescing PM, scopoletin, and solanesol. These indicators generally tracked with expected ETS exposure levels, measuring highest in personal exposures of those who worked and lived in smoking environments and lowest in personal exposures of those living and working in non-smoking environments.

5. ETS Concentrations in Vehicles

Vehicles provide small-enclosed environments that can result in extremely high exposure to ETS when smokers are present. Investigators have used both direct and indirect methods to determine ETS levels in vehicles. Offermann *et al.* (2002) measured levels of particulate matter (less than 3 µm in diameter) resulting from smoking a single low-tar cigarette inside a minivan under different ventilation conditions. Observed air exchange rates ranged from 4.0 ach for windows closed and ventilation off, to 71 ach for windows open and ventilation off. As shown in Table V-8, during smoking, average ETS-associated RSP levels were 92 µg/m^3 when the windows were open and vents closed, 693 µg/m^3 when windows were closed but with the vents open, and 1,195 µg/m^3 when the windows and vents were closed. The outdoor respirable particulate matter concentration during these tests ranged from 4 - 7 µg/m^3. The increase in inside-vehicle concentration over that found outdoors was 13-times greater with the driver's window open/ventilation off, 115-times greater with windows closed/ventilation on, and 300-times greater with windows closed/ventilation off.

Modeling analyses also indicate that particulate matter from ETS can be extremely high in vehicles. Based on field data taken from the literature, Klepeis *et al*. (2001a) used a modeling approach to calculate a mean ETS-particle ($PM_{2.5}$) point-estimate of 2,000 µg/m^3 in vehicles.

Park *et al.* (1998) used a modeling approach based on cigarette emissions and ventilation rates to estimate RSP and formaldehyde levels in vehicles. Levels of ETS constituents in an automobile were estimated under simulated "stop and go" driving conditions. Three different automobiles were tested under a variety of ventilation conditions to calculate air exchange rates. Using ETS emission values obtained from

NRC (1986), it was calculated that RSP and formaldehyde levels could reach peak levels of 2.06 mg/m^3 and 0.13 mg/m^3 (0.11 ppm), respectively. The calculated in-vehicle peaks were projected to occur if a person smoked for 6-minutes (with one window 50% open) while driving at 20 mph, and was stationary for 2-minutes. The formaldehyde concentration would exceed the NIOSH recommended maximum occupational level of 0.1 ppm. Furthermore, the simulations predicted that with the windows closed and with smoking occurring for 6-minutes of driving at 20 mph and 4 minutes of stopping, RSP could peak at 4.36 mg/m^3 and formaldehyde could reach 0.41 mg/m^3 (0.33 ppm). Thus, the researchers concluded that in-vehicle ETS exposures could be quite high when an automobile is stationary.

6. Modeling Studies to Estimate Indoor Air Concentrations of ETS

Models are a useful tool to estimate indoor concentrations of ETS based on source strength (number of cigarettes smoked), air exchange rates, and the volume of a room. The models can be used with population surveys and questionnaire results to determine patterns of cigarette use and exposure to cigarette constituents in different indoor environments. This approach tends to be much less costly and time consuming than direct exposure assessment. One drawback of models is that they have not yet been systematically validated by comparison with actual exposure measurements (Klepeis, 1999). However, the database of exposure-related information (e.g., survey data) that can be incorporated into the models is rapidly expanding, and as a result, models will continue to increase in reliability in predicting exposures under a variety of conditions (Klepeis, 1999).

Nazaroff and Singer (2002) used a material-balance model to estimate exposures of juveniles and non-smoking adults to fifteen Hazardous Air Pollutants (HAPs) contained in ETS. The model incorporated published values on smoking behavior, housing, and demographics, along with new emission measurements. Taken in combination with health-based guidelines, these results suggest that three aldehydes (i.e., acrolein, acetaldehyde and formaldehyde) pose particular long-term risks to non-smokers who live in a household with smokers. The authors estimate that the entire population of non-smokers in the U.S. living with smokers inhales a total of 260 kg of acrolein per year. Inhaled acrolein from all U.S. ambient sources is estimated at about 300 kg/year; thus, indoor ETS alone contributes about as much acrolein to overall human intake as all outdoor sources combined. Similarly, nationwide, the contribution to human inhalation intake of acetaldehyde from ETS in homes is similar to the intake from ambient air. ETS is a strong source for formaldehyde; however, formaldehyde emissions to ambient air from other sources are stronger contributors to human inhalation exposure than ETS in homes.

Activity pattern data can be combined with field measurements to generalize results of small-scale ETS studies to a larger population. Klepeis *et al*. (2001a) conducted such an analysis based on activity data from the National Human Activity Pattern Survey for California (NHAPS-CA) sponsored by the USEPA in the mid-1990's and the CARB California Activity Pattern Survey conducted in the late 1980's. They estimated that from the late-1980's to mid-1990's there was about a 20% overall decrease in the

percentage of Californians exposed to smoking across all locations. However, in vehicles, the decrease over time was estimated to be only one percent. Additionally, the reduction in exposure in residences showed a smaller decrease (9%) than the overall reduction across locations. Klepeis *et al.* (2001a) calculated point-estimates of ETS-particle ($PM_{2.5}$) concentrations using field measurements from several studies. Estimated mean $PM_{2.5}$ concentrations in California, where restrictions prohibit smoking in workplaces and public buildings, are as follows: residence, 30 $\mu g/m^3$; office-factory, 0 $\mu g/m^3$; bar-restaurant, 100 $\mu g/m^3$; other indoor, 5 $\mu g/m^3$; in vehicle, 2,000 $\mu g/m^3$; and outdoor, 0 $\mu g/m^3$.

Burke *et al*. (2001) used the Stochastic Human Exposure and Dose Simulation (SHEDS-PM) model to predict $PM_{2.5}$ exposures in Philadelphia, Pennsylvania. This stochastic model randomly samples different input distributions to estimate population exposure to particulate matter. Burke *et al*. (2001) estimated that one-third of the population under study was exposed to ETS in homes. Investigators further calculated that when the effects of a single smoker were added to the distribution of indoor-residential $PM_{2.5}$ exposure, the exposure of those in the 75^{th} percentile would increase by about 10 $\mu g/m^3$ and those in the 90^{th} percentile by about 28 $\mu g/m^3$. Moreover, the median overall $PM_{2.5}$ exposure for those who were not exposed to ETS in their residences was 16 $\mu g/m^3$ compared to 20 $\mu g/m^3$ for the general population; for the 90^{th} percentile, the values were 32 vs. 59 $\mu g/m^3$, respectively.

Modeled RSP concentrations associated with ETS indicate that 70 - 90 percent of homes with one smoker smoking inside the home, would violate the annual average NAAQS for $PM_{2.5}$ of 15 $\mu g/m^3$ based on smoking alone. A model used by Repace *et al.* (2000) predicted annual average residential ETS-associated RSP levels between 20 and 35 $\mu g/m^3$. Model inputs were based on air exchange rates measured in southern California homes, an estimate of 14 mg RSP emitted per cigarette, and assuming 13 cigarettes were smoked per day in a home. The authors estimate that, for homes with very small volumes and poor ventilation, 10 percent would exceed an annual average of 50 $\mu g/m^3$ and one percent would exceed 85 $\mu g/m^3$ (Repace *et al*., 2000).

Models predict that Californians are exposed to less ETS today than they were in the 1980's. Miller *et al*. (1998) examined exposures of nonsmoking Californians to 17 toxic air contaminants (TACs) known to be present in ETS. These investigators used concentration data for a variety of indoor microenvironments (published between 1980 and 1996) in combination with the CARB's activity pattern survey findings (1991, 1992) to model Californians' ETS exposures for the late 1980's and to make predictions for the late 1990's. Two independent methods were used to simulate indoor concentrations – completely mixed room models, and tracer methods (which utilized published concentrations of ETS-related nicotine and respirable suspended particles). The modeling results for the late 1980's predicted that 52% of nonsmoking adults were exposed to ETS on any given day, and that 58 - 61% of this exposure occurred in residences and workplaces and up to 15% occurred in vehicles. For the 62% of adolescents (ages 12 - 17) who were exposed, 62 - 74% occurred in homes, 8 - 18%

was from transportation, and 4 - 15% was contributed by retail and other indoor environments (e.g., shopping malls, beauty salons, etc.). For the 33% of children (ages 7 - 11) exposed to ETS, 70 - 73% of total exposure was in the home, whereas 9 - 18% occurred in vehicles and 6 - 7% occurred in others' homes.

For the late 1990's, it was predicted that there would be a considerable drop in ETS exposures, where 16 - 19% of adults, 33 - 35% of adolescents, and 21 - 23% of children were expected to be exposed to ETS on any given day (Miller *et al.*, 1998). In these microenvironmental exposure simulations, only residences, transportation, and others' residences were examined due to smoking bans in public venues and workplaces. The results predicted that one's own home would be the major site of exposure for all age groups: 58 - 69% for adults, 58 - 66% for adolescents, and 72 - 83% for children. In California, on average, ETS contributes 4 - 30% of indoor household concentrations of benzene, ethylbenzene, styrene, o-xylene, and m-, p-xylene (Miller *et al*., 1998).

Models indicate residences that allow smoking also have higher PM levels than smoke-free homes. Özkaynak *et al*. (1996) determined that for residences in which smoking was reported, average PM_{10} levels were 30 $\mu g/m^3$ higher than those without smoking. Samples from 31 homes showed that smoking contributed 30% of indoor $PM_{2.5}$ mass and 24% of indoor PM_{10} mass. Investigators used a mass-balance model to estimate a $PM_{2.5}$ source strength for cigarettes of 13.8 $\pm$ 3.6 mg/cigarette. Data for these analyses were collected in Riverside, California, during the PTEAM study.

7. Summary of Indoor and Personal Exposure Concentrations

Restrictions on smoking in California from the late-1980's to mid-1990's in workplaces and in public locations such as restaurants, bars, and gaming clubs have led to a reduction in smoking in indoor environments in California, with commensurate reductions in indoor concentrations of ETS and non-smokers' exposure levels. A number of additional studies published since 1996 have shown that ETS constituents are present at lower concentrations following smoking bans than they were prior to the bans, and that levels can be considerably higher in smoking versus comparable nonsmoking areas. Nonetheless, despite California's smoking bans, high indoor ETS concentrations still can be found in smokers' homes, in private vehicles, and in some non-compliant public establishments. This is of particular concern because when children are present in these locations, they may experience high levels of exposure to ETS.

As shown in Table V-9, the literature reflects the great efficacy of workplace smoking bans in reducing indoor ETS concentrations. Several studies showed levels less than 1.0 $\mu g/m^3$ nicotine where smoking was banned vs. levels that were many times higher where smoking was permitted (Hammond *et al*., 1995; Hammond 1999; Jenkins *et al*., 2000). However, certain workplaces, such as the small percent of free-standing bars that are not yet compliant with California's workplace smoking ban (Weber *et al.*, 2003), would likely have higher elevated levels of ETS, based on measurements made across many studies in such locations (e.g., Hammond, 1999; Siegel and Skeer, 2003).

Results from other recent studies indicate that a ban on smoking also results in lower RSP concentrations in a given environment, similar to the reductions seen with nicotine. For example, $PM_{3.5}$ measurements made at hospitality venues averaged 231 µg/m^3 and ranged from 44 - 686 µg/m^3 before a smoking ban, but ranged from 2.5 – 119 µg/m^3 after implementation of a smoking ban (Repace, 2004). At a church bingo site in northern California, RSP levels were 87 - 348 µg/m^3 above background levels with smoking permitted, and less than 15 µg/m^3 above background levels when smoking was banned (Switzer *et al*. 2001). Generally, levels of RSP also appear to have decreased even in locations where smoking is still allowed, perhaps due to factors such as increased social pressure to avoid smoking indoors and increased attention to ventilation in such establishments. Recent residential RSP measurements in California are limited to two single home studies (Ott *et al*., 2005; Klepeis *et al.,* 1999), in which very short-term, peak room levels ranged up to 350 µg/m^3 where one cigarette was smoked, and up to 5,500 µg/m^3 where three cigarettes were smoked.

Across the years, studies indicate that mean nicotine concentrations have decreased in most indoor environments, although to a somewhat lesser extent in homes than in workplaces and restaurants. Comparison of mean nicotine concentrations from the studies reported in USEPA (1992) and Guerin *et al*. (1992), with data published after 1996, reveals that residential mean nicotine concentrations ranged from 2 – 29 µg/m^3, with the highest measurements over 200 µg/m^3. In newer studies when smokers were present, mean nicotine concentrations ranged from 1 - 6 µg/m^3, with peaks up to 29 µg/m^3. When smoking is permitted at a workplace or public place, nicotine concentrations also appear to be decreasing. In studies conducted before 1997, mean nicotine concentrations in offices and restaurants ranged from 1 to 36 µg/m^3. In a more recent review, Hammond (1999) reported means ranging from 2 - 8 µg/m^3 for these locations, and Jenkins *et al.* (2000) reported office levels up to 16.7 µg/m^3. It appears that as smoking has become a less accepted social behavior, individuals may not be smoking in indoor public locations that permit smoking as much as they did previously.

Very high ETS concentrations have been measured in vehicles when a smoker is present. Levels of RSP ranged from 92 µg/m^3 (with windows open and vent closed) to 1,195 µg/m^3 (windows and vent closed) inside a minivan (Offermann *et al*. 2002). In-vehicle concentrations of RSP also have been estimated to range from 2,060 to 4,360 µg/m^3 under stop-and-go driving conditions (Park *et al*., 1998).

Table V-9 summarizes the results of studies discussed in this section, with emphasis on studies published since 1996. The table is intended to provide a succinct summary of information on indoor concentrations; consequently, it combines data from different averaging times and different size cuts of RSP. The reader is referred to Tables V-6 through V-9 for detailed information about the studies summarized here.

Table V-9

Summary of Indoor Concentrations of Nicotine and RSP[1]

Environment	Nicotine Concentrations µg/m^3		RSP Concentrations µg/m^3	
	Mean	Range	Mean	Range
Homes				
With smoking	~ 0.4 - 6.3	0.02 – 29.2	44 – 125.6	12 - 825
			(Peak values: 1 cig: 160-350; 3 cig: 5,500)	
Non-smoking	0.072 – 0.19	0 - 0.19	20 – 87.8	8 - 100
Overall (including earlier studies)	2 - 29	0 - 292	~ 15 – 700	0.7 – 3,150
Offices/public buildings				
With smoking	0.7 – 21.95	0.2 – 71.5	~ 2.5 – 99	0 – 392
Non-smoking	0.086 – 2.83		2 – 25	2 – 35
Overall (including earlier studies)	< 0.1 - 75	0 - 199	~ 2.5 - 720	0 – 1,088
Vehicles				
Windows open, vent closed	-	-	92	
Windows closed, vent open	-	-	693	
Windows and vent closed	-	-	1,195	
Entertainment Venues				
Restaurants, bars, taverns				
With smoking	2.3 – 40	0 – 70	~ 35 – 986	0 – 1,370
Smoking prohibited			2.5 – 62	0 – 66
Casinos, betting establishments	9.8 – 11	6 - 16	< 20 – 205	
Bowling alleys, billiard halls				
With smoking	10.5 – 13		1 hall: 686	
Non-smoking			1 hall: 119	
Bingo Parlors				
With smoking	76.0		87 – 348[2]	
Smoking prohibited			$\leq$ 15[2]	

1. Averaging times vary across studies. Table V-9 is intended to provide a succinct overview of concentrations of ETS.
2. Mean concentration above outdoor levels; single studies, short averaging times.

E. EXPOSURE ESTIMATION SCENARIOS

1. Introduction

Relative to ETS exposure, current smoking practices and state regulations suggest that children in California can roughly be divided into three exposure groups: (1) children who have little or no exposure to ETS; (2) children with smoking parents or guardians who take some measures to limit their child's exposure; and (3) children highly exposed to ETS through smoking parents, guardians, or peer groups. Likewise, adults generally experience virtually no exposure, regular but limited exposure in public places, or substantial exposure through extensive contact with smokers. However, unlike adults, children are often not able to move away from ETS sources; when with smoking adults, they may not have a choice as to whether they are exposed. Similarly, peer pressure can be a significant factor -- non-smoking teens may feel pressure to "hang out" with their smoking friends or risk being excluded from peer social groups.

These diverse exposures make a population-weighted, statewide exposure estimate complex to calculate and less informative than estimates for illustrative scenarios covering a range of exposures. Accordingly, exposures that might result from several scenarios were estimated to provide an indication of exposure for a range of subgroups of the population over a 24-hour day. Eight exposure scenarios were developed: four for children and four for adults. The scenarios simulate the ETS exposure a non-smoker might experience in different situations ranging from low to high exposure, plus one "maximum exposure" scenario.

2. Background and Calculations

An individual's exposure to an air pollutant in a given environment is dependent on two factors: the concentration of the pollutant in that environment and the amount of time the individual spends in that environment. Exposure is calculated as the product of these two factors, and the result is a time-integrated exposure estimate (National Academy of Sciences (NAS), 1991; Federal Register, 1992). When concentration is measured in $\mu g/m^3$ and time in hours, the unit of exposure is $\mu g\text{-}hr/m^3$. *Total indoor air exposure* is the sum of the environment-specific exposures (time-integrated exposures) associated with time spent indoors. *Total 24-hour exposure (*or *total daily exposure),* is the sum of the different exposures experienced by an individual in the many locations they visit during the 24-hour day, both indoors and outdoors.

Another common method of expressing exposure is to estimate the *time-weighted average exposure concentration*. This is essentially the average of the concentrations experienced by an individual across their 24-hour day, each weighted by the duration of time the individual experienced that concentration, and is expressed in concentration units (i.e., $\mu g/m^3$).

In the exposure scenarios discussed below, exposure estimates are presented for the time-integrated exposure in the major environments visited by the hypothetical person,

their total indoor and outdoor exposures, their total 24-hour exposure, and their time-weighted 24-hour average concentration. Nicotine concentration data recently collected by the CARB in public places (see Section C, above) are used as inputs for outdoor concentrations in the simulated exposures. No measurement data are available to estimate outdoor background levels. In these cases, the exposure is assumed to be zero, reflecting a non-smoking environment. In-home levels of nicotine are drawn from the literature discussed above in Section D. Workplace levels are also based on data discussed in Section D.

3. Scenarios

a. Overview

The following exposure scenarios were used to estimate exposures of specific subgroups of the California population with low to high ETS exposures:

Children

C1– Children's Low Exposure Scenario:
Child (8 years old) living in a non-smoking household exposed to nicotine while playing outdoors in an area that is adjacent to a neighboring business' smoking area.

C2 – Children's Medium Exposure Scenario:
Child (8 years old) living in a smoking household with an average number of cigarettes smoked indoors, and also exposed to nicotine while playing outdoors in an area that is adjacent to a neighboring business' smoking area.

C3 – Children's High Exposure Scenario:
Child (8 years old) living in a smoking household with a somewhat high number of cigarettes smoked indoors, and also exposed to nicotine while in the car and at an amusement park.

C4 – Children's Maximal Exposure Scenario:
Child (8 years old) living in a smoking household with a high number of cigarettes smoked indoors, a high number of cigarettes smoked in the car while in transit, and experiencing the highest outdoor levels measured in CARB's outdoor monitoring tests.

College Student

S1 – College Student Low Exposure Scenario:
Non-smoking college student living in an apartment with a non-smoking roommate who visits a campus designated smoking area.

S2 – College Student High Exposure Scenario:
Non-smoking college student living in an apartment with smoking roommates, who visits the campus designated smoking areas, and travels to the airport on a given day when smoking occurs both in the car and at the airport.

Traveler

T1 – Business Traveler's Low Exposure Scenario:
Non-smoking business traveler who is exposed to nicotine while in line at the Automatic Teller Machine (ATM) at the bank, waiting outside the airport terminal, and dining at an outdoor restaurant located next to an office building smoking area.

T2 – Business Traveler's High Exposure Scenario:
Non-smoking business traveler who is exposed to the same exposure scenarios as in T1, except that he/she spends the first hour of the business lunch with a client at a free-standing bar that is non-compliant with California's workplace smoking ban, and is also exposed in the car.

b. Assumptions and Scenario Results

The specific average nicotine concentrations used for outdoor locations in the exposure estimation scenarios are indicated in Table V-10. These are averages calculated from CARB's outdoor monitoring scenarios (Section C). The concentrations used for indoor locations in the scenarios are taken from the literature, as indicated in Table V-9, and are specified in the footnotes for each scenario below.

Table V-10

Summary of Measured Outdoor Nicotine Average Concentrations Used in the Estimation of Scenario-Based Exposure

Outside Location	Nicotine Concentration Mean of 1-hour Averages ($\mu g/m^3$)
Airport Terminal	0.72
College Smoking Area	0.051
Local Government Office Complex	0.097
Public Office Building Complex	0.19
Amusement Park Smoking Area	2.4

Estimates of mean residential indoor nicotine concentrations used in the scenarios are 0, 3.0, 6.0, and 29.2 $\mu g/m^3$ for low, medium, high, and maximally exposed scenarios, respectively, based primarily on measurements taken by Glasgow *et al*. (1998) and Hammond, 1999 (see Tables V-6 and V-9).

Children's Scenarios and Assumptions

The assumptions for scenarios C1 and C2, children's low and medium exposures, are:

1. Indoor time at home (14 hours) includes all time spent in the home – sleeping, eating, watching television, etc.

2. Time spent indoors at school is 5 hours per day. This includes all classroom/study time indoors.

3. About 1 hour of the day is spent indoors elsewhere (other than at home and school), such as at an after-school care facility.

4. Outdoor time at home (1 hour) primarily includes playing in the yard before and after school.

5. Outdoor time at school (2 hours) includes morning arrival time (15 minutes), recess (15 minutes), lunch time (30 minutes), physical education class (45 minutes), afternoon pick-up time (15 minutes).

6. Outdoor time at other places (1 hour) is assumed to be playing outdoors at an after-school care facility or an activity at some other location adjacent to a neighboring business's smoking area.

7. Nicotine concentration for neighboring business smoking area: the mean measured 1-hour average nicotine concentration from the Public Office Building Complex smoking area is used for the nicotine concentration in the neighboring business smoking area. Nicotine concentrations were measured at a Government Office Complex smoking area as well as at a Public Office Building Complex smoking area. The mean 1-hour nicotine concentration was higher at the Public Office building complex. Because the overall objective of this exercise is to characterize the exposures of certain subgroups of the population who are exposed, and because office buildings are more predominant than government buildings, the Public Office Building Complex measurement was selected for use as a surrogate for levels children might be exposed to when playing near a smoking area immediately adjacent to their play area.

8. Nicotine concentration for smoking outdoors at home: because there are no data for outdoor ETS concentrations at home, the mean measured 1-hour average nicotine concentration in a designated smoking area at a college is used as a surrogate for the outdoor nicotine concentration at home. The college area concentration resulted from the smoking of two to six cigarettes per hour, and thus is reasonable to use as a surrogate for outdoor exposures at home with a smoker present.

9. Nicotine concentration inside the home is assumed to be 3.0 µg/m^3. Glasgow *et al.* (1998) reported homes with 50 or fewer cigarettes smoked per week had indoor nicotine levels less than 3.0 µg/m^3. Thus, it is estimated that a home with moderate smoking (up to 50 cigarettes per week) would have levels up to 3.0 µg/m^3.

C1– Children's Low Exposure Scenario: child (8 years old) living in a non-smoking household exposed to nicotine while playing outdoors in an area that is adjacent to a neighboring business' smoking area.

SCENARIO C1: CHILDREN'S LOW EXPOSURE						
Environment	ETS Present	Time Spent in Environment (hours)	Nicotine Concentration in Environment (µg/m^3)	24-hour Time-integrated Exposure in Environment (µg-hr/m^3)	Percent of Total Exposure (%)	Time-weighted Average Concentration (a) (µg/m^3)
Indoor						
Home	No	14	0	0		
School	No	5	0	0		
Other	No	1	0	0		
Total Indoor		**20**		**0**	**0**	**0**
Outdoor						
Home	No	1	0	0		
School	No	2	0	0		
Other	Yes	1	0.19 (b)	0.19		
Total Outdoor		**4**		**0.19**	**100**	**0.048**
Total =		**24**		**0.19**	**100**	**0.008**

a) Time-weighted average concentration (TWAC) is the average of the concentrations of a substance to which a person is exposed over a period of time, such as a 24-hour day. For example, if a person is exposed to *x* µg/m^3 for 20 hours and *y* µg/m^3 for 4 hours, the 24-hour TWAC is calculated as (*x* x 20 + *y* x 4) / 24.

b) The Public Office Building Complex mean 1-hour average is used here, as explained in the text.

Results of this scenario illustrate that young children in non-smoking households would likely have very low exposures, and virtually all of that exposure would result from outdoor smoking.

C2 – Children's Medium Exposure: child (8 years old) living in a smoking household with an average amount of smoking indoors, also exposed to nicotine while playing outdoors in an area that is adjacent to a neighboring business' smoking area.

SCENARIO C2: CHILDREN'S MEDIUM EXPOSURE						
Environment	ETS Present	Time Spent in Environment (hours)	Nicotine Concentration in Environment ($\mu g/m^3$)	24-hour Time-integrated Exposure in Environment ($\mu g\text{-}hr/m^3$)	Percent of Total Exposure (%)	Time-weighted Average Concentration ($\mu g/m^3$)
Indoor						
Home	Yes	14	3.0 (a)	42.0		
School	No	5	0	0		
Other	No	1	0	0		
Total Indoor		**20**		**42**	**99.43**	**2.1**
Outdoor						
Home	Yes	1	0.051 (b)	0.051		
School	No	2	0	0		
Other	Yes	1	0.19 (c)	0.19		
Total Outdoor		**4**		**0.24**	**0.57**	**0.06**
Total =		**24**		**42.24** (d)	**100**	**1.8**

a) Mid-value, Glasgow *et al*. (1998), see Table 5.
b) The mean 1-hour average concentration in a College Outdoor Smoking Area, used as a surrogate for parents smoking in the yard where the child is playing. The number of cigarettes smoked per hour in the College Outdoor Smoking Area (two to six), serves as a reasonable surrogate for levels when smoking occurs outdoors at the home.
c) The mean 1-hour average concentration in a Public Office Building Complex outdoor smoking area is used.
d) Totals may not add up exactly due to rounding.

Results of the children's medium scenario illustrate the impact of living with a smoking parent. The majority of the child's exposure stems from their time spent indoors at home.

The assumptions for Scenario C3, children's high exposure, are:

1. The value of 6.0 $\mu g/m^3$ is used for the indoor home concentration in this scenario, based on the results of Glasgow *et al.* (1998) showing an average of 5.4 $\mu g/m^3$ and a maximum of 29.2 $\mu g/m^3$ in homes, and Hammond (1999), who reported indoor means in smokers' homes ranging from 1.5 - 5.8 $\mu g/m^3$.

2. The child travels for two hours each way to and from an amusement park in a car with smoking parents. Measurements of RSP levels in a car with smoking and some ventilation were used to derive an estimated nicotine concentration in the vehicle. The average RSP concentrations in a vehicle during smoking was 92 µg/m^3 with windows open and ventilation off (Offermann *et al*., 2002). In the same study, the average RSP concentration in a vehicle during smoking was 693 µg/m^3 with windows closed and ventilation on. The average of these two values is 392.5 µg/m^3 of RSP. As stated elsewhere in this report, nicotine concentrations are approximately 8 percent of the RSP concentrations in ETS. Eight percent of 392.5 µg/m^3 is 31.4 µg/m^3, the value used to represent a medium nicotine concentration in a car with smokers. The concentration data and calculations used here provide reasonable estimates, which may be an underestimate, in consideration of the RSP and nicotine data in Badre *et al*. (1978). Their findings were many times higher than the measured levels in Offermann *et al.* (2002), and modeled estimates developed by Klepeis *et al.* (2001) and Park *et al.* (1998), discussed in an earlier section.

3. The child spends the times indoors and outdoors at the amusement park as indicated in Scenario C3 below, with a total of two of the outdoor hours spent at smoking areas across the day when the parents took smoking breaks.

   C3 – Children's High Exposure Scenario: child lives in a smoking household with a somewhat high level of smoking indoors is also exposed to nicotine while in the car and at the amusement park.

SCENARIO C3: CHILDREN'S HIGH EXPOSURE						
Environment	ETS Present	Time Spent in Environment (hours)	Nicotine Concentration in Environment ($\mu g/m^3$)	24-hour Time-integrated Exposure in Environment ($\mu g\text{-}hr/m^3$)	Percent of Total Exposure (%)	Time-weighted Average Concentration ($\mu g/m^3$)
Indoor						
Home	Yes	8	6.0 (a)	48.0		
Theme Park	No	2	0	0		
Other	No	2	0	0		
Transit- in car	Yes	4	31.4 (b)	125.6		
Total Indoor + Car		**16**		**173.6**	**97.3**	**10.9**
Outdoor						
Home	Yes	0.5	0.051 (c)	0.026		
Theme Park – Smoking Area	Yes	2	2.4 (d)	4.80		
Theme Park – Non Smoking Areas	No	5.5	0	0		
Total Outdoor		**8**		**4.826**	**2.70**	**0.60**
Total		**24**		178.4 (e)	**100**	**7.4**

a) Glasgow *et al.* (1998), and Hammond (1999). See Table V-6.
b) Nicotine concentration inside a car with smokers is derived from RSP data (Offermann *et al.*, 2002). Nicotine concentration is assumed to be 8% of RSP concentration. Full calculation is presented in the assumptions for Scenario C3.
c) The mean 1-hour average concentration in a College Outdoor Smoking Area is used as a surrogate for parents smoking in the yard where the child is playing.
d) The mean 1-hour average concentration measured in Amusement Park Smoking Areas.
e) Totals may not add up exactly due to rounding to significant digits.

This scenario illustrates the high exposures that would be experienced by a child living in a heavy smoking household with parents or guardians who also smoke in the car. The child's exposure is further increased when the parents visit the outdoor smoking area at the amusement park for smoking breaks. This scenario illustrates the substantial exposure, 4.826 $\mu g\text{-}hr/m^3$, which can occur outdoors at smoking areas visited by many smokers.

C4- Children's Maximally Exposed Scenario

Child (8 years old) living in a smoking household with a high number of cigarettes smoked indoors, a high number of cigarettes smoked in the car while in transit, and experiencing the highest outdoor levels measured in CARB's outdoor monitoring tests. This scenario may represent the highest possible 99% of an exposed subpopulation.

The specific assumptions for Scenario C4, children's maximally exposed scenario, are:

1. The child makes a day trip to an amusement park with his smoking parents. The time spent in each microenvironment is identical to that in Scenario C3. A total of two of the outdoor hours are spent at smoking areas across the day when the parents take smoking breaks.

2. The indoor concentrations are elevated relative to those in C3. The home concentration is the maximum measured in Glasgow *et al*. (1998), 29.2 µg/m^3. The concentration inside the car is based on a RSP measurement in a car during smoking with windows closed and ventilation on (Offermann *et al*., 2002). The RSP concentration under those conditions was 693 µg/m^3. As stated elsewhere in this report, nicotine concentrations are approximately 8% of RSP concentrations: 8% of 693 is 55.4 µg/m^3.

3. Outdoor concentrations have been increased to the highest level measured in the CARB monitoring study at specified outdoor smoking areas.

SCENARIO C4: CHILDREN'S MAXIMALLY EXPOSED SCENARIO

Environment	ETS Present	Time Spent in Environment (hours)	Nicotine Concentration in Environment (µg/m^3)	24-hour Time-integrated Exposure in Environment (µg-hr/m^3)	Percent of Total Exposure (%)	Time-weighted Average Concen-tration (µg/m^3)
Indoor						
Home	Yes	8	29.2 (a)	233.6		
Theme Park	No	2	0	0		
Other	No	2	0	0		
Transit- in car	Yes	4	55.4 (b)	221.6		
Total Indoor + Car		**16**		**455.2**	**98.0**	**28.5**
Outdoor						
Home	Yes	0.5	0.150 (c)	0.075		
Theme Park – Smoking Area	Yes	2	4.6 (d)	9.2		
Theme Park – Non Smoking Areas	No	5.5	0	0		
Total Outdoor		**8**		**9.3**	**2.0**	**1.2**
Total		**24**		**464.5** (e)	**100**	**19.4**

a) Glasgow *et al.* (1998), maximum value, see Table V-6.
b) Nicotine concentrations inside a car with smokers are derived from RSP data (Offermann *et al.*, 2002). Nicotine concentration is assumed to be 8% of RSP concentration. The full calculation is presented in the assumptions for Scenario C4.
c) The highest 1-hour average concentration in a College Outdoor Smoking Area is used as a surrogate for parents smoking in the yard where the child is playing.
d) The highest 1-hour average concentration measured in Amusement Park Smoking Areas.
e) Totals may not add exactly due to rounding to significant figures.

This exposure scenario represents the upper limits for children's exposure. It illustrates an extremely high exposure that would be experienced by a child living in a heavy smoking household with parents or guardians who also smoke in the car. The child's exposure is further increased when the parents visit the outdoor smoking area at the amusement park for smoking breaks. The outdoor exposure in this scenario is approximately that of Scenario C3 and has the effect of increasing both indoor and outdoor exposure.

College Student Scenario Assumptions

Two scenarios with college students were developed:

1. In Scenario 1, the student is a non-smoker, lives in a non-smoking household, and is only exposed to ETS when talking with friends at an outdoor area set aside for smokers.

2. In Scenario 2, the student lives in an apartment with two smoking roommates, talks with friends at an outdoor smoking area on campus, and also makes a trip to the airport, with a roommate smoking in the car. He/she waits at the airport near an outdoor smoking area to pick up a friend. This individual also spends some time outdoors at home with a smoking roommate and attends an outdoor party where ETS concentrations are similar to those at the airport.

3. Like the children's scenarios, the College Smoking Area input data are used as surrogates for the "outdoors at home" levels.

4. Outdoors at the airport and outdoors at the college smoking area are taken directly from CARB's measured averages for those areas (see Section B above).

5. In Scenario 2, indoor home levels are assumed to be 6 µg/m^3**,** the higher mean exposure level taken from Glasgow *et al*. (1998) as discussed elsewhere in this chapter, because of the smaller volume of an apartment and the higher ETS concentrations that would be expected.

S1 – College Student Low Exposure Scenario: non-smoking college student living in an apartment with a non-smoking roommate and visits the campus designated smoking areas.

SCENARIO S1: COLLEGE STUDENT LOW EXPOSURE						
Environment	ETS Present	Time Spent in Environment (hours)	Nicotine Concentration in Environment ($\mu g/m^3$)	24-hour Time-integrated Exposure in Environment ($\mu g\text{-}hr/m^3$)	Percent of Total Exposure (%)	Time-weighted Average Concentration ($\mu g/m^3$)
Indoor						
Home	No	8	0	0		
College	No	8	0	0		
Other	No	2	0	0		
Total Indoor		**18**		**0**	**0**	**0**
Outdoor						
Home	No	1	0	0		
College	Yes	2	0.051 (a)	0.102		
Other	No	3	0	0		
Total Outdoor		**6**		**0.10**	**100**	**0.017**
Total =		**24**		**0.10**	**100**	**0.0043**

(a) The mean 1-hour average concentration in a College Outdoor Smoking Area.

Results from the College Student Low Exposure Scenario again illustrate that non-smokers living in non-smoking households have generally very low exposures, and that whatever exposure they experience is likely to occur from outdoor smoking.

S2 – College Student High Exposure Scenario: Non-smoking college student lives in an apartment with smoking roommates. Non-smoker visits the campus designated smoking areas, and goes to the airport in car with a smoker. Exposure while in the car is similar to that used and described in Scenario C3. The college student then attends an outdoor party with ETS levels similar to that at the airport.

SCENARIO S2: COLLEGE STUDENT HIGH EXPOSURE						
Environment	ETS Present	Time Spent in Environment (hours)	Nicotine Concentration in Environment ($\mu g/m^3$)	24-hour Time-integrated Exposure in Environment (μg-hr/m^3)	Percent of Total Exposure (%)	Time-weighted Average Concentration ($\mu g/m^3$)
Indoor						
Home	Yes	8	6.0 (a)	48		
College	No	7	0	0		
Other	No	1	0	0		
Transit-in car	Yes	1	31.4 (b)	31.4		
Total Indoor		**17**		**79.4**	**96.32**	**4.671**
Outdoor						
Home	Yes	1	0.051 (c)	0.051		
College	Yes	2	0.051 (d)	0.102		
Airport	Yes	0.5	0.72 (e)	0.360		
Outdoor Party	Yes	3.5	0.72 (f)	2.52		
Total Outdoor		**7**		**3.03**	**3.68**	**0.433**
Total =		**24**		**82.43** (g)	**100**	**3.435**

a) Indoor Home: Assume high-end data for the home from Glasgow *et al.* (1998) and Hammond (1999). See Table V-6.
b) Nicotine concentration inside a car with smokers is derived from RSP data (Offermann *et al*., 2002). Nicotine concentration is assumed to be 8% of RSP concentration. The full calculation is presented in the assumptions for Scenario C4. No nicotine data in cars is available.
c) Outdoor Home: Use the same input data as the College Smoking Area.
d) College Smoking Area: Average 1-hour (two to six cigarettes per hour).
e) Outdoor Airport: Student is meeting someone at the airport terminal at a specific outdoor location near a designated smoking area and waits for about 30-minutes.
f) Outdoor Party: Student attends a party with smoking levels and exposure comparable to those at the airport.
g) Totals may not add up exactly due to rounding to significant digits.

This scenario again illustrates the elevated exposures of those living with smoking household members. It also illustrates that adults can experience exposure in several outdoor locations in a day, depending on their specific activity patterns.

Business Traveler's Scenario Assumptions:

1. Non-smoking business traveler has a one-day trip by airline from northern to southern California, for a several-hour business meeting.

2. He/she visits the ATM for cash before driving to the airport, must wait outside the terminal near smokers before getting into the terminal, and travels to southern California.

4. During the meeting, he/she has a business lunch with a business client, sitting outdoors very near the smoking area of a nearby office building. Upon returning

to the airport to fly home, he/she again is outdoors near smokers for a time before getting inside the airport.

5. In Scenario T2, the business traveler is also exposed to smoke in the car going to and from the airport (such as if a smoking co-worker gave him a ride), and also is exposed during time spent in a non-compliant bar.

T1 – Business traveler's low exposure scenario: the non-smoking business traveler is exposed to nicotine while in line at the Automatic Teller Machine (ATM) at the bank, while waiting outside the airport terminal, and dining at an outdoor restaurant located next to an office building smoking area.

SCENARIO T1: BUSINESS TRAVELER LOW EXPOSURE						
Environment	ETS Present	Time Spent in Environment (hours)	Nicotine Concentration in Environment ($\mu g/m^3$)	24-hour Time-integrated Exposure in Environment (μg-hr/m^3)	Percent of Total Exposure (%)	Time-weighted Average Concentration ($\mu g/m^3$)
Indoor						
Home	No	9.5	0	0		
Airport	No	1.3	0	0		
Other-Bus. Meeting	No	5	0	0		
Other-inside plane	No	3	0	0		
Transit-in car	No	1	0	0		
Total Indoor + Car		**19.8**		**0**	**0**	**0**
SCENARIO T1: BUSINESS TRAVELER LOW EXPOSURE (CONT)						
Outdoor						
Home	No	0.5	0	0		
ATM-Bank	Yes	0.2	0.097 (a)	0.019		
Airport	Yes	0.5	0.72 (b)	0.36		
Dining	Yes	2	0.19 (c)	0.38		
Other	No	1	0	0		
Total Outdoor		**4.2**		**.759**	**100**	**0.181**
Total =		**24**		**.759** (d)	**100**	**0.032**

a) The mean 1-hour average concentration in a Local Government Office Building Complex Outdoor Smoking Area is used as input, assuming a low number of cigarettes smoked near the ATM.
b) The mean 1-hour average Airport Terminal concentration.
c) The mean 1-hour average Public Office Building Complex Outdoor Smoking Area.
d) Totals may not add up exactly due to rounding to significant figures.

The results for this business traveler scenario indicate that exposure during the day for a non-smoking traveler would be low, and would occur completely outdoors.

T2 – Business traveler's high exposure scenario: In this scenario, the traveler's day is similar to Scenario T1, except that she/he rides in a car with a co-worker who smokes, spends the first hour of the business lunch with the client at a free-standing bar that is non-compliant with California's workplace smoking ban, followed by an hour dining in the outdoor section of a restaurant very near the smoking area of a nearby office building. It is assumed that the co-worker smokes most of the time while in the car. As in previous scenarios, measurements of RSP levels in a car with smoking and some ventilation were used to derive an estimated nicotine concentration in the vehicle. The average RSP concentration in a vehicle during smoking was 92 $\mu g/m^3$ with windows open and ventilation off (Offermann *et al*., 2002). In the same study, the average RSP concentration in a vehicle during smoking was 693 $\mu g/m^3$ with windows closed and ventilation on. The average of these two values is 392.5 $\mu g/m^3$ of RSP. As stated elsewhere in this report, nicotine concentrations are approximately 8 percent of ETS RSP concentrations. Eight percent of 392.5 $\mu g/m^3$ is 31.4 $\mu g/m^3$, the value used to represent moderate nicotine concentration in a car with smokers. The concentration data and calculations used here provide reasonable estimates, which may be an underestimate, in consideration of the RSP and nicotine data in Badre *et al*. (1978). Their findings were many times higher than the measured levels in Offermann *et al.* (2002), and modeled estimates developed by Klepeis *et al.* (2001) and Park *et al.* (1998).

SCENARIO T2: BUSINESS TRAVELER HIGH EXPOSURE - BAR						
Environment	ETS Present	Time Spent in Environment (hours)	Nicotine Concentration in Environment ($\mu g/m^3$)	24-hour Time-integrated Exposure in Environment ($\mu g\text{-}hr/m^3$)	Percent of Total Exposure (%)	Time-weighted Average Concentration ($\mu g/m^3$)
Indoor						
Home	No	9	0	0		
Airport	No	1.3	0	0		
Other-Bus. meeting	No	5	0	0		
Other-inside plane	No	3	0	0		
Visit non-compliant bar	Yes	1	31.1 (a)	31.1		
Transit-in car	Yes	1	31.4(b)	31.4		
Total Indoor + Car		**20.3**		**62.5**	**98.5**	**3.08**
Outdoor						
Home	No	0.5	0	0		
ATM-Bank	Yes	0.2	0.097 (c)	0.019		
Airport	Yes	1	0.72 (d)	0.72		
Dining	Yes	1	0.19 (e)	0.19		
Other	No	1	0	0		
Total Outdoor		**3.7**		**0.929**	**1.5**	**0.25**
Total =		**24**		**63.43** (f)	**100**	**2.64**

a) From Seigel and Skeer (2003). The mean of average nicotine values reported in individual U.S. studies weighted by the number of establishments sampled in each study. This mean is considered to be an upper bound for an assumed level of nicotine for bars in this exposure scenario. Because the estimate is based in part on data obtained from older studies of bars where smoking was allowed, and smoke would have been more concentrated than it would be in non-compliant California bars.

b) Nicotine concentration inside a car with a smoker is derived from RSP data (Offermann *et al.*, 2002). Nicotine concentration is assumed to be 8% of RSP concentration. The full calculation is presented in the assumptions for Scenario T2. No nicotine data in cars is available.

c) The mean 1-hour average concentration in a Local Government Office Building Complex Outdoor Smoking Area is used as input, assuming a low number of cigarettes smoked near the ATM.

d) The mean 1-hour average Airport Terminal concentration.

e) The mean 1-hour average Public Office Building Complex Outdoor Smoking Area.

f) Totals may not add up exactly due to rounding to significant figures.

The results for the business traveler spending time in a non-compliant bar indicate that the major exposure of this nonsmoking traveler would result from spending time in the non-compliant bar and riding with a smoker in a car. This scenario illustrates that non-smoking business travelers travelling with smoking co-workers or working with smoking clients would likely be exposed to higher levels of ETS, on average, to the extent that they visit smoking environments that they would not otherwise visit.

4. Summary and Conclusions

Table V-11 below summarizes the results of all of the exposure scenario calculations. The total 24-hour air exposure for individuals in each scenario is presented, along with the 24-hour average air concentration that such an exposure represents. The total indoor exposure for each scenario and the percent of the indoor exposure to the total exposure (indoor plus outdoor) is also provided.

Table V-11

Summary of Nicotine Exposure Scenario Results[a]

Exposure Scenario	Total 24-hour Air Exposure (time-integrated exposure) (µg-hr/m^3)	Total Indoor Exposure (µg-hr/m^3)	Percent Contribution of Indoor Exposure to the Total Exposure (%)	Average 24-hour Air Concentration (time-weighted exposure) (µg/m^3)
C1 – Children Low	0.19	0	0	0.008
C2 – Children Medium	42	42	99	1.8
C3 – Children High	178	174	97	7.4
C4 – Children Maximally Exposed	465	455	98	19.4
S1 – College Student Low	0.10	0	0	0.0043
S2 – College Student High	82	79	96	3.44
T1 – Business Traveler Low	0.76	0	0	0.032
T2 – Business Traveler High – (Bar)	63	62.5	98.5	2.64

a) Rounded results from previous tables.

The results of the scenario calculations show a wide range of possible exposures in subgroups of the population for which exposure scenarios were developed. For individuals living in non-smoking homes and having only very brief encounters with ETS, exposures are very low, less than 1 µg-hr/m^3. Some individuals in the population would be expected to have near-zero exposures, if their activity patterns do not bring

them near smokers other than on rare occasions. The primary, and often the only, exposure for those individuals occurs outdoors in locations over which the individual typically has little control. For non-smokers whose work or other activities bring them into contact with outdoor smokers regularly, 100% of their exposure can be attributable to proximity to outdoor smoking.

For those living in homes with smokers, indoor and in-vehicle exposures are predominant and high, as would be expected, ranging up to 455 µg-hr/m^3, and potentially even higher in the actual population. These high exposures are due in part to the time spent in those locations as well as to the number of cigarettes typically smoked there and the trapping effect of enclosed environments such as apartments and cars. Such exposures are especially of concern for young children, both because they are likely to recur daily and because of the potential additional physiological sensitivity of developing children.

Nonsmokers who visit non-compliant bars with smoking business associates, clients, or friends likely experience relatively high exposures to ETS. However, compliance with California's workplace restrictions in free-standing bars is increasing by almost eight percent a year (Weber *et al*., 2003), so within a few years, it is likely that nearly all bars in California will be compliant.

Conclusions

Based on the available literature, trends in California smoking and exposure, and the scenarios developed above, one can conclude that:

- Exposures to ETS are highly variable in California.

- Outdoor smoking appears to be the primary source of exposure for individuals who live in non-smoking homes in California, based on the prohibition of smoking in indoor workplaces and illustrated by the scenarios above.

- Outdoor smoking can contribute from near zero to 100% of people's exposures to ETS. However, the outdoor time-weighted average concentrations of ETS are low (maximum 24-hour average = 1.2 µg/m^3 for all scenarios) compared to indoor or in-car exposures.

- Indoor exposures contribute most to exposure for those living in homes with smokers. Children living with smokers are especially likely to be impacted, since they spend a large portion of their time inside the home and in other locations where the smoking parent or guardian spend time, such as outdoors at home and in the family car.

- Concentrations in cars with smokers can be very high, and so can contribute the most to the exposure of those who regularly ride in cars with smokers.

F. BIOLOGICAL MARKERS OF EXPOSURE TO ETS

1. Introduction

This section addresses the use of biological markers (biomarkers) to measure ETS exposure. Information from the OEHHA report (OEHHA, 1997): *Health Effects of Exposure to Environmental Tobacco Smoke* was used as a starting point for the development of this section. The OEHHA report presented a great deal of information on the philosophy behind and rationale for using biologic markers of tobacco smoke exposure. Concentrations in physiologic fluids of adults, comparisons of levels in smokers, ETS-exposed non-smokers, and unexposed non-smokers, and concentrations in physiologic fluids of infants and children, in breast milk and amniotic fluid were described. The use of levels of exhaled carbon monoxide and blood levels of carboxyhemoglobin, as well as thiocyanate levels in blood, urine and saliva as biomarkers of ETS exposure were also addressed, as were DNA and protein adducts and other approaches of assessing tobacco smoke exposure. This updated section generally presents a combination of relevant older data and new studies as a single coherent document rather than separating the findings of the previous report. Where appropriate, discussions on previous findings are either included within sections or presented in the opening paragraphs. The major updates to information presented in the 1997 OEHHA report are highlighted below.

New studies presented in the update to this section strongly reinforce the findings in the 1997 OEHHA report regarding physiologic fluid levels of cotinine in adults, as well as the strong dose-response relationship between levels of this metabolite and ETS exposure. The results of recent large-scale studies provide useful correlations between daily cigarette exposures and cotinine levels. Similar studies using personal exposure monitors provide a link to average ETS atmospheric concentration and physiologic cotinine levels. Improved laboratory techniques are described with levels of detection sufficiently low that non-ETS exposed non-smokers can be distinguished based on cotinine levels from nonsmokers with low ETS exposure levels. Most studies presented in the 1997 OEHHA report did not have low enough levels of detection to do this. New studies also reinforce previous findings regarding appropriate cutoff cotinine levels to distinguish between smokers and non-smokers.

New to the biomarkers discussion is the use of hair nicotine levels as a useful biomarker of exposure. This science is still in its infancy, but results thus far indicate that hair nicotine is more useful in characterizing long-term exposure to ETS than cotinine.

The children studies presented in the 1997 OEHHA report address cotinine and nicotine levels in physiologic fluids of infants and children as well as in amniotic fluid and breast milk. The update reinforces the previous findings, while adding new light on half-lives of cotinine both in normal children and asthmatics. Recent studies also better characterize exposure patterns in infants and children based upon cotinine levels. New with this update is information on other biomarkers of ETS exposure in children, including carcinogenic nitroso-compounds, thiocyanate and protein adducts.

Recent work using other biomarkers such as thiocyanate reinforced the lack of specificity found in the 1997 OEHHA report. DNA and protein adducts of tobacco specific metabolites are generally not useful in distinguishing between non-smokers exposed to ETS and those who are not, a finding that is also consistent with the 1997 OEHHA report.

Introductory subsections of this section are basically unchanged from the 1997 OEHHA report. These subsections describe the basic science behind the use of biomarkers, and little has changed in this area.

2. Introduction to Biomarkers of ETS Exposure

Measured biological parameters, such as the concentrations of metabolites, signaling compounds or tissue constituents, may be used as indices of either the extent of exposure to an external stimulus, such as a toxic environmental contaminant (biomarkers of exposure), or of the extent of a specific response to such as stimulus, including biochemical or histological damage, altered physiology, *etc.* (biomarkers of effect). The current section examines the utility of biomarkers specifically to assess the extent of exposure to ETS. This can be assessed directly by the analysis of physiologic fluids (urine, saliva, and serum) or human hair for tobacco smoke constituents or their metabolites. Nicotine, cotinine, thiocyanate, carboxyhemoglobin, hydroxyproline, N-nitrosoproline, aromatic amines, and certain protein and DNA adducts have been used as indicators of exposure to tobacco smoke. With the exception of the DNA adduct measurements, which may for some purposes be regarded as an early-stage biomarker of adverse genotoxic effects, these biomarkers do not indicate the presence of, or susceptibility to, disease due to exposure to tobacco smoke. Rather, these biomarkers simply reflect that the individual has been exposed to tobacco smoke. While few of the biomarkers listed above are entirely specific to tobacco smoke, when other known sources are accounted for, the presence of these marker compounds in tissues or body fluids can be attributed to smoke exposure. The appropriateness of a given biomarker depends on the nature of the study and the type of exposure being assessed (e.g. recent or long-term).

The relationship between a biomarker and exposure is complex, and varies as a function of both environmental and physiologic factors. The degree of exposure is a function of the time an individual spends in each setting and the air concentration of tobacco smoke constituents in that environment. Factors affecting air concentrations include smoking intensity, room size, room ventilation, and the furnishings and construction materials of the room. For a given air concentration, several factors will affect an individual's intake, such as gender, age, weight, and activity level (and corresponding inhalation rate) at the time of exposure. In addition, individual differences in uptake, distribution, and metabolism will affect the concentration of the indicator compound in tissues or body fluids. Racial differences in metabolism may also affect the biomarker concentration. Caraballo *et al.* (1998) review of the NHANES III data, found among smokers that African Americans had substantially higher cotinine concentrations than did whites or Hispanics at all levels of cigarette consumption. While the presence of a biomarker indicates that tobacco smoke exposure has occurred, and

a given individual will show a positive association between ETS exposure and biomarker levels, biomarker concentrations across individuals correlate only approximately with the amount of exposure to tobacco smoke. The atmospheric lifetime of a biomarker must also be considered when designing a study that attempts to characterize long-term exposure.

a) Biomarkers: Nicotine and Cotinine

i) Nicotine and Cotinine: General methodological issues

Cotinine, the major metabolite of nicotine, has emerged over the past 20 years as the biomarker of choice for most field exposure studies and for validation of smoking status. The update to the 1997 OEHHA report primarily focuses on the new data from large epidemiologic studies relating cotinine in body fluids to levels of second hand smoke exposure. Many small scale studies linking cotinine levels to ETS exposure have been done over the last decade that are not mentioned simply because the results echo those of the larger studies.

In general, the presence of nicotine or its metabolites in physiological fluids can be attributed to exposure to tobacco smoke. The few exceptions include occupational exposure to tobacco leaves and nicotine products, use of smokeless tobacco products, chewing of nicotine gum, and use of nicotine patches or other smoking cessation aides. Low levels of nicotine have been found in tea and in edible solanaceous plants including eggplant, green pepper and tomato, but these sources are not considered to be significant in comparison to tobacco sources (OEHHA, 1997; Tunstall-Pedoe *et al*., 1991; Pirkle *et al*., 1996).

As biomarkers of exposure, nicotine and/or cotinine concentrations are typically measured in blood, saliva or urine. Quantitative assessment of exposure has been done using all three fluids. Recent work by Bernert *et al*. (2000), using sensitive laboratory techniques, indicate that salivary and serum cotinine levels are approximately equal, where it had previously been felt that the salivary glands tend to concentrate cotinine over serum by 20 – 40% (Curvall *et al*., 1990). The kidney concentrates cotinine, with urinary levels increased by a factor of five or six over serum (OEHHA, 1997; Benowitz, 1996; Peterson, 1997). Investigators over the last decade have also used nicotine in human hair as a biomarker for tobacco smoke exposure.

Urinary cotinine excretion is variable across and within individuals, depending on renal function, urinary flow rate, and urinary pH (OEHHA, 1997). Urinary results may be expressed as nanograms of cotinine per milligram of creatinine, to correct in part, for differences in dilution effects. Because the amount of endogenous creatinine produced is a function of muscle mass, and hence, age and sex, individual excretion rates of creatinine are also variable. In particular, cotinine:creatinine ratios may not be appropriate for comparisons between males and females. In addition, low levels of creatinine in infants relative to adults may result in cotinine:creatinine ratios for infants that fall into the range reported for active smokers (OEHHA, 1997). In general, it is preferable to collect urine over 24 hours, although it is impractical in most cases.

ii) Nicotine and Cotinine – Duration in body fluids/hair

The average half-life of cotinine in adults, in different body fluids (plasma, saliva, urine) is about the same, approximately 15 - 19 hours, making it a good indicator of integrated ETS exposure over the previous two to three days. While the half-life of cotinine has been well studied in adults, little data exist for infants and children. Etzel *et al.* (1985) found half-lives of approximately 68 hours in neonates with wide variability. The USEPA lists half-lives of 60 hours in infants under 18 months and 40 hours in children over 18 months (USEPA, 1992). Recent work, however, by Leong *et al.* (1998) found similar half-lives of about 27 and 28 hours (no statistical difference) between children under and over two years of age. They postulated that higher cotinine levels in infants are actually due to greater exposure rather than slower metabolism. Cotinine levels in children are discussed in much greater detail in subsection 8**.** Clearly, more work is needed in this area. Nicotine, with its shorter half-life of approximately two hours, is a good indicator of recent exposure.

Hair nicotine has recently been used as an indicator of longer-term exposure, on the order of months to years. Hair grows at approximately 1 cm per month, and nicotine deposited within the hair shaft is stable throughout the life of the hair. Nicotine is deposited in the hair shaft both systemically during the synthesis of the hair shaft and by uptake from atmospheric exposure. The contributions of nicotine to the hair shaft from these two processes are an area of debate. Mizuno *et al.* (1991) proposed that the dominant process is the systemic pathway based on a constant level of nicotine along the shaft in smokers and a downward gradient toward the root in persons that had quit smoking. They did not evaluate the atmospheric pathway. In contrast, Zahlsen and Nilsen (1990) reported such a gradient in both smokers and non-smokers. In addition, these workers and others report a large nicotine:cotinine ratio in hair of approximately 15:1, which is essentially the inverse of the ratio of these compounds found in bodily fluids. Hence, they postulated that absorption of nicotine from the atmosphere was the predominant pathway for uptake (Zahlsen and Nilsen, 1990, Nilsen *et al.*, 1994). Addressing this controversy, work done by Gerstenberg et al. (1995) on rat hair demonstrated that the processes are of the same order of magnitude, with up to ten-fold higher levels in pigmented vs. unpigmented rat hair. The affinity of nicotine for melanin was noted also by Uematsu *et al*. (1995). More work in this area is clearly needed.

The value of hair nicotine as a biomarker for ETS exposure is less controversial. Zahlsen *et al*. (1996) found that hair nicotine levels tracked both smoking habits consistently among smokers and ETS exposure among non-smokers. Hair nicotine can be used to distinguish between ETS exposed and non-exposed children, and was found to be a better indicator of level of exposure than urinary cotinine in children (Al-Delaimy *et al*., 2001; Al-Delaimy *et al.,* 2002; Nafstad *et al*., 1995). Pichini *et al*. (1997) found that hair nicotine levels in infants was consistent with exposure by questionnaire while serum cotinine levels were below detection limits. Hair nicotine has also been used as a marker of gestational smoking (Eliopoulos *et al*., 1996), and as a marker for compliance with smoking cessation (Uematsu *et al*., 1995).

3. Analytical Methods for Nicotine/Cotinine

Laboratory methods are available that accurately quantify nicotine and cotinine in body fluids or hair. Inter-laboratory studies outlined in OEHHA (1997) found that gas chromatography and radioimmunoassay techniques reliably quantify nicotine and cotinine in plasma and urine, and both techniques are capable of discriminating between smokers and non-smokers. High performance liquid chromatography (HPLC) and gas chromatography are the most specific, especially when combined with mass spectrometry (Haufroid and Lison, 1998) and both techniques have been widely used. Levels of detection for cotinine vary from as low as 0.05 ng/ml for gas chromatography/mass spectrometry to as high as 1.0 ng/ml (Phillips *et al*., 1999) for radioimmunoassay, depending on the methodology followed. The 1997 OEHHA report documents substantial inter-laboratory variability, with many laboratories unable to detect cotinine in exposed non-smokers. Addressing the need for greater analytical accuracy in exposed non-smokers, Phillips et al. (1999) recently developed a chromatographic method utilizing tandem mass-spectrometry for detection of saliva cotinine with sufficient sensitivity to 0.05 ng/ml. This sensitivity will reliably distinguish between exposed and unexposed non-smokers. Similar methods were used in the NHANES III Study (Caraballo *et al*., 1998). Hair nicotine levels have been measured by radioimmunoassay techniques (e.g., Eliopoulos *et al*., 1996), that can also differentiate between ETS-exposed and unexposed non-smokers reliably (Al-Delaimy *et al*., 2001).

a) Cotinine Concentrations in Body Fluids

The levels of ETS encountered by ETS-exposed non-smokers during their daily activities are sufficiently high that nicotine and cotinine are detected in their urine, blood, saliva and hair. Given its longer half-life, high sensitivity, specificity and ease of measurement as a biomarker, cotinine in body fluids, rather than nicotine, has emerged as the biomarker of choice for most ETS studies. Numerous studies are available that report concentrations of cotinine in the physiologic fluids of smokers and non-smokers. Because several recent, very large scale studies have published their results since the printing of the 1997 OEHHA report, the numerous smaller studies will not be discussed here, except to say that cotinine levels seen in these studies tend to agree with those discussed below.

The 1997 OEHHA report found cotinine levels in saliva and plasma of non-smokers typically in the range of 0.5 - 15 ng/ml, and urinary concentrations of 50 ng/ml or higher. The Health Survey for England (Jarvis *et al.*, 2001), with over 20,000 participants, compared the plasma cotinine concentrations in non-smoking partners of smokers to partners of non-smokers. The study found that non-smokers in non-smoking homes had average plasma cotinine levels of 0.31 ng/ml, while non-smokers with partners smoking 30 or more cigarettes daily, had an average plasma cotinine of 1.99 ng/ml. There was a very strong positive relationship between number of cigarettes smoked by the partner and plasma cotinine levels in the non-smoker. Cotinine levels were also related to the partner's cotinine levels, with plasma cotinine averaging 0.31 ng/ml when the partner's cotinine was less than 15 ng/ml, and 1.30 ng/ml when the partner's was

over 4.0 ng/ml (Jarvis *et al.,* 2001). Analyzing data from the Sixteen City Study, LaKind *et al*. (1999) reported that non-smokers exposed to ETS in both the home and work environment had average salivary cotinine levels of 1.78 ng/ml, while unexposed non-smokers had cotinine levels averaging 0.182 ng/ml. Lee (1999) found a similar relationship between ETS exposure and serum cotinine. In four large scale studies listed in the review (over 18,000 subjects), average cotinine levels in non-exposed non-smokers was about 0.7 ng/ml, while in the most heavily exposed non-smokers the levels averaged about 2.5 ng/ml, which is consistent with the findings of the Health Survey of England. There are many smaller scale studies that reinforce these numbers as well. In six studies reviewed by Lee (1999), cotinine concentrations in urine varied widely, ranging from a low of 4.0 ng/ml to a high of 680 ng/mg-creatinine. Most studies show urinary cotinine levels in non-smokers to be less than 10 ng/ml or 10 ng/mg-creatinine (cf. Table 8 in Lee (1999)). The studies showing the higher values may not have removed subjects from the study that had cotinine values in the smoker range, so the higher number may not be truly reflective of non-smokers. Galanti *et al.* (1998) reported urinary cotinine concentrations among 2,431 young men in the Belgian Armed Forces averaging 32 ng/mg-creatinine in non- or ex-smokers and 717 ng/mg-creatinine in active smokers. In this study, an ex-smoker was someone who had not smoked in the last month.

Studies of individuals exposed in locations of exceptionally high concentrations of ETS provide some indication of the maximum concentrations of nicotine and cotinine reported in non-smokers. Jarvis *et al*. (1992), as described in the 1997 OEHHA report, reported a median salivary cotinine concentration of 7.95 ng/ml in 42 nonsmoking bar staff in England, with a maximum concentration of 31.3 ng/ml. Maskarinec *et al*. (2000) reinforced these findings in a similar population of bar staff. Using personal exposure monitors (as described below), the highest salivary cotinine level among bartenders (i.e., 95^{th} cotinine percentile) was as high as 20 ng/ml. The 1997 OEHHA report describes a study involving individuals exposed to ETS on commercial airline flights. The highest average urinary cotinine concentration among the study participants was 30 ng/mg-creatinine (Mattson *et al.,* 1989). In a flight attendant study by Lindgren *et al*. (1999), urinary cotinine concentrations as high as 36 ng/mg-creatinine were measured, reinforcing the previous findings. Haufroid and Lison (1998) assert that urinary cotinine levels in non-smokers are always less than 100 ng/mg-creatinine.

b) Relationship Between Cotinine Levels and Air Nicotine Levels by Personal Exposure Monitoring

The 1997 OEHHA report presented a study by Hoffmann *et al*. (1984) that linked air nicotine levels to salivary cotinine levels. These workers evaluated salivary cotinine in a closed room with 10 non-smoking volunteers. ETS was generated via a smoking machine. At a nicotine concentration of 280 $\mu g/m^3$, salivary nicotine levels reached an average of 880 ng/ml after 60 minutes of exposure, while cotinine climbed to 3.4 ng/ml six-hours post exposure. Experiments such as these have been replaced by personal exposure monitoring, where the subject wears a monitor that collects air close to the subjects breathing zone for a set period of time. Air concentrations of nicotine, respirable particulate matter, ultraviolet-absorbing particulate matter, solanesol,

scopoletin, and 3-ethenyl pyridine are typically measured. The metabolite of interest, typically cotinine, is analyzed at various times before, during and after the monitoring time frame. This type of monitoring, in theory, provides an exposure picture that closely approximates day-to-day living.

As a prelude to the following studies, concerns have been raised regarding the validity of the findings regarding workplace and home exposures to ETS. A multitude of concerns regarding the Sixteen City Study are discussed in USEPA (1996). Among the many concerns is the low nicotine concentrations measured in workplaces, which are significantly lower than nicotine concentrations measured as area concentrations at worker's desks in similar studies (Hammond *et al.*, 1999). Nicotine concentrations reported by Phillips *et al.* (1998) also are lower than in comparable studies (Phillips and Bentley (2001), or in area studies (Hammond *et al*., 1999). In presenting the data, the CARB is not endorsing findings regarding the contribution of the workplace vs. the home environment to ETS exposure. Rather, we are simply presenting data linking physiologic cotinine levels to measured atmospheric nicotine levels.

Phillips *et al.* (1999) has performed personal exposure monitoring on over 1,000 subjects in eight European cities, three Asian cities and in Sydney, Australia. Their data allow categorization of exposure into a number of environments (i.e., non-smoking work/home, smoking work/home, and combinations of these), depending on the study. Table V-12 presents the 24-hour time weighted average air concentrations of nicotine and salivary cotinine in European and Asian housewives.

Table V-12

Median Nicotine Concentrations in Inhaled Air with Corresponding Salivary Cotinine Concentrations

Location	Nicotine (μg/m^3)		Cotinine (ng/ml)	
	SH	NSH	SH	NSH
Stockholm	1.1	<0.08	2.9	<1.0
Barcelona	0.74	0.11	1.4	<1.0
Turin	1.1	0.14	1.4	<1.0
Paris	0.52	0.13	1.3	<1.0
Bremen	0.49	<0.08	1.4	<1.0
Lisbon	0.19	<0.08	1.2	<1.0
Basel	0.6	<0.08	1.0	<1.0
Prague	0.72	0.15	1.2	<1.0
Hong Kong	<0.06	<0.06	<1.0	<1.0
Kuala Lumpur	0.18	<0.06	1.0	<1.0
Beijing	1.4	0.15	<1.0	<1.0
Sydney	0.3	<0.08	1.4	<1.0

SH: Smoking home, NSH: Non-smoking home.
Adapted from Phillips et al., 1999 – Tables 1 and 2.

These data clearly demonstrate the increased cotinine in housewives of smoking vs. non-smoking homes. Many of the cotinine values measured were near or below the limit of quantification for radioimmunoassay, hence stronger trends were unable to be derived. Variations in home nicotine levels are strongly influenced by season and climate (i.e., ventilation).

Phillips *et al*. (1998) looked at subgroups based on lifestyle in some studies. In Prague, subjects were divided into six lifestyle groups (Table V-13).

Table V-13

Effect of Home vs. Work Smoking Environment on Exposure to ETS

Cell	Home Environment	Work Environment	Arithmetic mean cotinine (ng/ml)	Arithmetic mean nicotine (μg/m3)
1	Smoking	[a] -	2.4	1.3
2	Non-smoking	[a] -	0.98	0.31
3	Smoking	Smoking	2.7	2.3
4	Smoking	Non-smoking	1.9	1.3
5	Non-smoking	Smoking	1.4	1.1
6	Non-smoking	Non-smoking	0.71	0.25

Adapted from Phillips *et al*., 1998.
(All subjects had cotinine < 25 ng/ml).
a - implies non-working

These data further reinforce the relationship between ETS exposure and cotinine levels discussed previously. The data also demonstrate that the home environment is a greater contributor to ETS exposure than the work environment. Workplace nicotine levels in this study are lower than those measured in other similar studies (cf. Table V-9).

Phillips and Bentley (2001) conducted a different subgroup analysis in Bremen. Nicotine and cotinine were averaged over 24 hours and 7 days during both winter and summer on people either living and working in smoking locations or living and working in non-smoking locations (Table V-14).

Table V-14

Seasonal Effect on ETS Exposure

Cell	Locations	Length of monitoring	Arithmetic mean cotinine (ng/ml)	Arithmetic mean nicotine (μg/m3)
1	Smoking	24 hour –winter	1.6	2.7
2	Smoking	7 day – winter	1.6	2.1
3	Smoking	24 hour-summer	0.94	1.1
4	Smoking	7 day-summer	0.76	1.6
5	Non-smoking	24 hour –winter	0.73	0.36
6	Non-smoking	7 day – winter	1.2	0.27
7	Non-smoking	24 hour-summer	0.56	0.11
8	Non-smoking	7 day-summer	0.55	0.05

Adapted from Phillips and Bentley, 2001.
(All subjects had cotinine < 25 ng/ml).

Once again, these data provide support for the close relationship between ETS exposure and cotinine, as well as the importance of ventilation on ETS exposure.

In the Sixteen City Study, similar to Phillips and Bentley (2001), La Kind *et al*. (1999) analyzed personal exposure on over 1,000 subjects in 16 American cities. These workers divided their subjects into four cells and found the following (Table V-15):

Table V-15

Effect of Home Versus Work Smoking Environment on Exposure to ETS

Cell	Home Environment	Work Environment	Median cotinine (ng/ml)	Median nicotine ($\mu g/m^3$)
1	Smoking	Smoking	1.78	1.55
2	Smoking	Non-smoking	0.807	0.49
3	Non-smoking	Smoking	0.347	0.11
4	Non-smoking	Non-smoking	0.182	0.03

(All subjects had cotinine < 15 ng/ml).

Similar to Phillips *et al*. (1998), they concluded that the home environment was more significant, in terms of exposure, than the work environment. Once again, the validity of workplace nicotine levels was challenged (USEPA, 1996). Limited information on cotinine concentrations in California subjects is available. In the Sixteen City Study by Jenkins *et al*. (1996), Fresno was the only California region evaluated. Atmospheric nicotine concentrations, both at work and away from work, were among the lowest of the cities tested. These low concentrations contrast with data from an earlier, large multinational study that included a center located in Los Angeles (Riboli *et al*., 1990). These researchers studied 100 non-smoking women with the following marital and employment status: 13% were married to a smoker and employed; 39% were married to a smoker and unemployed; 16% were not married to a smoker and employed; and 32% were not married to a smoker and unemployed. The mean urinary cotinine:creatinine ratio was approximately 8.5 ng/mg for the entire population, and 10.5 ng/mg for those with detectable urinary concentrations. The differences in cotinine levels were found to be large and statistically significant between the 13-centers, and the concentrations at the Los Angeles center was one of the three highest in the study.

c) Nicotine and Cotinine: Comparison of Levels in Smokers, and ETS-exposed and Unexposed Non-smokers

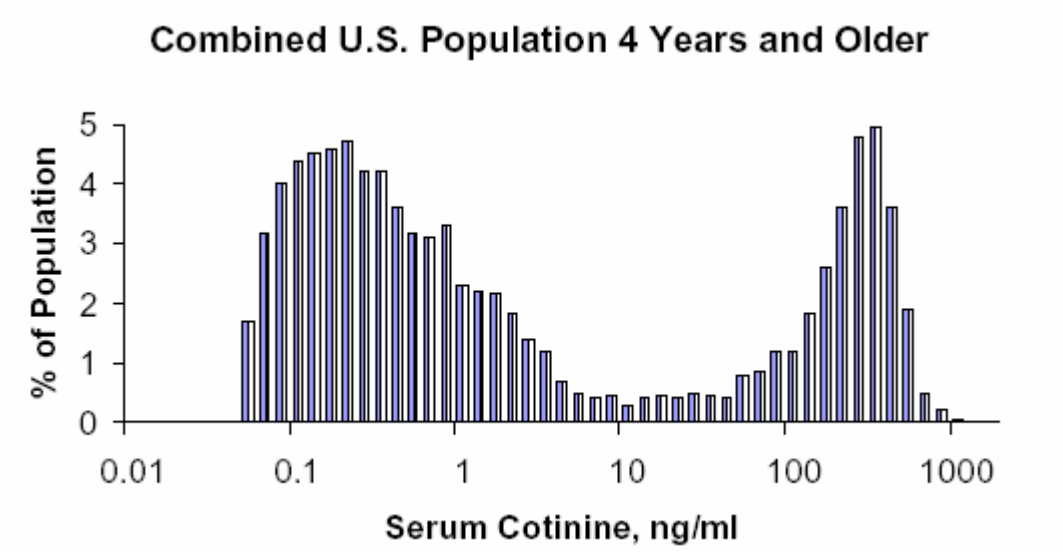
Cotinine assays using serum, saliva or urine can consistently distinguish between smokers and non-smokers. Ogden *et al.* (1997), in a nationwide survey, found the mean salivary cotinine in active smokers to be 352.9 ng/ml. Findings from this study and from OEHHA (1997) consistently show at least an order of magnitude difference in the cotinine concentrations between active and non-smokers. Data below also graphically depict this difference. In OEHHA (1997), findings were less consistent with regard to distinguishing between ETS-exposed and unexposed non-smokers, for reasons including limited analytical accuracy, misreporting of exposure, variations in

metabolism, and others. The more recent large studies, using sensitive analytical methods such as HPLC, have been consistently able to distinguish between ETS-exposed and unexposed non-smokers.

The relationship between ETS exposure and cotinine is clearly demonstrated in Figures V-1 through V-3, which present data from the very large NHANES III study and the Health Survey for England. Figure V-1 (Pirkle *et al*., 1996) below presents serum cotinine levels in over 10,000 participants in the NHANES III study.

Figure V-1

Distribution of Serum Cotinine Levels in the US Population Aged 4 Years and Older

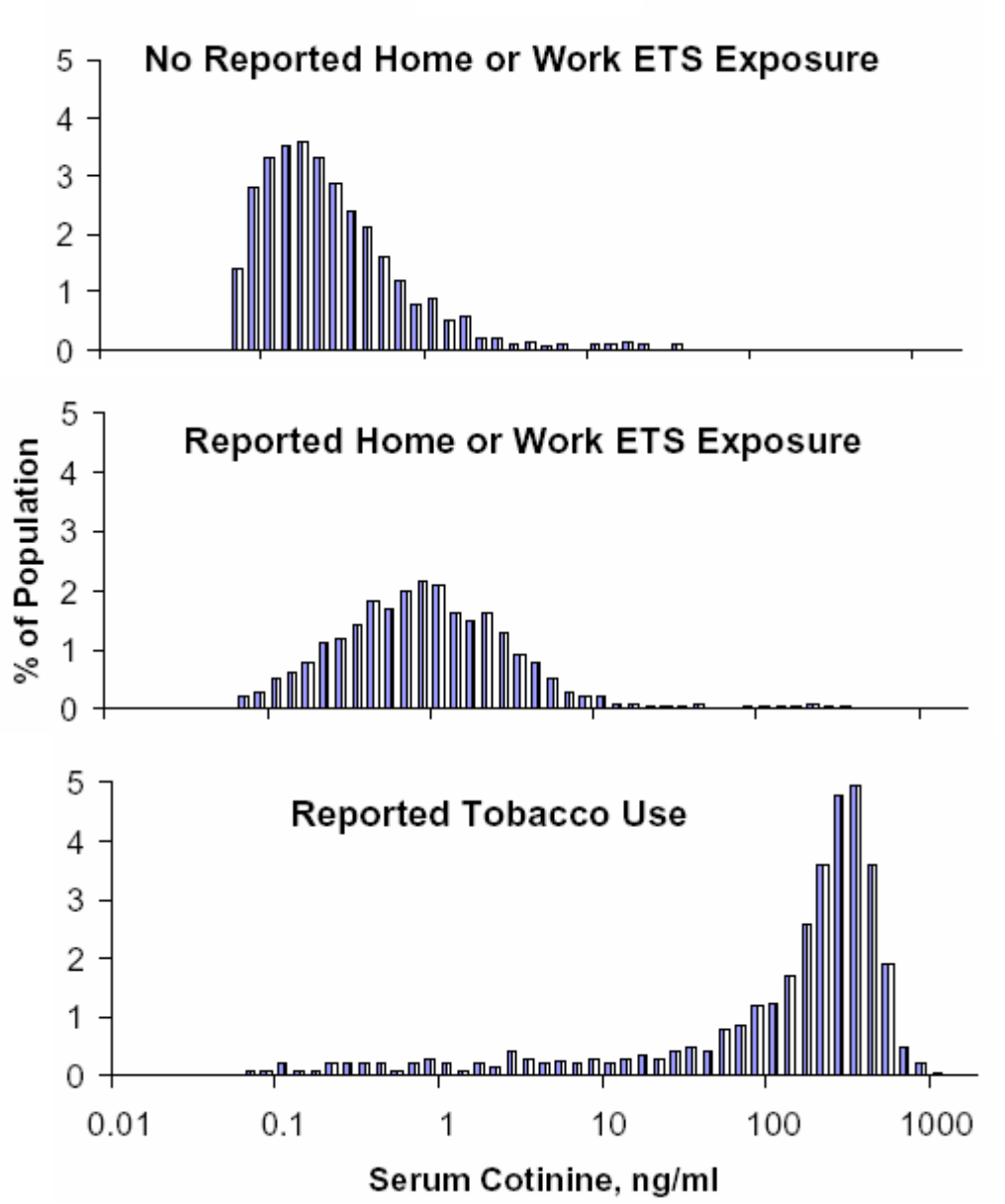


Distribution of serum cotinine levels in the U.S. population aged four years and older: Third National Health and Nutrition Survey, October 25, 1988, to October 21, 1991.
Source: Pirkle *et al*. (1996).

Figure V-2 (Pirkle *et al*., 1996) divides these data into groups based on type of exposure.

Figure V-2

Distribution of Serum Cotinine Levels in the U.S. Population Aged Four Years and Older by Tobacco Use

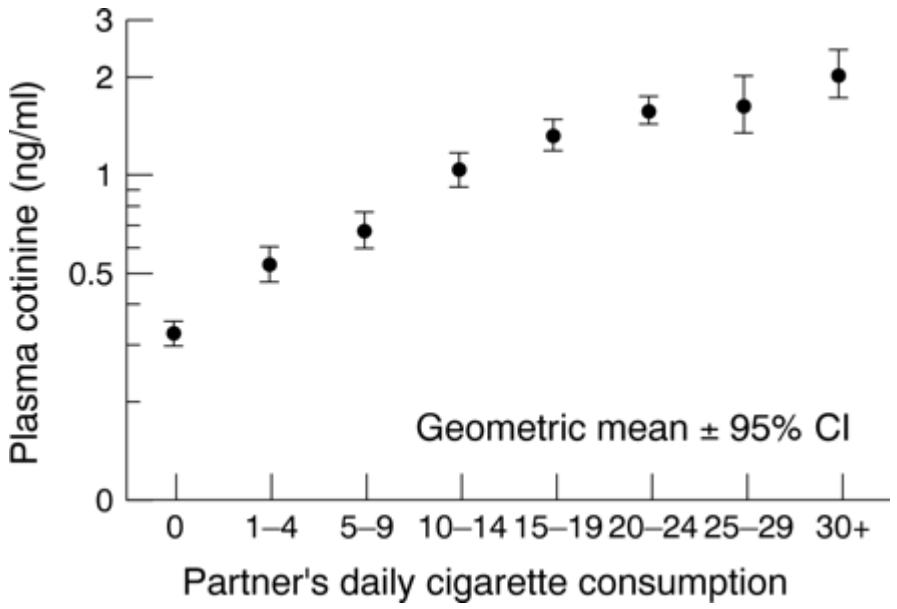


Distribution of cotinine levels in the U.S. population aged four years and older by reported environmental tobacco smoke exposure and tobacco use: Third National Health and Nutrition Examination Survey, October 25, 1988, to October 21, 1991. Source: Pirkle *et al*. (1996).

The bimodal distribution depicted above has been demonstrated in other studies. The lower hump represents non-smoking individuals exposed either in their work or home environment to environmental tobacco smoke. The higher cotinine hump represents active smokers. The values between 10 and 25 ng/ml cotinine represent an area of uncertainty as to whether these individuals are heavily exposed non-smokers or occasional smokers. The curve below, derived from the Health Survey of England (Jarvis *et al*., 2001) provides a detailed look at the cotinine concentrations in over 20,000 partners' of smokers based on the partners' cigarette consumption.

Figure V-3

Cotinine Concentrations Based on Partner's Cigarette Consumption



Source: Jarvis *et al*. (2001).

The power of these studies provides strong evidence that cotinine levels in non-smokers are almost always below 10 ng/ml. These data are well supported by numerous studies (OEHHA, 1997; Lee, 1999; Phillips *et al.*, 1999). Pregnant women with similar ETS exposures to non-pregnant subjects will have lower cotinine levels due to higher renal clearance rates (see reproductive health effects in Part B of this report). Authors usually list their cutoff level at which they designate a subject as a non-smoker, with almost all authors opting for a cutoff between 10 - 25 ng/ml. Caraballo *et al.* (1998), in reviewing the NHANES III data, found that a serum or plasma cotinine level below 15 ng/ml is consistent (i.e., 98 – 99 percent of the time) with non-smoking status. Maskarinec *et al*. (2000), evaluated 173 non-smoking bar staff using personal exposure monitors in Knoxville, Tennessee. Table V-16 lists the cotinine levels from a subset of the population with the highest ETS exposures:

Table V-16

Job-Related Cotinine and Nicotine Measured Concentrations

Home Status	Job Classification		Average Salivary Cotinine (ng/ml)	Shift Average Nicotine Concentration ($\mu g/m^3$)
Smoking	Wait Staff	Median	4.08	3.20
		Mean	4.32	12.1
		80^{th} percentile	6.05	17.8
		95^{th} percentile	11.1	54.0
	Bartenders	Median	4.85	12.6
		Mean	6.54	19.2
		80^{th} percentile	8.97	33.2
		95^{th} percentile	20.2	57.9
Non-Smoking	Wait Staff	Median	1.43	0.93
		Mean	2.61	3.32
		80^{th} percentile	3.62	4.47
		95^{th} percentile	8.24	18.2
	Bartenders	Median	2.00	3.90
		Mean	3.67	11.2
		80^{th} percentile	4.90	20.1
		95^{th} percentile	12.8	34.9

Adapted from: Maskarinec *et al*. (2000).

These data are not inconsistent with the findings of the larger studies discussed above. Rather, the high cotinine levels found in this study are consistent with those persons in the maximum ETS exposure percentile.

Etzel (1990) proposed the following range:

Salivary Cotinine Level	Smoking Classification
<5 ng/ml	Low-moderate passive smoking
>5 - <10 ng/ml	Heavy passive smoking
10 – 100 ng/ml	Infrequent to regular smoking with low nicotine content
>100 ng/ml	Regular active smoking

These ranges are consistent with data from the later, larger studies mentioned above.

4. Biomarkers: Carbon Monoxide and Carboxyhemoglobin

Carbon monoxide, both in exhaled alveolar air and as carboxyhemoglobin in blood, originates from endogenous processes as well as from environmental sources. In addition to cigarette smoke, common environmental sources include vehicle exhaust, gas stoves and furnaces, and kerosene space heaters. Although carbon monoxide and carboxyhemoglobin have been used to distinguish smokers from non-smokers (OEHHA, 1997), they are generally not the best indicators of ETS exposure because of their lack of sensitivity and specificity. In non-smokers exposed to environments heavily polluted with ETS, elevated levels of exhaled carbon monoxide and carboxyhemoglobin in blood have been detected when measured 30 minutes following cessation of exposure. However, the use of these biomarkers in distinguishing between subjects with no, little, or high levels of ETS exposures is limited (OEHHA, 1997).

5. Biomarkers: Thiocyanate

Hydrogen cyanide, in the vapor phase of tobacco smoke, is metabolized in the liver yielding thiocyanate (SCN-). Thiocyanate levels in blood, urine and saliva have been used to distinguish smokers from non-smokers, or in combination with assays for nicotine or cotinine, to distinguish smokers from individuals using smokeless tobacco or nicotine-containing products (OEHHA, 1997). Sources of thiocyanate are also present in the diet, particularly cruciferous vegetables; thus, levels of thiocyanate in body fluids are not specific to exposure to tobacco smoke. In studies examining the use of thiocyanate as a biomarker of ETS exposure, it was not possible to distinguish between ETS-exposed and unexposed non-smokers (OEHHA, 1997). Recent work by Scherer *et al*. (2000**)** reinforces these previous findings. In the study described in subsection 6, non-exposed non-smokers had average plasma thiocyanate levels of 22.0 µmol/L, which is higher, though not significantly different, than the corresponding level in ETS-exposed non-smokers (i.e., 19.6 µmol/L). These same subjects had cotinine levels of 0.71 and 1.32 ng/ml, respectively, which are consistent with findings described in section 2(c) of this report. For this reason, thiocyanate is not considered to be a reliable biomarker of ETS, and has not been widely used for monitoring ETS exposure.

6. Biomarkers: Protein and DNA Adducts

Protein and DNA adducts represent both markers of exposure and measures of a biochemical effect. Associations between levels of these adducts and cotinine have been reported (OEHHA, 1997), as well as for hemoglobin and 4-aminobiphenyl (Hammond *et al.*, 1995). New studies using hemoglobin and albumin adducts describe significant overlap in the levels between unexposed persons and passive smokers.

One of the more common protein adducts measured is the hemoglobin adduct of 4-aminobiphenyl. Tobacco smoke is the primary source of environmental 4-aminobiphenyl. Because of the relatively long half-life of these adducts, their levels reflect exposures occurring over the previous four months. Levels of 4-aminobiphenyl

in ETS-exposed non-smokers compared to those of active smokers present an interesting contrast to cotinine levels measured in these two groups. The levels of 4-aminobiphenyl adducts in non-smokers are approximately 10 - 20% of the levels measured in smokers. Although this finding appears to be inconsistent with the results for urinary cotinine (for which levels in ETS-exposed non-smokers are about 1% of those in smokers), the results are aligned with data on the relative levels of nicotine and 4-aminobiphenyl in mainstream and sidestream smoke (cf. USEPA, 1992: Table 3-1). Approximately twice as much nicotine is present in sidestream as in mainstream smoke, whereas about 31-times as much 4-aminobiphenyl is present in sidestream as in mainstream smoke. As a result, the smoker/non-smoker ratio for 4-aminobiphenyl is about 15-times higher than for cotinine.

Another group of protein adducts which have been measured are the albumin adducts of polycyclic aromatic hydrocarbons (PAHs). Multiple PAHs are present in tobacco smoke. Crawford *et al.* (1994) analyzed PAH-albumin levels in peripheral blood of 87 mothers and their preschool children (2 - 5 years of age). They found PAH-albumin levels were significantly higher in children whose mothers smoked than in the children of non-smoking mothers ($p < 0.05$). Among nonsmoking mothers, the regression of PAH-albumin against total ETS exposure also showed a significant association with cotinine ($r^2 = 0.25$, $p = 0.04$).

Scherer *et al*. (2000) performed biomonitoring of exposure to PAHs in a field study of 69 subjects using benzo[a]pyrene (a PAH present in tobacco smoke) adducts of hemoglobin and albumin as well as urinary 1-hydroxypyrene. Subjects were non-occupationally exposed to PAHs, and non-smokers wore personal exposure monitors to quantify their exposure to ETS. Statistically significant differences in urinary excretion of hydroxypyrene and benzo[a]pyrene adducts were seen between smokers and non-smokers, but no significant differences were seen between ETS-exposed and non-exposed non-smokers.

Hemoglobin adducts of 4-hydroxy-1-(3-pyridyl)-1-butanone (HPB) have been studied by Atawodi *et al*. (1998) and others. In 70 hospitalized patients, hemoglobin-HPB adduct levels in 18 smokers averaged 26 fmol/g Hb versus 19 fmol/g Hb in 52 never-smokers ($p = 0.02$) (Atawodi *et al.,* 1998). No significant difference was seen between current smokers and ex-smokers. Carmella *et al*. (1990) reported levels of Hb-HPB in 40 smokers averaged 80 fmol/g Hb and 21 non-smokers averaged 29 fmol/g Hb, with large heterogeneity for both smokers and non-smokers**.** Foiles *et al*. (1992) reported averages of 163 fmol/g HB and 68 fmol/g Hb in 37 non-smokers in a study of 100-smokers. Falter *et al.* (1994) reported averages of 69 fmol/g Hb and 34 fmol/g Hb for these same respective groups.

Bono *et al*. (1999) looked at levels of N-(2-hydroxyethyl)valine (HOEtVal) on hemoglobin, an adduct formed from the reaction of ethylene oxide (in tobacco smoke) and valine residues on hemoglobin. Among 146 subjects, HOEtVal levels correlated well with the number of cigarettes smoked, and the difference between smokers and non-smokers was significant. However, no significant difference in HOEtVal levels between passive smokers and non-smokers was seen.

DNA adducts of tobacco smoke constituents can also be measured. The distribution of DNA adducts of benzo[a]pyrene diol epoxide, the ultimate carcinogenic metabolite of benzo[a]pyrene, has been analyzed by Denissenko *et al.* (1996) in the P53 tumor suppressor gene. These authors reported that exposure of human bronchial epithelial cells to benzo[a]pyrene diol epoxide resulted in strong and selective DNA adduct formation within the P53 gene at mutational hotspots identified in non-radon associated human lung cancer tissues obtained from smokers. This mapping of DNA adduct formation to mutational hotspots provides a direct etiologic link between a specific tobacco smoke carcinogen and human cancer. PAH-DNA adducts have been noted in smokers in many other studies.

7. Biomarkers: Other

Biomarkers of ETS exposure with high specificity for tobacco smoke include the metabolites of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK). NNK is found only in tobacco products, therefore, its metabolites, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) and its glucuronide (NNAL-Gluc), are specific to tobacco exposure. Hecht (2002) evaluated 16 carcinogen metabolites that appear in the urine following tobacco exposure for their utility as biomarkers. Among the compounds evaluated, in addition to NNAL and its glucuronide (NNAL-Gluc), were nitrosamines, PAHs, mercapturic acids, benzo[a]pyrenes and naphthols. Of these, NNAL plus NNAL-Gluc showed the highest specificity for tobacco exposure and the best ability to differentiate those with and without ETS exposure. Hecht *et al*. (2001) demonstrated the utility of this biomarker in a study of ETS exposure in children described below (Section 8b).

Taniguchi *et al.* (1999) studied urinary levels of trans, trans-muconic acid, a metabolite of benzene and sorbic acid, in both passive smokers and active smokers. There was significant overlap between the light active smoker group and non-smokers exposed to ETS. Ruppert *et al*. (1997) also studied the urinary excretion of this compound. There was no significant difference in urinary levels between non-smokers living in smoking homes and those living in non-smoking homes. Hence, the usefulness of this compound as a biomarker is probably limited, particularly in view of the ubiquitous presence of benzene in ambient air from fuels.

8. Biomarkers and Children

a) Nicotine and Cotinine: Studies in Infants and Children

ETS exposure among infants and children was described in OEHHA (1997). It is addressed here as a separate subsection to reflect the unusual exposure scenario associated with *in utero* exposure, and the involvement of two metabolizing systems, maternal and fetal, in affecting and in being affected by levels of nicotine and cotinine. Infants can be exposed prenatally to tobacco smoke constituents if the mother smokes or if the mother is exposed to ETS during pregnancy. Postnatal ETS exposure may occur directly, via inhalation, and indirectly, from ingestion of breast milk.

Cotinine has been detected in fetal fluids as early as seven-weeks gestation in both active and passive smokers (Jauniaux *et al*., 1999). In a study of 85 pregnant women, cotinine levels above 25 ng/ml in maternal serum and above 250 ng/ml in maternal urine were associated with detectable cotinine levels in amniotic and coelomic fluids, and fetal serum. In active smokers, positive linear correlations were reported between maternal urine and amniotic fluid cotinine concentration, between maternal urine cotinine concentration and number of cigarettes smoked per day, and between maternal and fetal serum cotinine concentrations. Nafstad *et al*. (1996) measured cotinine in cord serum, and found a significant correlation between the average number of cigarettes smoked by mothers and the concentrations of cotinine in cord serum. Using linear regression analysis of data from daily smokers, the reported increase in the concentration of cotinine in cord serum was 4.4 ng/ml per daily cigarette smoked.

In infants and children, nicotine and cotinine have been measured in hair, serum, saliva, and urine. Consistent with earlier reports, recent studies have shown that in children who are exposed to smoke, cotinine levels are associated with the age of the child, with the highest concentrations found in younger children. Irvine *et al*. (1997) studied children from ages 2 - 12 years old, from 501 families with at least one parent who smoked. They reported a stepwise reduction in salivary cotinine levels with ascending age, with the largest reduction detected between preschool 4-year olds and children from ages 5 - 7 years old. Similarly, Preston *et al.* (1997) reported that in a group 175 children (ages 2 - 11 years old), there were statistically significant differences in cotinine concentrations between age groups. The highest concentrations of urinary cotinine were found in the youngest children (2 - 4 years old) and the lowest concentrations in the oldest children (8 to 11 years old). They also reported that children from ages 2 - 4 years old, with smoke exposure exceeding 1-pack per day, had mean cotinine levels almost two-fold greater than older children having similar exposures. Kohler *et al*. (1999) examined passive smoke exposure in children 1-month to 11-years old. In this study, children were considered passive smokers if their urinary nicotine metabolite concentration (i.e., cotinine plus hydroxycotinine) was greater than 10 nmol/L. In addition to finding the highest concentrations in the youngest children, they also found that younger children ($\leq$ 5 years old) were identified as passive smokers more frequently than children over 5 years old (i.e., 83.7% vs. 52.4%, $p < 0.001$). Mannino *et al*. (2001) also found age to be an important factor. They analyzed NHANES III data (i.e., data collected in 1994 as part of the U.S. Third National Health and Nutrition Examination Survey) from over 5,500 children, ages 4 - 16 years old. Their analysis showed that age was an important predictor of serum cotinine levels, both in children exposed to smoke and in those not exposed to smoke, although the effects were opposite in these two groups. In children exposed to smoke, the highest levels of cotinine were found in the youngest children, but in the children of nonsmokers, older children appeared to have higher cotinine levels, presumably from sources outside the home.

Several researchers have suggested that the higher concentrations of cotinine found in infants and younger children exposed to ETS are likely due to greater exposure, or a higher relative dose of nicotine, rather than slower cotinine metabolism (Willers *et al*., 1995; Leong *et al*., 1998; Mannino *et al*., 2001). Infants have a higher ventilation rate

than older children or adults. It is also possible that they spend less time outdoors than older children and/or since they are less mobile, they are not able to leave a smoky environment. While the half-life of cotinine has been well studied in adults, little data exist for infants and children. Etzel *et al*. (1985) reported an average cotinine half-life in neonates of 68 hours, with a range of 37 - 160 hours, which is greater than that reported in adults. More recent findings indicate there is no difference in half-life between infants and older children. Leong *et al*. (1998) reported no significant difference in the half-life of cotinine in children under two years of age compared to older children. In this study, the urinary elimination half-life of cotinine was measured in 31 infants and young children (mean age, 4.8 months; range, 0-22 months) and compared to that in 23 older children (mean age, 95.6 months; range, 39-174 months). The median half-life was approximately 28 hours in the younger group (range 6 – 259 hours), and 27 hours in older children (range 10 – 99 hours); this difference was not statistically significant. By contrast, Dempsey *et al.* (2000) found the half-life of cotinine in newborns to be consistent with what they had previously found in adults, reporting values in neonates of 16.3 hours in blood (95% CI: 12.4 - 23.9 hours) and 22.8 hours in urine (95% CI: 19.5 - 25.8 hours).

The Dempsey *et al.* (2000) and the Etzel *et al.* (1985) studies, were similar in design (e.g., both collected urine samples from newborns during the first week of life). However, in the studies by Etzel *et al*. (1985) and Leong *et al.* (1998), data were normalized by creatinine concentrations while the data in Dempsey *et al.* (2000) was not. Most likely, this accounts for much of the difference in the results among these studies. It is common to correct for the effect of hydration on concentrations of urinary cotinine by adjusting the urinary cotinine level for urinary creatinine concentrations. Dempsey suggests that in neonates, however, adjusting for creatinine may lead to an overestimation of half-life. During the first week of life, neonates excrete a maternal load of creatinine, and therefore, their urinary creatinine concentrations do not reflect endogenous production. If this is true, then normalizing cotinine by urinary creatinine concentrations leads to an underestimation of cotinine during the first few days of life, which would result in an overestimation of the cotinine half-life. It thus appears that a half-life of 15 – 19 hours for the elimination of urinary cotinine may be a reasonable range for infants, children and adults.

In addition to their work on cotinine, Dempsey *et al*. (2000) also measured half-lives of nicotine, 3´-hydroxycotinine and their conjugates. They reported that the half-life of nicotine in newborns is 11.2 hours in blood (95% CI: 8.0 - 18.9 hours) and 9 hours in urine (95% CI: 7.0 - 12.4 hours), which is three to four times longer than adults. The elimination half-lives for the other metabolites were 13 hours for conjugated nicotine, 19.8 hours for conjugated cotinine, 18.8 hours for 3´-hydroxycotinine, and 19.4 hours for conjugated 3´-hydroxycotinine.

Regardless of age, there are data to suggest that asthmatic children may have a lower clearance rate of ETS than nonasthmatic children. Klein and Koren (1999) compared concentrations of nicotine and cotinine in asthmatic and healthy (non-asthmatic) children (ages 2 to 18 years) exposed to similar degrees of ETS. Urine samples were collected from 71 asthmatic children and 81 controls, hair was collected from

64 asthmatics and 77 controls, and parents provided information regarding smoking in the home. On average, the asthmatic children in this study were exposed to fewer cigarettes per day at home, although this difference was not statistically significant. Similarly, mean urine cotinine concentrations were lower in asthmatic children, although not statistically significant. In contrast, hair nicotine concentrations were almost twofold higher in asthmatic children compared to nonasthmatic controls ($p < 0.0001$), and the ratio of urine cotinine to hair cotinine was almost threefold lower in asthmatic children ($p < 0.0001$). Klein and Koren (1999) suggest that these data indicate a lower clearance rate of ETS in asthmatic children, and therefore a higher systemic exposure.

Mannino *et al.* (2001), who analyzed NHANES III data from over 5500 children across the U.S., found that the strongest predictor of cotinine levels in ETS-exposed children was the number of cigarettes smoked in the home. Studies have consistently shown that increased cotinine levels in ETS-exposed children are associated with the number of cigarettes smoked in the home, as well as the number of parents who smoke, particularly if mothers smoke (Irvine *et al*., 1997; Preston *et al*., 1997; Oddoze *et al*., 1999). Recent studies have also shown that, similar to adults, there are differences in cotinine levels among racial/ethnic groups. Mannino *et al.* (2001) reported the lowest mean cotinine levels among Mexican-American children, and the highest among black children in their study. Similar results were reported by Tang *et al.* (1999). The Tang study is discussed in greater detail below.

b. Other Biomarkers of ETS Exposure Measured in Children

In a study of elementary school-aged children, metabolites of the lung carcinogen 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) were measured and quantified in urine (Hecht *et al.,* 2001). NNK is found only in tobacco products; therefore, the metabolites 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) and its glucuronide (NNAL-Gluc) in urine are specific biomarkers of tobacco exposure. Two hundred and four children, grades 2-5, were included in this study (mean age = 8.9 yrs). Questionnaires were administered to caregivers about ETS exposure in the home. Urine samples from all of the children were analyzed for total cotinine (cotinine plus its glucuronide); a subset of 74 samples was also analyzed for the metabolites NNAL and NNAL-Gluc. Of the 204 children in the study, more than 34% had total cotinine levels ≥ 5 ng/ml urine, the cutoff used in this study to indicate ETS exposure. Among the samples with total cotinine ≥ 5 ng/ml, which were also analyzed for NNAL and NNAL-Gluc, 52 of 54 (96%) were positive for one or both of these carcinogen metabolites. NNAL or NNAL-Gluc was also detected in 10 of 20 samples (50%) in which total cotinine was < 5 ng/ml. The more frequent detection of NNAL and NNAL-Gluc than of total cotinine may be due to pharmacokinetic differences of these metabolites. In this study, NNAL plus NNAL-Gluc correlated with total cotinine ($r = 0.71$; $p < 0.0001$). Concentrations of NNAL, NNAL-Gluc and total cotinine are shown in Table V-17 below. Concentrations of cotinine, NNAL, and NNAL-Gluc were not significantly different in samples collected twice from the same children at 3-month intervals. Authors noted that levels of NNAL plus NNAL-Gluc were comparable to those they observed in previous studies of adults exposed to ETS. Authors also noted that while it is likely that

uptake of nicotine and NNK by the children in this study was attributable to ETS, it is possible that some of the children may have smoked a cigarette.

Table V-17

Concentrations of NNAL, NNAL-Gluc, and Total Cotinine (mean ± SD) in the Urine of Elementary School-aged Children[1]

Group	No. of children[2]	NNAL (pmol/ml)	NNAL-Gluc (pmol/ml)	NNAL + NNAL-Gluc (pmol/ml)	Total cotinine (ng/ml)
All	74	0.081 (± 0.030)	0.040 (± 0.050)	0.056 (± 0.076)	12.0 (± 17.8)
ETS exposure reported in questionnaire	38	0.032 (± 0.039)	0.064 (± 0.056)	0.095 (± 0.088)	24.5 (± 22.4)
No ETS exposure reported in questionnaire	35	0.010 (± 0.020)	0.026 (± 0.040)	0.035 (± 0.058)	5.0 (± 8.7)
Total cotinine[3] < 5 ng/ml	20	0.005 (± 0.010)	0.012 (± 0.020)	0.016 (± 0.030)	1.2 (± 1.6)

[1]Source: Hecht *et al.*, 2001.
[2]One child did not have questionnaire data on exposure.
[3]Total cotinine < 5 ng/ml is the cutoff used by the authors to indicate ETS exposure.

Nafstad *et al.* (1996) examined the relationship between maternal smoking habits and concentrations of thiocyanate and cotinine in cord blood. (The results regarding cotinine are summarized above.) The women in this study were self-reported non-smokers, occasional smokers, and daily smokers. Among newborns of mothers smoking 1-9 cigarettes per day, the median concentration of thiocyanate was 43 μmols/L (25-75th percentile: 23-58 μmol/L) and among newborns of mothers smoking 10 or more cigarettes per day the median thiocyanate concentration was 62 μmols/L (25-75th percentile: 44-71 μmol/L). The correlation between the average number of cigarettes smoked by the mothers and the concentration of thiocyanate in cord serum was 0.46 ($p = 0.003$), and the correlation between thiocyanate and cotinine was 0.63 ($p < 0.001$). Using linear regression analysis of just the daily smokers, the increase in the concentration of thiocyanate in cord serum per daily cigarette smoked was 2.3 μmol/L.

In a study by Tang *et al.* (1994), 4 biological markers of ETS exposure were evaluated in a cohort of Hispanic and African-American preschool children. There were

109 children included in this study, from 1 to 6 years old. Investigators measured plasma cotinine, protein adducts of two carcinogens (*i.e.*, the hemoglobin adducts of 4-aminobiphenyl (4-ABP-Hb) and the albumin adducts of polycyclic aromatic hydrocarbons (PAH-albumin), and sister chromatid exchanges (SCEs; used as a general indicator of genetic damage). Information on ETS exposure at home was obtained by questionnaire. All of the biomarkers were higher in ETS-exposed children than in unexposed children. The differences were statistically significant for cotinine ($p < 0.001$), 4-ABP-Hg ($p < 0.05$) and PAH-albumin ($p < 0.05$). SCEs were marginally higher ($p = 0.076$). In addition, when children were grouped by exposure (no reported ETS exposure, exposure by household members other than the mother, or exposure from maternal smoking) all of the biomarkers increased across exposure groups, although the differences were not always statistically significant. And finally, African-American children had higher levels of cotinine ($p = 0.059$) and PAH-albumin ($p = 0.02$) than Hispanic children, after adjusting for exposure. Authors note that this finding is limited by small numbers and the possibility of exposure misclassification; however, it is consistent with other data showing ethnic variation in the internal dose of ETS observed in adults. It is also consistent with the results observed in children in the analysis by Mannino *et al.* (2001), as previously discussed.

9. Summary and Conclusions

Cotinine, the major metabolite of nicotine, has emerged over the past 20 years as the biomarker of choice for most field exposure studies and for validation of smoking status. Physiologic cotinine concentrations differ typically by several orders of magnitude between smokers and ETS-exposed non-smokers. Cotinine is a sensitive enough biomarker that its concentrations can reliably distinguish between non-ETS-exposed persons and ETS exposed non-smokers with low, moderate and high levels of exposure. However, due to a half-life of around 20 hours, cotinine levels in body fluids reflect exposures only during the preceding day or two. To the extent that these exposures are typical, cotinine levels are a good measure of an individual's general ETS exposure. However, when exposures are episodic or characteristic of a particular environment (e.g., work vs. home), the timing of sampling is critical to avoid over- or under-estimation of exposure. Sampling at multiple, varied times, and/or measurement of tissues reflecting longer-term exposures, such as hair, are useful in this context. Future data may show that the relationship between ETS exposure and cotinine levels are potentially strong enough to link adverse health outcomes to physiologic cotinine levels. These same data may be useful in determining which study subjects may actually be smokers rather than ETS-exposed non-smokers that would otherwise skew study findings. Results from ongoing personal exposure monitoring studies are shedding light on the relationship between inhaled nicotine concentrations and physiologic cotinine concentrations. These studies also show that there is a relationship between the relative contributions to ETS exposure in the home and workplace with the smoking activity found in those environments.

Hair nicotine is an emerging biomarker that may be as effective as cotinine in determining levels of ETS exposure. Hair nicotine has the important advantage of providing an integrated measure of exposure over a period of months. As such, it is

less susceptible to measurement errors associated with the timing of sample collection, as may occur with cotinine measurements in body fluids in cases of episodic versus continuous passive exposure. However, relatively few studies have used hair nicotine as a biomarker for ETS. Larger studies are needed to determine the effects of hair color and hair treatments on nicotine binding, and show that hair nicotine is a viable biomarker for ETS.

Another tobacco-specific biomarker with good ability to differentiate among smokers, non-smokers with ETS exposure, and those without, is NNAL. This metabolite of the carcinogen, NNK, has been detected in several body fluids in association with tobacco exposure. Assayed in conjunction with its glucuronide conjugate, it is an especially attractive compound for analyses of urine. However, it has thus far not been widely applied in studies of passive smoking.

Other biomarkers of ETS exposure, such as DNA and protein adducts, link ETS exposure directly to carcinogenic metabolites. These biomarkers, while useful in linking tobacco smoke exposure to toxic or carcinogenic end points, are generally not used to distinguish between ETS-exposed non-smokers and unexposed non-smokers. The use of carbon monoxide and thiocyanate as ETS biomarkers are not specific to tobacco smoke and therefore have limitations for use as biomarkers. Cotinine, nicotine, and NNAL/NNAL-Gluc are the only biomarkers that have been demonstrated to be both tobacco-smoke specific and able to reliably distinguish between ETS exposed and unexposed non-smokers. Of these, the assays for cotinine have been the best developed and most widely applied. For this reason, cotinine is currently the preferred biomarker for comparison among studies of ETS exposure. When attempting to quantify degrees of ETS exposure, the other biomarkers discussed in this chapter are of less utility.

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VI.

ATMOSPHERIC PERSISTENCE

Among other factors, the combustibility of tobacco components, insufficient supply of oxygen, and the existence of a temperature gradient in the burning cone, makes ETS a mixture of several thousand compounds. Due to the complex chemical nature of ETS, a discussion of the atmospheric persistence of the "mix" as a whole is not practical. However, there are data on the atmospheric reactions that occur to several groups of ETS-related chemicals. Therefore, in this chapter we provide a general discussion of what is known about the atmospheric persistence of chemical groups within ETS, including nicotine, N-Nitrosamines and PAHs.

Studies show that the combustion of cigarettes include at least three important types of reactions, including: pyrolysis, pyrosynthesis, and distillation (NCI, 1998). The result of these reactions is the production of thousands of gaseous and particle constituents. Eventually, this complex mixture undergoes additional chemical reactions as the mix is diluted with ambient air, yielding individual compounds with their own atmospheric lifetimes. According to the Morawska *et al.* (1997) chamber and indoor environment study, the lifetime of the mixture of ETS constituents in the air may be up to several hours depending on the air ventilation rate, humidity, and atmospheric conditions.

A. ATMOSPHERIC REACTIONS OF GASEOUS SPECIES

Gaseous ETS constituents can react in the atmosphere with other pollutants and sunlight to form new chemical species (see Table III-2 in Chapter III for a list of gaseous components found in ETS; Appendix A contains a comprehensive list). For example, 1,3-butadiene can initially react in the atmosphere with the hydroxyl radical (OH), nitrate radical (NO_3), and ozone (O_3) to form acrolein and formaldehyde (Agency for Toxic Disease Registry (ATSDR), 1993; Skov *et al*., 1992). Gaseous species may also transform into particle phase species. For example, gas phase ammonia can react with gaseous nitric acid to form particulate ammonium nitrate (CARB, 1997a). Exposure to ammonium nitrate has been found to cause burning or irritation of eye and skin (CARB, 1997a). Alternatively, as ETS ages, semi-volatile constituents of ETS, such as nicotine and neophytadiene, may shift from particulate phase to the gaseous phase.

Gaseous ETS components primarily react with the following:

- Sunlight through photolysis
- O_3 (ozone)
- OH radical during the daylight hours
- NO_3 radical during the nighttime hours
- Gaseous nitric acid (HNO_3)
- Nitrogen dioxide (NO_2)
- Hydroperoxy radical (HO_2) mainly during afternoon/evening hours

Important reaction processes for most gas-phase organic compounds are photolysis and subsequent reaction with O_3, as well as the OH and NO_3 radicals. For a few compounds, one or more of the other reactive chemical species (HO_2, NO_2, and/or HNO_3) may react at significant rates. For example, HO_2 radicals react with formaldehyde, acetaldehyde, and glyoxal, NO_2 reacts with conjugated dienes, and gaseous HNO_3 reacts with the amines. Table VI-1 provides examples of the atmospheric lifetimes and the dominant removal processes for some of the gaseous species found in ETS.

Table VI-1

Estimated Atmospheric Lifetimes of Selected ETS Constituents

	Dominant Removal Process	Atmospheric Lifetime
1,3 Butadiene	OH radical	2 hours[1/]
Acetaldehyde	OH radical	9 hours[1/]
Acrolein	OH radical	7 hours[1/]
Benzene	OH radical	10 days[2/]
Formaldehyde	Photolysis	4 hours
N-Nitrosodimethylamine	Photolysis	5 minutes
Toluene	OH radical	2.5 days
PAHs (gas phase)	OH radical	3-27 hours

Source: CARB, 1998.
1/ 12-hour average daytime (OH of 2.0x 10^6 molecule/cm^3)
2/ 1 day = 12-hour of OH of 2.0x 10^6 molecule/cm^3

Gaseous species absorbed by particles may be unavailable for further chemical reaction. Gaseous species adsorbed to particles may be degraded by photolysis and reaction with trospheric O_3, dinitrogen pentaoxide (N_2O_5), NO_2, HNO_3, nitrous acid (HNO_2), sulfuric acid (H_2SO_4), and hydrogen peroxide (H_2O_2).

B. ATMOSPHERIC REACTIONS OF PARTICULATE SPECIES

Particles in the range of 0.01-10 µm are often referred to as PM_{10}. Typically, all of the ETS associated particles fall in the range of between 0.01 and 1.0 µm (USEPA, 1992). ETS contains particulate species which have their own atmospheric persistence rates based on the particle size. The two most important processes affecting particle ETS species in the atmosphere are:

- Dry and wet deposition (i.e., physical removal) of particles, and
- Atmospheric transformations of species adsorbed to the particles.

Dry deposition is broadly defined as the transport of air pollutants from the atmosphere onto surfaces in the absence of precipitation (Davidson and Wu, 1989; Seinfeld and Pandis, 1998). Major factors affecting dry deposition are atmospheric turbulence, chemical, and physical properties of the air pollutants and the nature of the depositing

surface. Particles in the size range of 0.05 to 1 µm are expected to reside in the atmosphere for long time periods and can be transported over long distance (Cohen, 1998).

Virtually complete removal of particles in the range of 0.1 to 10 µm in diameter is expected by wet deposition (Leuenberger *et al*., 1985; Ligocki *et al*., 1985a, b). Since ETS particles are in this size range (0.1 – 1 µm), they are expected to be efficiently washed from the atmosphere by wet deposition. Wet deposition occurs due to events such as rain, cloud, fog, or snow.

C. NICOTINE

Nicotine is the principal alkaloid in tobacco and a major contributor to the addictive properties of tobacco. In ETS, studies report that nicotine is most commonly found in the gas phase within the environment (Eudy *et al*., 1986; Thome *et al*., 1986; Eatough *et at.*, 1986; Hammond *et al*., 1987). Organic compounds with vapor pressure between 10^{-6} and 10 Pascals (Pa) at ambient temperatures are classified as semi-volatile organic compounds. At 298 ºK, nicotine has a vapor pressure of 2.7 Pa and is almost entirely present in gas phase (Van Loy *et al.,* 2001). Less than five percent of ETS nicotine has been associated with the particulate phase (Jenkins *et al*., 2000). Also, in sidestream smoke, the alkaline nature of ambient air leads to gas phase nicotine rather than in the particulate phase.

The semi-volatile constituent of ETS, such as nicotine, exhibit different atmospheric persistency depends on environmental conditions. In ambient air, nicotine may react with photochemically generated hydroxyl radicals and with ozone. The reported half-life of nicotine in the ambient atmosphere is approximately 1 day (Spectrum Laboratories, Inc., 2003).

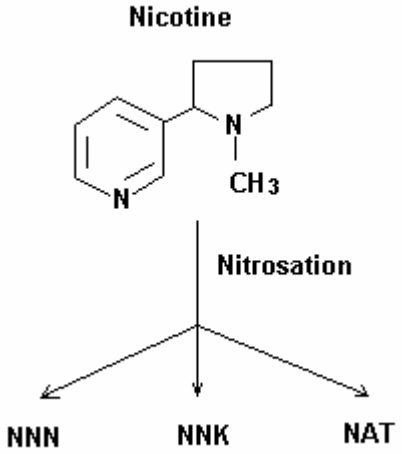
In indoor air, gas phase nicotine is rapidly diffuse to surrounding surfaces with which it interacts and expected to be removed from the environment at a faster rate than other ETS components (Eudy *et al*., 1986; Eatough *et al*., 1986). Studies show that the nicotine level decreases rapidly as consequence of sorptive uptake on different surrounding surfaces (Eatough *et al*., 1986; Piade *et al*., 1999; Von Loy *et al*., 2001). Therefore, nicotine is a reasonable indicator of ETS exposures occurring within the previous few hours, with its indoor half-life of approximately two hours (Trinh and Huynh, 1989). Research also indicates that sorbed nicotine present on surrounding materials, such as walls and carpets, may be re-emitted to the environment over time (Trinh and Huynh, 1989). According to the Piade *et al.* (1999) study, as much as 1 mg of nicotine can be adsorbed and re-emitted from 1 m^2 of cotton cloth over a few hours. The Van Loy *et al*. (2001) chamber experiments also observed desorption of nicotine from surrounding materials. After flash evaporation of nicotine in a 20 m^3 environmental test chamber with a carpet floor covering (measured nicotine air concentration of 4.4 µg/m^3), the chamber was flushed with clean air for three days. After resealing the chamber, the nicotine concentration slowly rose back to 1 µg/m^3, which demonstrates the effect of nicotine being re-emitted from surrounding material surfaces.

D. TOBACCO-SPECIFIC N-NITROSAMINES

While nicotine has not been identified as a carcinogen, several tobacco-specific nitrosamines (TSNAs), which are derived from nicotine and other tobacco alkaloids, may be carcinogenic (Hecht and Hoffmann, 1988). TSNAs (see Figure VI-1) are formed by N-nitrosation of nicotine during the curing, processing, fermentation, and combustion of tobacco products (IARC 1986; Ashley *et al*., 2003). The yield of TSNA from smoking depends on the nitrate content of tobacco. Certain flue-cured tobaccos exposed to NOx during the curing process contain higher levels of TSNAs (Ashley *et al*., 2003).

Figure VI-1

Nicotine Conversion



NNN : *N'*-nitrosonornicotine
NNK: 4-(methylnitrosamino-)-1-(3-pyridyl)-1-butanone
NAT: *N'*-nitrosoanatabine

N'-nitrosonornicotine (NNN) and 4-(methylnitrosamino-)-1-(3-pyridyl)-1-butone (NNK) are believed to be the most potent carcinogens of the TSNA class (Ashley *et al*., 2003). N-nitroso compounds are degraded in the presence of ultraviolet and visible light. When heated to decomposition, these compounds emit toxic fumes of nitrogen oxides (NTP, 2002).

E. PAH AND PAH-DERIVATIVES

Researchers have identified at least ten polycyclic aromatic hydrocarbon (PAH) isomers in ETS, which have been identified as cancer causing toxic air contaminants (Hoffmann and Hoffmann, 1997; OEHHA, 1997). Some PAHs react with NOx emissions in the atmosphere to mutate to form nitro-derivative PAHs (CARB and OEHHA, 1994). Both gas and particle phase PAHs have been measured in ETS (Gundel *et al*., 1995). Table VI-2 shows a list of identified gaseous and particulate PAHs that have been identified in ETS.

Table VI-2

PAHs Detected in ETS

Gas-Phase PAHs	Particle-Phase PAHs
1-methylnaphthalene	1,2-benzofluorene
2-methylnaphthalene	Anthracene
Anthracene[1/]	Benz[a]anthracene
Benz[a]anthracene[1/]	Benzo[a]pyrene
Chrysene[1/]	Benzo[b]fluoranthene
Fluoranthene[1/]	Benzo[k]fluoranthene
Fluorene	Chrysene
Naphthalene	Fluoranthene
Phenanthrene	Phenanthrene
Pyrene[1/]	Pyrene
	Triphenylene

Source: Gundel *et al*., 1995.

1/ PAHs are distributed between the gas and particle phases.

One of the most potent cancer-causing PAHs in ETS is benzo[a]pyrene. Benzo[a]pyrene exists almost entirely in the particle phase in the atmosphere with a size of three µm or less and, therefore, subject to wet and dry deposition (CARB, 1997b). The average half-life of particle benzo[a]pyrene in the atmosphere is estimated to be about 3.5 to 10 days and lifetime of 5 to 15 days (CARB, 1997b). Other lifetimes of PAHs are shown in Table VI-3.

Table VI-3

Estimated Atmospheric Lifetimes of Selected PAHs

PAHs in ETS	Lifetime due to reaction with:		
	OH [a/]	NO_3 [b/]	O_3 [c/]
1- methylnaphthalene	3.5 hrs	50 days	>125 days
2-methylnaphthalene	3.6 hrs	40 days	>40 days
Anthracene	1.4 hrs		
Fluoranthene	~3.7 hrs [d/]	~85 days	
Pyrene	~3.7 hrs [d/]	~30 days	

Source: CARB, 1998.

a/ For a 12-hr daytime average OH radical concentration of 1.5×10^6 molecule cm^{-3} (Prinn *et al*., 1987).

b/ For a 12-hr average nighttime NO_3 radical concentration of 2.4×10^8 molecule cm^{-3} and an NO_2 concentration of 2.4×10^{12} molecule cm^{-3} (Atkinson, 1985).

c/ For a 24 hr average O_3 concentration of 7×10^{11} molecule cm^{-3} (Logan, 1985).

d/ Using estimated OH radical reaction rate constant correlation with ionization potential (Biermann *et al*., 1985; Arey *et al*., 1990; Atkinson, 1990).

Volatile, 2- to 4-ring PAHs exist in the atmosphere mostly in the gas phase (Atkinson and Arey, 1994). The gas-phase PAHs react with hydroxyl (OH) radicals, NO_3 radicals, and ozone in the atmosphere, with the OH radical reaction generally dominating as the PAHs loss process (Atkinson and Arey, 1994). The products of the OH radical reactions with PAHs include formation of hydroxyl-PAH, nitro-PAH, and ring-opened dicarbonyls (CARB, 1997b). The estimated half-life of the gas phase 2- and 4-ring volatile PAHs in the atmosphere due to reaction with the OH radical are in the range of 2 to 19 hours and have a lifetime of 3 to 27 hours, (Atkinson and Arey, 1994).

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APPENDIX A

List of Known ETS Constituents

As Approved
by the Scientific Review Panel
on June 24, 2005

Appendix A

List of Known ETS Constituents

Constituent	Reference
γ-Butyrolactone	3,4,8
β-Carboline	3
β-Carotene	3
α-Ketoglutaric acid	5
β-Methylvaleric acid	5
β-Phenethyl alcohol	5
γ-Sitosterol	5
β-Sitosterol	5
α-Socratine	5
β-Socratine	5
γ-Socratine	5
1,12-Benzoperylene	5
1,1-Dimethylhydrazine	6,7
1,2,4-Trimethylbenzene	5
1,2-3,4-5,6-Tribenzanthracene	5
1,2-3,4-Dibenzopyrene	5
1,2-5,6-Dibenzanthracene	5
1,2-7,8-Dibenzoflourene	5
1,2-7,8-Dibenzonaphthacene	5
1,2-Benzanthracene	5
1,2-Benzofluorene	2,5
1,2-Benzonaphthacene	5
1,2-Benzopyrene	5
1,3,5-Trimethylbenzene	5
1,3,5-Trimethylbenzene	5
1,3,5-Trimethylbenzene	5
1,3-Butadiene	1,5,6,7
1,3-Dimethoxypyrogallol	3
1,8,9-Perinaphthoxanthene	5
1,8-Dimethylnaphthalene	5
1,8-*p*-Menthadiene	5
11,12-Benzofluoranthene	5
1-Aminonaphthalene	1
1-Azafluororanthene	3
1-Azapyrene	3
1-Methylchrysene	5
1-Methylnaphthalene	2
1-Methylpyrene	5
1-Naphthol	3,5

1-Naphthylamine	3,7
2- Aminonaphthalene	1
2,1-Naphtho-1,2-fluorene	5
2,3'-Bipyridyl	3
2,3-Benzofluorene	5
2,3-Butanedione	5
2,3-Dimethylaniline	3
2,3-Dimethylmaleic anhydride	3
2,3-Dimethylpyrazine	3
2,3-Pentanedione	5
2,4-Dimethylaniline	3
2,4-Lutidine	3
2,4-Xylenol	5
2,5-Dimethylaniline	3
2,5-Dimethylphenanthrene	5
2,5-Lutidine	3
2,6-Dimethylaniline	3,6
2,6-Dimethylpyridine	5
2,6-Lutidine	3
2',3'-Naphtho-3,4-pyrene	5
2-Aminobiphenyl	3
2-Ethylaniline	3
2-Methyl-1-naphthylamine	3
2-Methylanthracene	5
2-Methylfuran	5
2-Methylnaphthalene	2,5
2-Methylpyridine	3,5
2-Naphthol	3,5
2-Naphthylamine	3,4,6,7,8
2-Nitropropane	1,3,6,7
2-Picoline	3
2-Toludine	1,4,6,7,8
2-Vinylphenol	3
3,4-8,9-Dibenzopyrene	5
3,4-9,10-Dibenzopyrene	5
3,4-Benzofluoranthene	5
3,4-Benzopyrene	5
3,4-Dihydro-3,4-benzopyrene	5
3,5-Xylenol	5
3-Aminobiphenyl	1,3
3-Ethenylpyridene	1
3-Ethylaniline	3
3-Hydroxyisoeugenol	3
3-Methyipyridine	8
3-Methyl-1,2-benzanthracene	5

3-Methylcatechol	3
3-Methylpyrene	5
3-Methylpyridine	3,4,5
3-Picoline	3
3-Pyridyl ethyl ketone	5
3-Pyridyl methyl ketone	5
3-Pyridyl propyl ketone	5
3-Vinylphenol	3
3-Vinylpyridine	3,4,6,8
4-Aminobiphenyl	1,3,4,6,7,8
4-Azafluorene	3
4-Ethylcatechol	3
4-Methylcatechol	3
4-Methylpyrene	5
4-Picoline	3
4-Vinylcatechol	3
4-Vinylguaiacol	3
4-Vinylpheno	3
5,6-Cyclopentenobenzanthracene	5
5-Methylchrysene	1,3,6
6,7-Cyclopentenobenzanthracene	5
7,8-Benzofluoranthene	5
7H-Dibenzo[c,g]carbazole	1,3,5,7
8,9-Benzofluoranthene	5
8-Methylfluorene	5
9,10-Dimethyl-1,2-benzanthracene	5
9-Methyl-1,2-benzofluorene	5
9-Methylfluorene	5
9-Methylphenanthrene	5
A fluorenecarboxyoic acid	5
AaC	6
Acenaphthene	2,5
Acenaphthylene	2,5
Acetaldehyde	1,3,5,6,7
Acetamide	6,7
Acetic acid	3,4,5,6,8
Acetone	1,3,4,5,6,8
Acetylene	5,6
Acridine	3
Acrolein	1,3,4,5,6,7,8
Acrylamide	6
Acrylonitrile	1,6,7
Adipic acid	5
Aluminum	5
Ammonia	1,3,4,5,6,8

Anabasine	3,5,6
Anatabine	3,4,5,6,8
Aniline	4,6,7,8
Anodmine	5
Anthanthrene	5
Anthracene	1,2,3,5
Anthraceno-2,3-9,10-phenanthrene	5
Arachidic acid	5
Argon	6
Arsenic	1,5,6,7
Azulene	5
Benz[a]acridine	3
Benz[c]acridine	3
Benz[f]indene	3
Benzaldehyde	5
Benzene	1,3,4,5, 6,7,8
Benzimidazole	3
Benzo[a]pyrene	1,2,3,4,5,7,8
Benzo[a}anthracene	1,2,3,4,7,8
Benzo[b]fluoranthene	1,2,3,7
Benzo[b]fluorene	3
Benzo[b]furan	3,6
Benzo[c]fluorene	3
Benzo[c]phenanthrene	3
Benzo[e]pyrene	3
Benzo[f]quinoline	3
Benzo[ghi]perylene	3
Benzo[h]quinoline	3
Benzo[j]fluoranthene	1,3,7
Benzo[k]fluoranthene	1,2,3,7
Benzo[m,n,o]fluoranthene	5
Benzoic acid	3,4,5,8
Benzophenanthrene	5
Benzyl alcohol	5
Beryllium	1,6
Butane	5
Butylbenzene	5
Butyraldehyde	1,5
Butyric acid	5
C25-C33 paraffins	5
Cadmium	1,4,5,7,8
Caffeic acid	3,6
Calcium	5
Campesterol	3,6
Caproic acid	5

Caprylic acid	5
Captan	7
Carbazole	3
Carbon dioxide	3,4,5,6,8
Carbon monoxide	1,3,4,5,6,7,8
Carbon oxysulfide	5
Carbonyl sulfide	3,4,8
Catechol	1,3,4,5,6,8
Cerotic acid	5
Chlorinated dioxins and furans	1
Chlorogenic acid (3-o-caffeoyl-d-quinic acid)	3
Cholesterol	4,6,8
Chromium VI	1,5,6,7
Chrysene	1,2,3,5,7
Cichoriin	3
Cobalt	6
Collidine	5
Copper	5
Coronene	3,5
Cotinine	3,5
Coumarin	3
Crotonaldehyde	1,3,5
Cyanogen	5
Cycloartenol	3
Dibens[a,j]anthracene	3
Dibenz[a,,j]acridine	3,7
Dibenz[a,c]anthracene	3
Dibenz[a,h]acridine	1,3,7
Dibenz[a,h]anthracene	3,7
Dibenz[a,j]acridine	1,3,7
Dibenzo[a,e]fluoranthene	3
Dibenzo[a,e]pyrene	7
Dibenzo[a,h]pyrene	3,7
Dibenzo[a,i]pyrene	1,3,7
Dibenzo[a,l]pyrene	3,7
Dibenzo[b,d]furan	3
Dibenzo[c,g]carbazole	6
Diethyl ketone	5
Diethylene glycol	5
Dimethylamine	5
Dimethylchrysene	5
Dimethylfluoranthene	5
Dimeyhtlamine	4,5,8
Dipentene	5
Dipropyl ketone	5

Ergosterol	3
Esculetin	3
Ethane	5
Ethanol	5
Ethyl β-methylvalerate	5
Ethyl acetate	5
Ethyl carbamate	6
Ethyl isovalerate	5
Ethyl *n*-butyrate	5
Ethyl *n*-caproate	5
Ethyl propionate	5
Ethylamine	5
Ethylbenzene	1
Ethylene	5
Ethylene glycol	5
Ethylene oxide	6
Ethylphenols	3
Eugenol	3
Ferulic acid	3
Fluoranthene	3,6
Fluoranthene	2,5
Fluorene	2,3,5
Formaldehyde	1,3,4,5,6,8
Formic acid	3,4,5,6,8
Furan	5,6
Furfural	5
Furoic acid	5
Glu-P-1	6
Glu-P-2	6
Glutamic acid	5
Glutamine	5
Glutaric acid	5
Glycerol	5,6
Glycolic acid	3,4,8
Guaiacol (2-Methoxyphenol)	3,5
Gudham	5
Harman (1-methyl-β-carboline)	4,8
Heptylic acid	5
Hydrazine	1,3,4,6,8
Hydrogen cyanide	1,3,4,5,6,8
Hydrogen sulfide	5,6
Hydrogen thiocyanide	5
Hydroquinone	1,3,5,8
Indeno[1,2,3-cd]pyrene	1,3,6
Indole	3,6

Ionene	3
IQ	6
Iron	5
Isobutane	5
Isobutylene	5
Isobutyraldehyde	5
Isobutyric acid	5
Isoeugenol	3
Isoprene	1,5,6
Isopropylbenzene	5
Isoquinoline	3
Isosqualene	5
Lactic acid	3,4,5,6,8
Lathrein	5
Lauric acid	5
Lead	1,5,6,7
Levantenolide	3
Levulinic acid	5
Limonene	6
Linoleic acid	3,5,6
Linolenic acid	3,5,6
Lohitam	5
Lutidine	5
Magnesium	5
Maleic anhydride	3
Maleic hydrazide	6
Malic acid	5
Malonic acid	5
Manganese	5
m-Cresol	1,3,5
Mercury	1
Mesitol	5
Methane	5,6
Methanol	3,5,6
Methyl acetate	5
Methyl chloride	4,5,8
Methyl ethyl ketone	1,5
Methyl formate	6
Methyl nitrate	5
Methylacetylene	5
Methylamine	4,5,6,8
Methyleugenol	6
Methylglyoxal	5
m-Hydroxyacetophenone	5
m-Toluidine	3

Myosmine	3,5
Myristic acid	5
N'-Nitrosoanabasine	1,3
N'-Nitrosoanatabine	1,3
N'-Nitrosonornicotine	1,3,4,6,7,8
Naphthalene	2,5,6
Naphtho[2,3-b]pyrene	3
Neophytadiene	3
n-Hentriacontane	6
Nickel	1,4,5,6,7,8
Nicotinamide	5
Nicotine	1,3,4,5,6,7,8
Nicotine-N'-oxid	3
Nicotinic acid	5
Nicotrine	3
Nicotyrine	5
Nitrobenzene	6
Nitrogen oxides	3,4,6,8
Nitromethane	6
N-Methylmyosmine	5
N-Methylpyrrolidine	6
N-Nitrosodiethanolamine	1,3,4,7,8
N-Nitrosodiethylamine	1,3,4,6,7,8
N-Nitrosodimethylamine	1,3,4,6,8
N-Nitroso-di-n-butylamine	1,3,6,7
N-Nitrosodi-n-propylamine	3,6
N-Nitrosoethylmethylamine	1,3,6
N-Nitroso-n-methylethylamine	3,7
N-Nitrosopiperidine	3,6,7
N-Nitrosopyrrolidine	1,3,4,6,7,8
NNK 4-(N-methyl-N- nitrosamino)-1-(3-pyridyl)-1-butanone	1,3,4,6,8
Nonylic acid	5
Nornicotine	3,5
Nornicotyrine	3,5
Norphytene	3
o-Anisidine	7
Obeline	5
o-Cresol	1,3,5
Oleic acid	3,6
Oleic acid	5
o-Toluidine	3
Oxalic acid	5
Palmitic acid	3,5,6
Palmitoleic acid	5

Palmitone	5
p-Cresol	1,3,5
Perylene	3,5
Phenanthrene	2,3,5
Phenanthridine	3
Phenol	1,3,4,5,6,8
Phenylacetylene	5
PhlP	6
Phthalic acid	5
P-Hydroxyacetophenone	5
Phytadienes	5
Phytol	3
Phytone	3
Picoline	5
Plastoquinone	3
Poikiline	5
Polonium-210	3,4,6,8
Potassium	5
Propane	5
Propionaldehyde	1,5
Propionic acid	5,6
Propylbenzene	5
Propylene	5
Propylene oxide	5,6
p-Toluidine	3
Pyndine	6
Pyrene	3,5
Pyridine	1,3,4,5,6,8
Pyridine-3-aldehyde	5
Pyrrole	1,5,6
Pyrrolidine	6
Pyrrolo[2,3-b]pyridine	3
Pyruvic acid	5
Quinoline	1,3,4,5,6,8
Quinoxaline	3
Reductic acid	5
Resin acid	5
Resorcinol	1,3,5
Scopoletin	3,5,6
Scopoletin-β-gentiobioside	3
Scopolin	3
Sitosterol	3,6
Skatole	6
Sodium	5
Solanesenes	3

Solanesol	3,5,6
Solanone	3
Squalene	3,5
Stearic acid	3,5,6
Stigmasterol	3,5,6
Strontium	5
Styrene	1,6,7
Succinic acid	3,4,5,8
Succinic anhydride	3
Thiocyanogen	5
Titanium	5
Toluene	1,3,4,5,6,7,8
Triethylene glycol	5
Trimethylamine	5
Triphenylene	2,3
Trp-P-1	6
Trp-P-2	6
Urethane	1,3,7
Veleric acid	5
Vinyl chloride	1,3,6,7
Xylenes	1
Xylenols	3
Zinc	4,5,8

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APPENDIX B

ETS Emissions Calculation Methodology

As Approved
by the Scientific Review Panel
on June 24, 2005

Overview

ETS is a complex mixture of compounds and it would be difficult and impractical to quantify emissions based on individual compounds. We are unaware of any studies that quantify ETS emissions based on the sum of all individual compounds. Adequate analytical methods do not exist for some suspected compounds in ETS, and the cost of sampling and analysis would be high. Therefore, staff selected three compounds to characterize ETS emissions: nicotine, respirable suspended particulate (RSP), and carbon monoxide (CO). These compounds all have specific health effects associated with their exposures and have been used as markers for ETS exposure.

Nicotine emissions are unique to tobacco products and have been linked to health effects (Benowitz, 2002). Particulate matter emissions from tobacco products have been linked to respiratory problems, such as asthma, and the development or exacerbation of cardiovascular disease (Smith and Fischer, 2001). Likewise, CO has also been linked to cardiovascular and birth weight effects (Horner, 2000).

Methodology

In general, our estimate of ETS emissions is based on data from emission rate studies and tobacco product sales tax data compiled by the California State Board of Equalization (CBOE). For purposes of this estimate, we assumed that cigarette consumption among the smoking population was uniform.

Limited data exists on pipe tobacco emissions and consumption information indicates that pipe tobacco consumption is far less than for cigarettes and cigars (USDA, 2003a). Therefore, staff based the ETS emission estimate predominantly on cigarette and cigar consumption. The estimate of ETS emissions is based on the following equation:

$$\text{Emissions (tons/yr)} = [EF \times N \times CF \times 90\%] \qquad \text{(Equation 1)}$$

Where: EF = Average cigarette or cigar emission factor (mg/cig)
N = Number of cigarettes or cigars per year (cig/yr)
CF = Unit conversion factor (tons/mg)

We adjusted the number of cigarettes and cigars by 90% to account for the finding that smokers do not typically consume one hundred percent of a cigarette. In a study measuring mass emission rates from cigarettes, Hildemann, *et al.,* 1991, found that smokers consumed approximately 90% of cigarettes and cigars.

Assumptions Used to Estimate Outdoor ETS Emissions

As previously mentioned in Chapter IV, there is limited information pertaining to direct measurements of indoor vs. outdoor cigarette consumption in California -- making it difficult to accurately determine. However, other germane information can assist staff in estimating outdoor ETS emissions. Outdoor ETS emissions include direct emissions from outdoor smoking, plus ETS emissions generated indoors, which eventually ventilate outside. Since 1998, under Assembly Bill 13, all workplaces (including bars and restaurants) are smoke-free in California. In addition, smoking behavior has changed as well. Based on the 2002 California Adult Tobacco Survey (CATS), over 80% of all California homes with children are smoke-free. For California smokers, 50% have reported smoking bans in their homes. Therefore, with no indoor smoking in workplaces and other public venues, and indoor smoking bans in half of all California residences with a smoker, we assume that most physical smoking occurs outdoors. Furthermore, for ETS generated indoors, building ventilation studies show that 50 - 80% of indoor air gets exchanged with outdoor air (Rogge *et al.*, 1994).

Next, we made assumptions as to what a typical smoking adult lifestyle entails. For instance, an adult might work 60% of the day and spend 40% of the day at home (not including sleeping hours). According to the 2002 CATS, the average smoker in California consumes 15-cigarettes per day and either has a home smoking ban or no home smoking ban (50% of California smokers have reported a home smoking ban). From this information, we developed two smoking adult lifestyle scenarios to provide insight on the relative amounts of indoor vs. outdoor ETS emissions (Table B-1).

Table B-1

Cigarette Consumption Based on Adult Lifestyles
(15 cigarettes per day)

Adult Lifestyle 1 (Home Smoking Ban)				Adult Lifestyle 2 (No Home Smoking Ban)			
* % of Time at Work	Cigarettes Consumed at Work	* % of Time at Home	Cigarettes Consumed at Home (Outside/Inside)	* % of Time at Work	Cigarettes Consumed at Work	* % of Time at Home	Cigarettes Consumed at Home (Outside/Inside)
60	9	40	6 / 0	60	9	40	** 3 / 3

* Percent of non-sleeping hours.
** Based on 50% ventilation.

For Adult Lifestyle 1 (home smoking ban), all 15-cigarettes are smoked outdoors, since no smoking is allowed in the workplace or in the home. This amounts to 100% outdoor ETS emissions. However, for Adult Lifestyle 2 (no home smoking ban), emissions from 12 of 15 cigarettes (80%) consumed are estimated outdoor ETS emissions. This assumes a 50% ventilation rate from indoors to outdoors. In the time spent at home, we assume six cigarettes per day are smoked indoors (15 cigarettes x 0.4 = 6), although, smoking rates may vary throughout the day. All six cigarettes are assumed to be

smoked inside the home, however, 50% of the emissions, or essentially the emissions from three cigarettes, are assumed to ventilate outdoors. Therefore, staff estimates at least 80 - 90% of cigarette emissions are outdoor emissions.

Cigarette Emission Factors

Staff conducted a literature search to review the research on cigarette emission factors for nicotine, RSP, and CO. The search found five-studies on nicotine emission rates, six on RSP, and three on CO. The most pertinent studies are shown in the following tables. While the studies evaluated emissions from major national cigarette and cigar brands, the results are applicable to California since many of the same brands are also marketed in the state.

Table B-2 shows the results found for nicotine emission factors from three studies, where the average nicotine emission rate was 1.44 milligrams per cigarette (mg/cig). Martin *et al*. (1997) chose the top fifty U.S. market brand styles (determined by market share) and a national average cigarette (Kentucky Research-K1R4F). Nicotine emissions were reported in relation to the mainstream (MS) tar content of the cigarette. The fifty top selling cigarettes represented over 65% of the U.S. cigarette market and included full flavor (FF) (≥ 13.5 mg/cig MS tar), full flavor low tar (FFLT) (7.5 - 13.4 mg/cig MS tar), and ultra low tar (ULT) (≤ 7.4 mg/cig MS tar) cigarettes. Their results showed a 0.1 mg mean difference among all cigarette types.

Table B-2

Nicotine Emission Factor Studies

Study #	Authors	Emission Factor
1	Martin *et al*. (1997)	1.59 mg/cig
2	Daisey *et al*. (1998)	0.92 mg/cig
3	Nelson (1994)	1.8 mg/cig
Avg.		**1.44 mg/cig**

Daisey *et al*. (1998) determined the emission factors of six major cigarette brands smoked in California and a national average cigarette (Kentucky Research-K1R4F). The six major brands represented a market share of over 63% in 1990, and included five filtered and one unfiltered brand; two were mentholated and one brand was low tar. The nicotine emission factors for all six brands showed a coefficient of variability of over 26% (0.92 ± 0.24 mg/cig). In Nelson (1994), the top 50 brands of cigarettes were analyzed for emissions generated by a person in an unventilated room.

Table B-3 is a summary of the pertinent studies on RSP emissions. From five studies, the average RSP emission rate was 13.3 mg/cig. Repace (2001) based his RSP

emission factors (i.e., 14 and 10.9 mg/cig) on a habitual smoker model that utilizes different numbers of smokers per unit volume.

Table B-3

RSP Emission Factor Studies

Study #	Authors	Emission Factors
1	Repace (2001)	14 mg/cig
2	Nelson *et al.* (1997)	14 mg/cig
3	Martin *et al*. (1997)	13.7 mg/cig
4	Nelson (1994)	13.8 mg/cig
5	Repace (2001)	10.9 mg/cig
Avg.		**13.3 mg/cig**

Nelson *et al*. (1997) generated ETS in an environmental chamber in which five replicate runs were performed, while six smokers each smoked one popular "light" cigarette. RSP yields were determined using the method in Martin et al. (1997), which draws in air at 2 liters/min with a personal sampling pump through a 1.0-µm pore membrane filter.

Martin *et al*. (1997) found a range in RSP emission rate from 10.5 mg/cig for ULT to 14.9 mg/cig for FF, with an average of 13.7 mg/cig among the three MS tar cigarette categories. Nelson (1994) reported an average RSP emission factor of 13.8 mg/cig.

Table B-4 is a summary of the two studies on CO. Nelson *et al*. (1997) determined a CO emission factor of 61.9 mg/cig by non-dispersive infrared gas analysis (cf. Martin *et al*., 1997). Martin *et al*. (1997) reported CO emission rates of 47.8 mg/cig for ULT to 57.5 mg/cig for CO for FF, with an average of 55.1 mg/cig among the three MS tar categories. The average CO emission factor from the two studies is 58.5 mg/cig**.**

Table B-4

CO Emission Factor Studies

Study #	Authors	Emission Factors
1	Nelson *et al.* (1997)	61.9 mg/cig
2	Martin *et al.*, 1997	55.1 mg/cig
Avg.		**58.5 mg/cig**

Cigar Emission Factors

Staff conducted a literature search on cigar emission factor studies for nicotine, RSP, and CO. Three studies were found: one for nicotine, one for RSP, and two for CO.

For nicotine, premium (i.e., large) cigars were smoked under test conditions established by the International Committee for Cigar Smoke Study (ICCSS) (Hoffmann and Hoffmann, 1997). The ICCSS specifies that one 20-milliliter (mL) volume puff be taken within a 1.5-second interval every 40 seconds, using a standardized smoking machine. An average emission factor was determined after three runs. For small cigars, the cigarette-smoking parameters of the Federal Trade Commission were followed, in which one 35-mL puff is taken within a 2-second duration every minute, using a standardized smoking machine. The nicotine emission factors for small and large cigars are 3.8 and 13.3 mg/cigar, respectively.

For RSP, data from Repace *et al*. (1998) were evaluated in which three experiments were conducted. In the first experiment, one Santona cigar was smoked by a person in a 97 m^3 parlor for 1.3 hours. The number of air changes per hour (ach) was 2.5. For this cigar, the RSP emission factor was 78 mg/cigar. In the second experiment, a Paul Garmirian cigar was smoked by a person in a 97 m^3 parlor for 1.5 hours with an ach of 1.2. For this cigar, the RSP emission factor was 86 mg/cigar. In the third experiment, a Marsh Wheeling Stogie was smoked by a person in a 51 m^3 office for 20 minutes with an ach of 3.8. The emission factor for this cigar was 53 mg/cigar. The average RSP emission factor from these three experiments was 72 mg/cigar.

For CO, an emission factor was derived from two studies: Repace *et al*., (1998), and Klepeis *et al*., (1999). Over 13 different experiments were conducted in the two studies. A summary of the experimental parameters are in Table B-5. The overall average CO emission rate was 1,025 mg/cigar.

Table B-5

Experimental Parameters for Cigar CO Emission Factors (Source: Repace *et al*. (1998) and Klepeis *et al.* (1999))

Cigar Brand	Machine or Person	Cigar Duration (min)	Air Exchange Per Hour	Volume of Testing Area (m^3)	Emission Factor (mg/cigar)
Santona	Person	76	2.5	97	1,100
Marsh Wheeling Stogie	Person	20	3.8	51	1,140
N/A	Machine	11	7.2	521	1,200
N/A	Machine	11	7.2	521	1,300
Sante Fe Fairmount	Machine	20	2.1	49.6	1,200
Imported Ashton	Machine	28	1.8	49.6	1,200
Swisher Sweets	Machine	42	0.96	49.6	980
Dutch Masters El Presidente	Machine	9	0.06	49.6	750
Antonio y Cleopatra Grenadiers	Machine	17	3.0	49.6	630
Sante Fe Fairmont	Machine	7.8	4.5	49.6	1,100
Sante Fe Fairmont	Machine	24	0.12	49.6	1,100
Antonio y Cleopatra Grenadiers	Machine	10	0.12	49.6	860
Antonio y Cleopatra Grenadiers	Machine	12	4.5	49.6	780

Number of Cigarettes and Cigars

To calculate the number of cigarettes smoked in California, data from CBOE, which maintains a statewide inventory of annual cigarette pack distributions, were used. The CBOE collects taxes at the point of distribution from certified vendors, who may conduct business in multiple counties. Distribution is defined as: “the sale or use or the placing of cigarettes in retail stock for the purpose of selling the cigarettes to consumers” (Revenue & Taxation Code sections 3001-30018). Thus, taxes are incurred at the wholesale level. To estimate statewide emissions, we assumed that distribution represented actual consumption, as consumers generally do not maintain large

inventories. In fiscal year 2001-02, the CBOE reported that over 1.27 billion packs of cigarettes were distributed in California. Since the average cigarette pack contains 20 cigarettes, the total number of cigarettes distributed in California was calculated to be 25.4 billion (i.e., total cigarettes = (20 cigarettes/pack x 1.27 billion packs)).

In 2002, the U.S. Department of Agriculture (USDA) estimated that smokers in the U.S. consumed 4.1 billion large cigars (10% increase vs. 1998), and 2.2 billion small cigars (28% increase vs. 1998) (USDA, 2003b). While the USDA, does not compile California-specific cigar inventories, California accounts for 6% of nationwide cigarette sales. On this basis, staff estimated that the number of large and small cigars smoked in California to be 247-million (6% of 4.1 billion) and 135-million (6% of 2.2 billion), respectively.

Statewide ETS Emissions Inventory

Using the methodology described above, staff estimated total statewide ETS emissions for nicotine, RSP, and CO. Table B-6 shows our estimates of statewide emissions.

Table B-6

2002 California Statewide ETS Emissions (Tons/Year)

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

[a] Staff estimates 80 - 90% of total emissions reside outdoors.

Countywide emissions were also calculated using Equation 1 (see p. B-1) adjusted for the total number of cigarettes smoked per county (i.e., percent of total California smokers per county multiplied by the total number of cigarettes). Attachment A presents our estimated emission results by county.

Emissions by Age

We also estimated ETS emissions amongst two age groups: adults (18 years and older) and adolescents (12-17 years of age). These two age groups comprise virtually all smokers, with adults accounting for about 95% of all California smokers.

For this analysis, we used data from the Tobacco Control Section of the California Department of Health Services (CDHS). Under Proposition 99 (The Tobacco Initiative), CDHS routinely conducts surveys to determine the prevalence of smoking in California. Specifically, we used smoking prevalence data from the 2002 Adult California Tobacco Survey (CTS) and the 2001 Adolescent California Student Tobacco Survey (CSTS) in

Attachment B. The number of smokers (adult or adolescent) per county was calculated using 2002 population data for each county, multiplied by the established smoking prevalence for the county or region, as follows:

No. Smokers per County = [County Population x County Smoking Prevalence]

In 2002, we estimate the number of adult and adolescent smokers in California to be over 4.2 million and 400,000, respectively.

The number of cigarettes smoked per county was calculated by taking the number of smokers (adults and adolescents) in each county as a statewide percentage, then multiplying by the total number of cigarettes smoked statewide, as follows:

No. Cigarettes per County = [Smokers per County (%) x Total Cigarettes Statewide]

A complete summary of estimated total smokers and cigarettes in each county or region is in Attachment C.

In Table B-7, the total adult and adolescent cigarette emissions of nicotine, RSP, and CO in California were estimated to be 36.4, 335, and 1,476 tons/yr, respectively.

Table B-7

Estimated Adult and Adolescent Cigarette Emissions of Nicotine, RSP, and CO (Tons/Year)

	Adult (18+)	Adolescent (12-17)	[a] Total
Nicotine	32.9	3.5	36.4
RSP	303	32	335
CO	1,335	141	1,476

[a] Staff estimates 80 - 90% of total emissions reside outdoors.

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Attachment A

2002 Estimated Adult and Adolescent Cigarette ETS Emissions Per California County or County Region (lbs/year)

Region	[a] Combined Adult & Adolescent		
	Nicotine	RSP	CO
Los Angeles	19,724	182,173	801,286
San Diego	5,677	52,433	230,628
Orange	5,394	49,817	219,119
San Bernardino	4,124	38,120	167,672
Riverside	4,116	38,012	167,194
Fresno, Madera, Merced, Stanislaus	3,978	36,204	159,246
Imperial, Inyo, Kern, Kings, Mono, Tulare	3,345	30,897	135,899
Alpine, Amador, Calaveras, El Dorado, Mariposa, Nevada, Placer, San Joaquin, Sierra, Sutter, Tuolumne, Yuba	3,299	30,454	133,959
Alameda	2,947	27,215	119,704
Sacramento	2,871	26,519	116,645
Butte, Colusa, Del Norte, Glenn, Humboldt, Lake, Lassen, Mendocino, Modoc, Plumas, Shasta, Siskiyou, Tehama, Trinity, Yolo	2,784	25,726	113,155
Santa Clara	2,676	24,712	108,696
San Luis Obispo, Santa Barbara, Ventura	2,605	24,064	105,845
San Mateo, Solano	2,164	19,985	87,904
San Francisco	1,923	17,757	78,103
Contra Costa	1,825	16,858	74,152
Marin, Napa, Sonoma	1,739	16,061	70,645
Monterey, San Benito, Santa Cruz	1,495	13,809	60,737

[a] Staff estimates 80 - 90% of total emissions reside outdoors.

Attachment B

The following table illustrates the adult and adolescent smoking prevalence within California regions in 2002. The data for these tables can be found from the County and Statewide Archive of Tobacco Statistics at http://webtecc.etr.org/cstats/ .

2002 Adult and Adolescent Smoking Prevalence by Region Within California

Region	Adult (%)
Los Angeles	16.0 (±0.8)
San Diego	15.1 (±1.2)
Orange	14.3 (±1.3)
Santa Clara	12.3 (±1.3)
San Bernardino	19.3 (±1.4)
Alameda	15.8 (±1.5)
Riverside	20.3 (±1.4)
Sacramento	17.6 (±1.4)
Contra Costa	13.7 (±1.4)
San Francisco	17.9 (±1.6)
San Mateo, Solano	14.8 (±1.4)
Marin, Napa, Sonoma	15.3 (±1.5)
Butte, Colusa, Del Norte, Glenn, Humboldt, Lake, Lassen, Mendocino, Modoc, Plumas, Shasta, Siskiyou, Tehama, Trinity, Yolo	19.5 (±1.5)
San Luis Obispo, Santa Barbara, Ventura	13.7 (±1.3)
Alpine, Amador, Calaveras, El Dorado, Mariposa, Nevada, Placer, San Joaquin, Sierra, Sutter, Tuolumne, Yuba	17.7 (±1.4)
Monterey, San Benito, Santa Cruz	15.9 (±1.5)
Fresno, Madera, Merced, Stanislaus	19.3 (±1.4)
Imperial, Inyo, Kern, Kings, Mono, Tulare	19.9 (±1.5)

Region	Adolescent (%)
Los Angeles	14.4 (±3.9)
San Diego	18.3 (±2.9)
Orange	15.0 (±2.7)
Santa Clara	13.7 (±2.0)
San Bernardino	14.5 (±3.8)
Alameda	11.4 (±4.3)
Riverside	13.7 (±3.5)
Sacramento, San Joaquin, Stanislaus, Yolo, Yuba	16.6 (±4.3)
Contra Costa, Marin, San Francisco, San Mateo, Solano	18.9 (±4.4)
Fresno, Imperial, Kern, Kings, Madera, Mariposa, Merced, Tulare	16.8 (±3.1)
Monterey, San Benito, San Luis Obispo, Santa Barbara, Santa Cruz, Ventura.	19.2 (±4.0)
Alpine, Amador, Butte, Calaveras, Colusa, Del Norte, El Dorado, Glenn, Humboldt, Inyo, Lake, Lassen, Mendocino, Modoc, Mono, Napa, Nevada, Placer, Plumas, Shasta, Sierra, Siskiyou, Sutter, Sonoma, Tehama, Trinity, and Tuolumne.	18.6 (±5.9)

Attachment C

2002 Estimated California County Information Regarding Population, Smokers, and Cigarettes

County	Population (age 12+)	Smokers	Smoker %	Cigarettes	County	Population (age 12+)	Smokers	Smoker %	Cigarettes
Alameda	1,220,022	187,823	4.06	1,031,274,433	Orange	2,392,579	343,813	7.43	1,887,764,881
Alpine	1,054	187	0.004	1,028,072	Placer	233,056	41,468	0.90	227,685,517
Amador	32,483	5,775	0.12	31,710,818	Plumas	18,237	3,540	0.08	19,438,077
Butte	177,815	34,521	0.75	189,541,487	Riverside	1,335,738	262,339	5.67	1,440,418,884
Calaveras	37,394	6,652	0.14	36,526,234	Sacramento	1,045,404	183,024	3.95	1,004,922,459
Colusa	15,494	3,003	0.06	16,489,793	San Benito	43,083	7,006	0.15	38,467,153
Contra Costa	816,686	116,349	2.51	638,833,408	San Bernardino	1,401,270	263,089	5.68	1,444,534,034
Del Norte	23,358	4,533	0.10	24,889,929	San Diego	2,354,432	361,871	7.82	1,986,916,617
El Dorado	139,742	24,869	0.54	136,548,878	San Francisco	682,900	122,549	2.65	672,878,091
Fresno	658,381	124,995	2.70	686,304,253	San Joaquin	480,685	84,516	1.83	464,050,153
Glenn	21,489	4,166	0.09	22,871,408	San Luis Obispo	216,343	30,504	0.66	167,487,083
Humboldt	108,782	21,121	0.46	115,967,477	San Mateo	583,632	88,148	1.90	483,990,274
Imperial	117,340	22,885	0.49	125,655,482	Santa Barbara	330,086	46,684	1.01	256,328,483
Inyo	15,598	3,083	0.07	16,929,654	Santa Clara	1,374,113	170,552	3.68	936,442,457
Kern	547,837	106,898	2.31	586,941,956	Santa Cruz	211,008	34,112	0.74	187,299,820
Kings	108,712	21,263	0.46	116,747,380	Shasta	142,217	27,613	0.60	151,615,865
Lake	52,691	10,226	0.22	56,147,122	Sierra	3,040	540	0.01	2,966,634
Lassen	29,534	5,736	0.12	31,495,866	Siskiyou	37,437	7,271	0.16	39,920,666
Los Angeles	7,941,811	1,257,271	27.16	6,903,261,516	Solano	327,497	49,781	1.08	273,330,417
Madera	105,238	20,002	0.43	109,823,664	Sonoma	388,079	60,444	1.31	331,875,994
Marin	213,100	33,194	0.72	182,258,636	Stanislaus	377,308	71,734	1.55	393,868,942
Mariposa	15,054	2,652	0.06	14,561,781	Sutter	66,116	11,762	0.25	64,579,930
Mendocino	73,687	14,297	0.31	78,502,053	Tehama	46,893	9,103	0.20	49,981,545
Merced	174,831	33,136	0.72	181,936,600	Trinity	11,286	2,193	0.05	12,038,575
Modoc	7,965	1,545	0.03	8,484,977	Tulare	291,303	56,909	1.23	312,470,195
Mono	11,107	2,197	0.05	12,065,267	Tuolumne	48,386	8,596	0.19	47,195,933
Monterey	333,276	54,181	1.17	297,488,537	Ventura	625,002	88,890	1.92	488,063,220
Napa	110,232	17,209	0.37	94,488,444	Yolo	148,886	28,677	0.62	157,457,005
Nevada	82,396	14,656	0.32	80,472,160	Yuba	48,446	8,516	0.18	46,761,128

APPENDIX C

ARB ETS Air Monitoring Study

State of California
California Environmental Protection Agency
AIR RESOURCES BOARD

Near-Source Ambient Air Monitoring of Nicotine as A Marker for Environmental Tobacco Smoke

Prepared by
Special Purpose Monitoring
Air Quality Surveillance Branch
Monitoring and Laboratory Division

October 14, 2003

Executive Summary

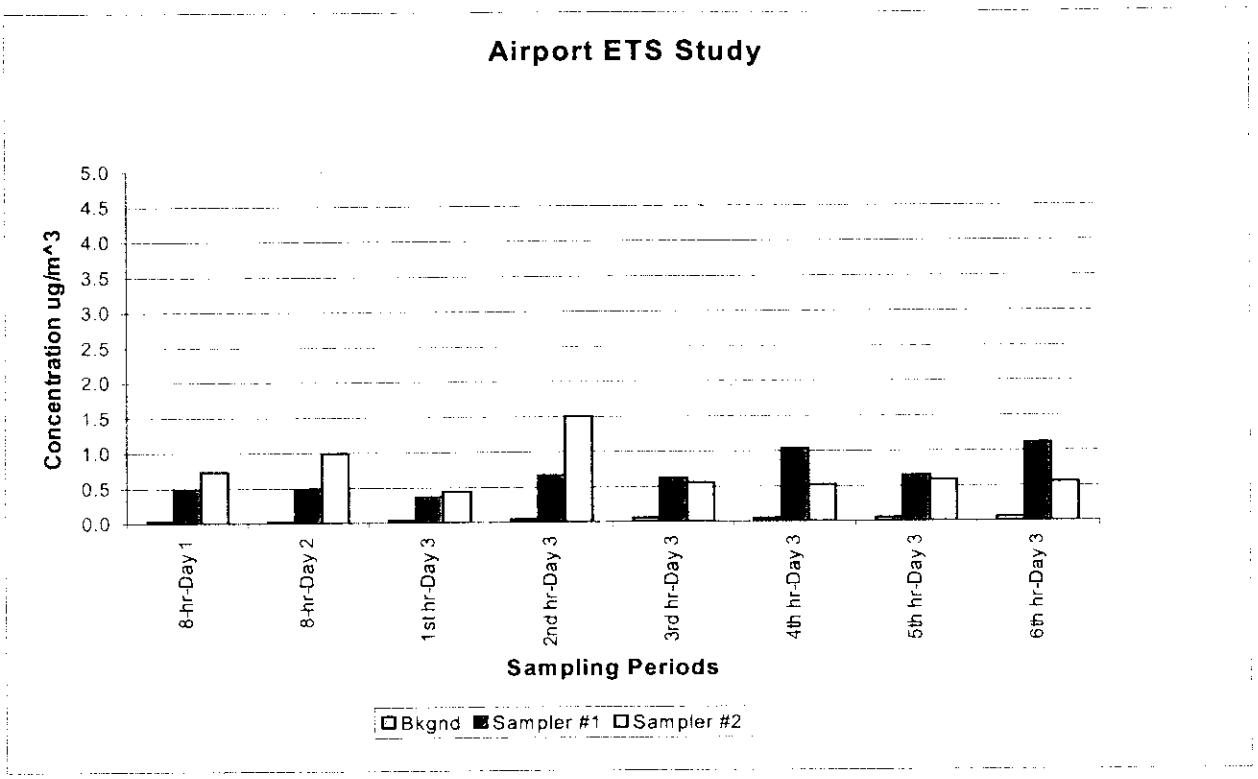
Near-Source Ambient Air Monitoring of Nicotine as A Marker for Environmental Tobacco Smoke

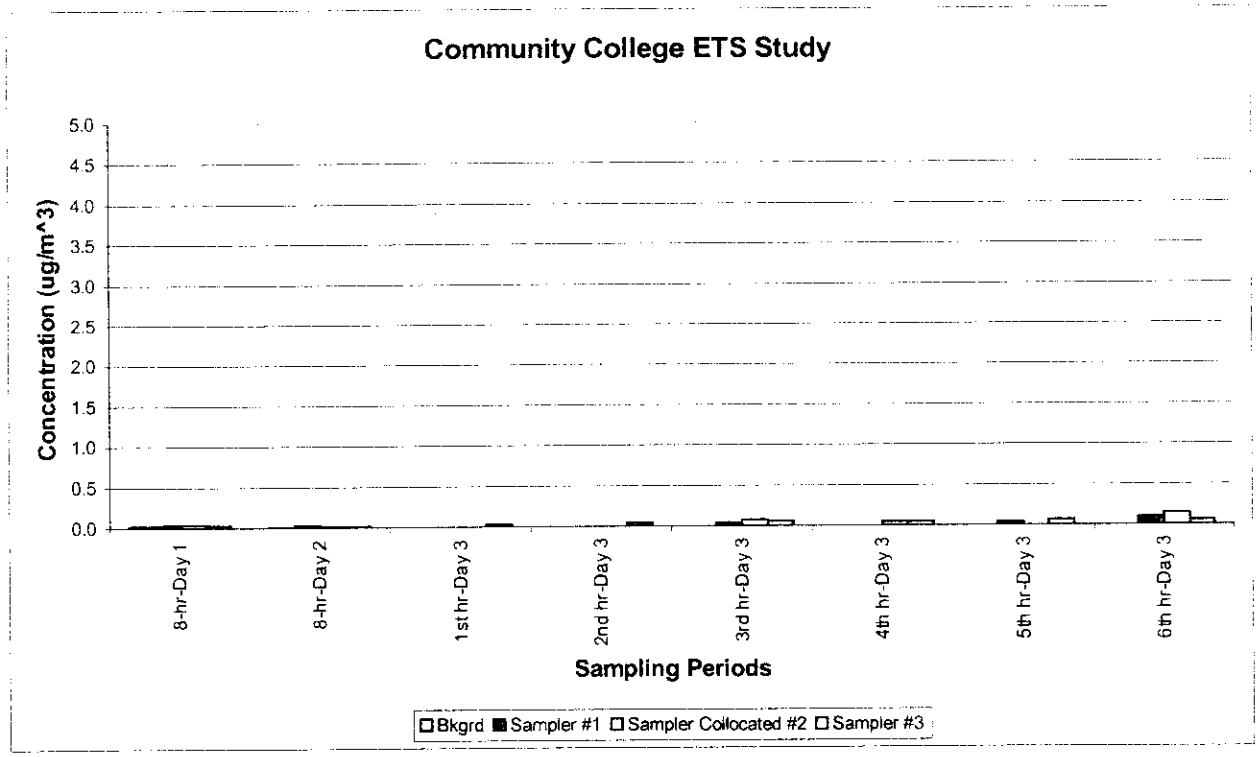
This report presents the results of near-source ambient air monitoring of nicotine as a marker for environmental tobacco smoke (ETS) in California. The objective of the ETS study is to measure ambient outdoor ETS, to which the public is exposed, as part of an evaluation by the Air Resources Board of ETS as a possible toxic air contaminant.

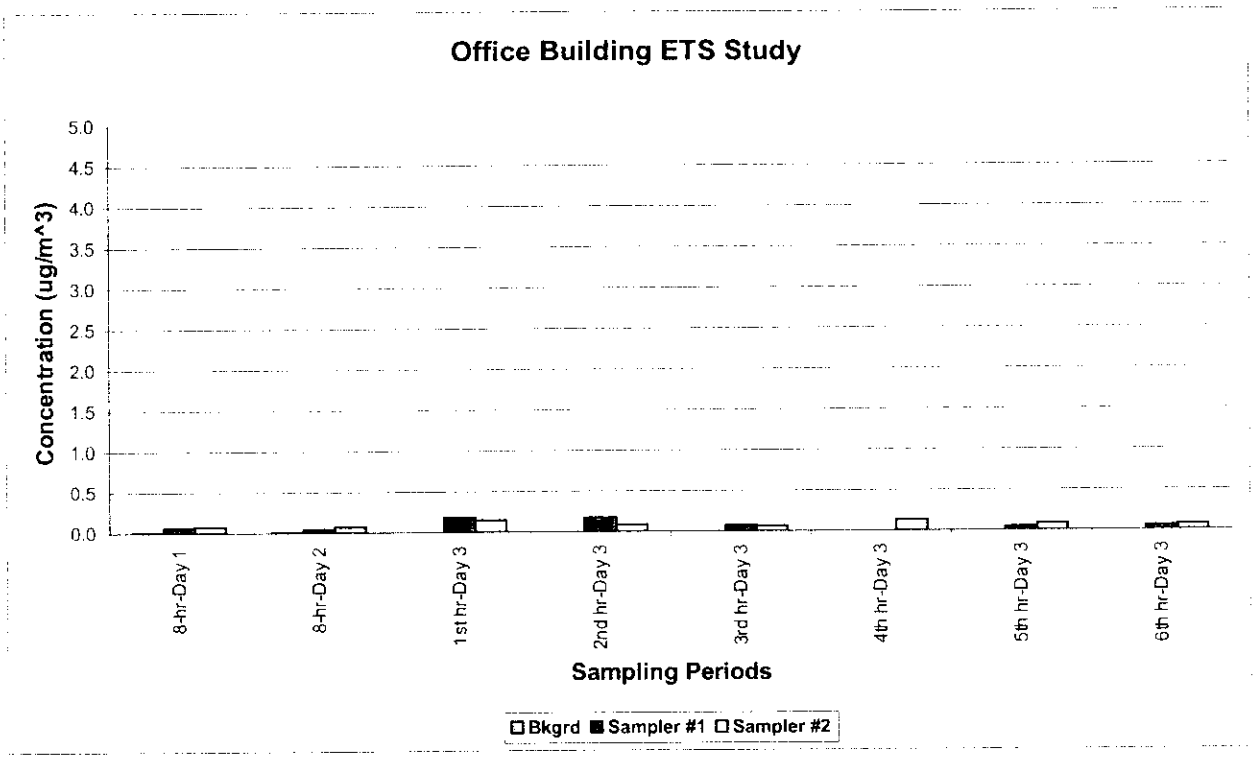
Several outdoor smoking areas were used in the monitoring including an airport, community college, two office buildings, and an amusement park.

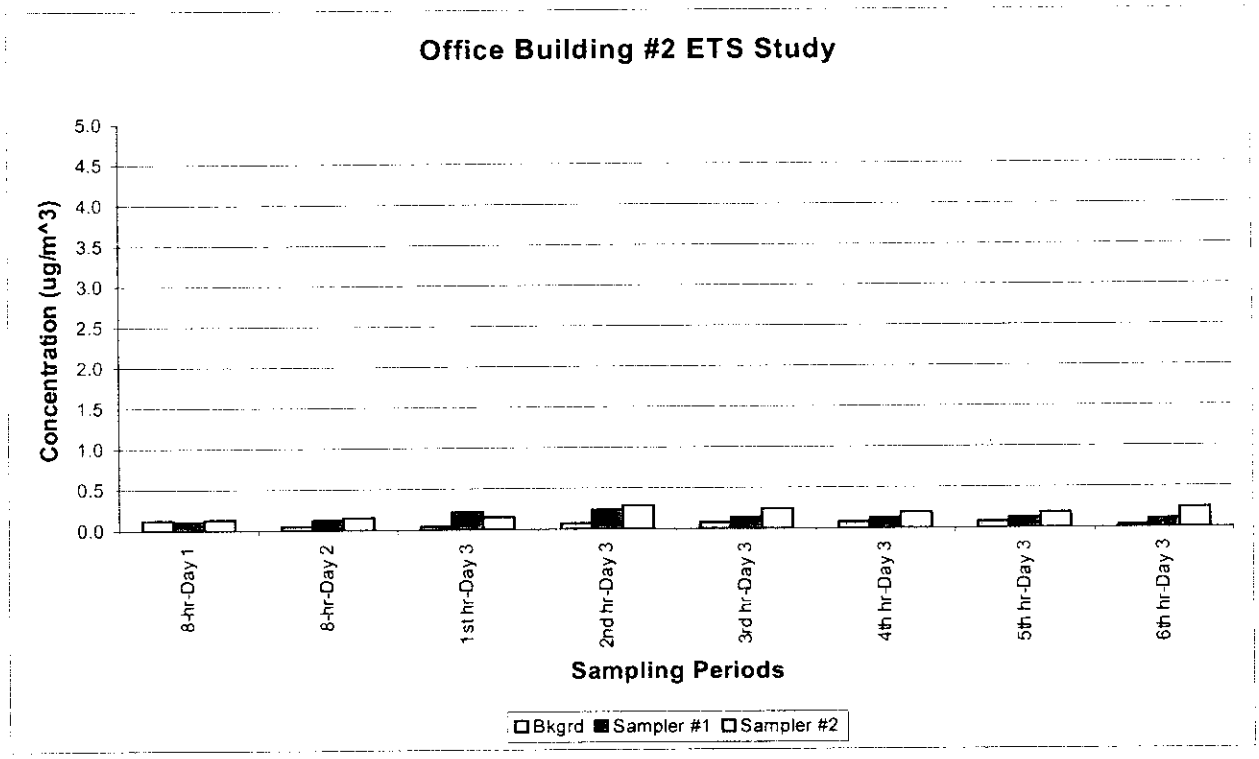
The duration of sampling at each ETS monitoring site took place during a three-day period, and included two days of 8-hour sampling and one day of six consecutive 1-hour sampling. Samples were collected on XAD-4 adsorbent resin cartridges and were analyzed by gas chromatography with a mass selective detector. The monitoring scenarios used were: normal, collocated, and spiked sampling. Normal sampling has two samplers adjacent to the smoking area and one for background (i.e., a sampler located upwind from the environmental tobacco smoke). Collocated sampling is the same except it incorporates another sampler collocated next to one of the two samplers within the smoking area, and spiked sampling includes an extra sampler placed next to the background sampler, which is spiked with known amounts of nicotine.

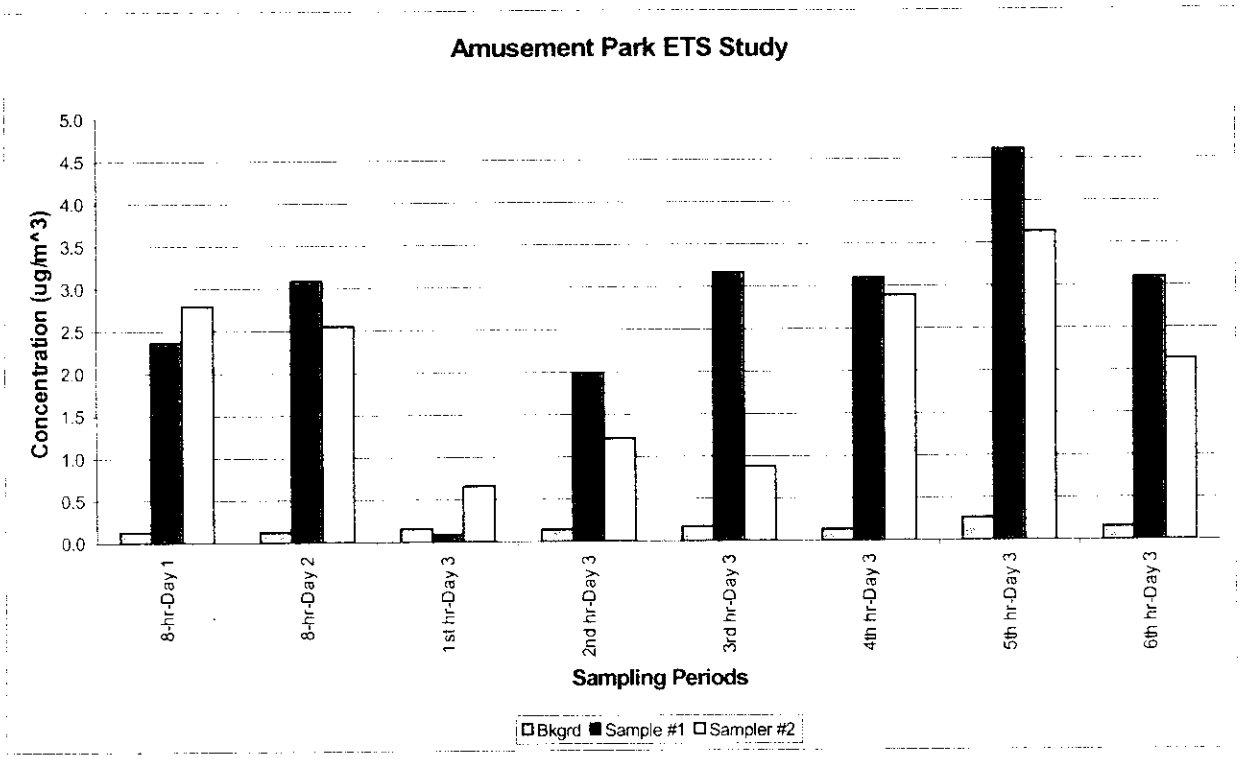
The method detection limit (MDL) used for the 1-hour samples were 0.0058 $\mu g/m^3$ and for the 8-hour samples were 0.00073 $\mu g/m^3$. The quantitative limit (EQL) used for the 1-hour samples were 0.029 $\mu g/m^3$ and 0.0036 $\mu g/m^3$ for the 8-hour samples. Any nicotine concentrations between the MDL and the EQL are considered Trace level. Concentrations measured below the MDL were reported as non-detect. Of the 85 samples collected within the smoking areas (spikes, blanks, and background samples excluded), 81 samples had measurable quantities of nicotine present, while 4 had trace level. The highest 1-hr concentration of nicotine, 4.6 $\mu g/m^3$, and the highest 8-hour concentration, 3.1 $\mu g/m^3$, were observed adjacent to a smoking area at the amusement park. All results are displayed in graphs at the end of this summary.


Airport ETS Study
Concentration ug/m^3
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
8-hr-Day 1
8-hr-Day 2
1st hr-Day 3
2nd hr-Day 3
3rd hr-Day 3
4th hr-Day 3
5th hr-Day 3
6th hr-Day 3
Sampling Periods
Bkgnd
Sampler #1
Sampler #2


Community College ETS Study
Concentration (ug/m^3)
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
8-hr-Day 1
8-hr-Day 2
1st hr-Day 3
2nd hr-Day 3
3rd hr-Day 3
4th hr-Day 3
5th hr-Day 3
6th hr-Day 3
Sampling Periods
Bkgrd
Sampler #1
Sampler Collocated #2
Sampler #3


Office Building ETS Study
Concentration (ug/m^3)
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
8-hr-Day 1
8-hr-Day 2
1st hr-Day 3
2nd hr-Day 3
3rd hr-Day 3
4th hr-Day 3
5th hr-Day 3
6th hr-Day 3
Sampling Periods
Bkgrd
Sampler #1
Sampler #2


Office Building #2 ETS Study
Concentration (ug/m^3)
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
8-hr-Day 1
8-hr-Day 2
1st hr-Day 3
2nd hr-Day 3
3rd hr-Day 3
4th hr-Day 3
5th hr-Day 3
6th hr-Day 3
Sampling Periods
Bkgrd
Sampler #1
Sampler #2


Amusement Park ETS Study
Concentration (ug/m^3)
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
8-hr-Day 1
8-hr-Day 2
1st hr-Day 3
2nd hr-Day 3
3rd hr-Day 3
4th hr-Day 3
5th hr-Day 3
6th hr-Day 3
Sampling Periods
Bkgrd
Sample #1
Sampler #2

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I. Monitoring Report Approval

Near-Source ambient air monitoring of nicotine as a marker for environmental tobacco smoke in Sacramento County, and southern California – 2003

Prepared by: Steven Aston, Air Resources Engineer, Monitoring and Laboratory Division

Approval: The following monitoring report has been reviewed and approved by the Monitoring and Laboratory Division

Dennis Goodenow, Manager
Special Purpose Monitoring

10-4-03
Date

Kenneth R. Stroud, Chief
Air Quality Surveillance Branch

10-15-03
Date

William V. Loscutoff, Chief
Monitoring and Laboratory Division

10.16.03
Date

II. ACKNOWLEDGEMENTS

We wish to acknowledge the individuals at each of the study locations that assisted in gaining permission for us to conduct these monitoring studies. Staff Jerry Wehling, Andy Cowell, and Steve Aston of the MLD-AQSB collected the ambient samples. We would also acknowledge Jim Stebbins, Robert Krieger, and Lynn Baker of SSD for their assistance with selecting the study locations, counting the number of smokers during each sampling period, and with developing the study design.

III. Introduction

At the request of the Stationary Source Division (SSD) of the Air Resources Board (ARB), the Monitoring and Laboratory Division (MLD) staff conducted five near-source, ambient air monitoring studies for Environmental Tobacco Smoke (ETS) using nicotine as a marker. The purpose of the study was to gather information to evaluate ETS as a Toxic Air Contaminant (TAC). The studies were conducted in Sacramento County, and in southern California between January 15, 2003 and June 19, 2003. This report presents the results of near-source ambient air monitoring of nicotine as a marker for Environmental Tobacco Smoke at smoking areas at an airport, community college, two office buildings, and an amusement park.

IV. Sampling

Air samples were collected by passing a measured volume of ambient air through an XAD-4 adsorbent resin-sampling cartridge, following the monitoring protocol contained in Attachment I. The XAD-4 resin sampling cartridges were stored in an ice chest (on dry ice). The flow rate of 15 slpm was measured pre- and post-sampling using a certified transfer standard. The exact operating interval for each sample was recorded in the sampling equipment's memory and was recorded on the filter media transfer sheets. The cartridges were protected from direct sunlight and positioned at least 1.5 meters above the ground. At the end of each sampling period the cartridges were placed in zip-lock bags and placed on dry ice. At the end of each ETS study the cartridges, on dry ice, along with the filter media transfer sheets, were taken directly to Trace Analytical Laboratory in the Department of Environmental Toxicology at UC Davis to perform the analysis.

Three different monitoring scenarios were used: normal, collocated, and spiked sampling (see the following and Figure 1):

Normal sampling: One sampler is placed within the smoking area and one sampler is placed on the perimeter of the smoking area, in the expected downwind direction. The third sampler is placed away from the smoking area in what is expected to be upwind location, referred to as a background sampler (three total samplers).

Collocated sampling: This is the same as normal sampling except an additional sampler is collocated next to one of the two samplers within the smoking area (four total samplers).

Spiked sampling: This is the same as collocated sampling except the additional sampler is collocated next to the background sampler and the XAD-4 sample media is spiked with known quantities of nicotine (four total samplers).

The sites did not comply with all the siting criteria for ambient air monitoring, but every effort was made to meet the micro-scale monitoring siting criteria in 40 CFR Part 58, Appendix E, and Volume II of ARB Quality Assurance Manual. A portable meteorological station was placed at or near the downwind sampler(s) when logistically possible. No meteorological data was collected at the second office building site or at the amusement park. Ambient temperature, scalar wind speed, vector wind direction, and relative humidity were measured continuously and reported in 15-minute averages. The height above ground of the sensors on the portable meteorological station is approximately 2.5 meters. Data is presented using wind rose graphs to show wind patterns and speeds. Air monitoring was not conducted if rain or strong winds occurred or were expected before the sampling period would end.

A. Sampling Equipment

Each sampler consisted of an XAD-4 adsorbent cartridge, Teflon tubing which was placed inside 11/4" PVC pipe, PVC sun shield, a tripod, and a 12-volt DC external battery powering a BGI PQ-100 air sampling pump which was stored in a Rubbermaid tub (see Figure 2). The XAD-4 spiked cartridges were removed from the ice chest containing dry ice and immediately connected to the Teflon tubing then covered by the PVC sun shield during sampling periods. The PQ-100 sampler's flow rate was pre-set at 15 slpm by using a certified volumetric flow meter. Samplers were leak checked prior to each sampling period, with sampling tubes installed. A Met-One meteorological station was set-up prior to sampling.

B. Monitoring and Duration

Several outdoor smoking areas were used in the monitoring including an airport (spiked sampling), college (collocated sampling), two office buildings (normal and spiked sampling), and an amusement park (normal sampling). These outdoor sites provided a broad variety of smoking areas.

Each study took place over a 3-day period, with sampling periods as follows:

- Two sampling days, which consisted of 8-hour sampling from 9:00 a.m. to 5:00 p.m.

- One sampling day, which consisted of 1-hour sampling for 6 consecutive hours from 9:00 a.m. to 3:00 p.m.

The number of monitors and filters depended on the sampling scenario used.

Smoking activity at each study was observed and documented during sampling periods by SSD staff. All activity data resides in SSD files.

V. Analytical Analysis

UC Davis Department of Environmental Toxicology's, Trace Analytical Lab (TAL) prepared the XAD-4 adsorbent cartridges, and analyzed the samples for nicotine using GC/MS. See Attachment II for full UCD analytical report.

VI. Monitoring Results

Wind speed and direction ' wind rose' diagrams for each of the study's sampling periods are shown Figures 3 through 11. Sample results for each sampling site, for each period, are included with the 'wind roses'. The method detection limit (MDL) used for the 1-hour samples were 0.0058 $\mu g/m^3$ and for the 8-hour samples were 0.00073 $\mu g/m^3$. The quantitative limit (EQL) used for the 1-hour samples were 0.029 $\mu g/m^3$ and 0.0036 $\mu g/m^3$ for the 8-hour samples. Any nicotine concentrations between the MDL and the EQL are considered Trace level. Concentrations measured below the MDL were reported as non-detect (ND). Of the 85 samples collected within the smoking areas (spikes, blanks, and background samples excluded), 81 samples had measurable quantities of nicotine present, while 4 had trace level. The highest 1-hr concentration of nicotine, 4.6073 $\mu g/m^3$, and the highest 8-hour concentration, 3.0958 $\mu g/m^3$, were observed adjacent to a smoking area at the amusement park. A summary of site data as follows:

Airport 1/15-17/2003	High Concentration $\mu g/m^3$ within smoking area 1hr = 1.4982 Low Concentration $\mu g/m^3$ within smoking area 1hr = .3622 Background High Concentration $\mu g/m^3$ 1hr = .0565 Background Low Concentration $\mu g/m^3$ 8hr = .0185
Community College 4/1,2,7/2003	High Concentration $\mu g/m^3$ within smoking area 1hr = .1463 Low Concentration $\mu g/m^3$ within smoking area 1 & 8hr = Trace Background High Concentration $\mu g/m^3$ 8hr = .0183 Background Low Concentration $\mu g/m^3$ 1hr = Trace
Office Building 4/9-11/2003	High Concentration $\mu g/m^3$ within smoking area 1hr = .1756 Low Concentration $\mu g/m^3$ within smoking area 1hr = .0387 Background High Concentration $\mu g/m^3$ 8hr = .0102 Background Low Concentration $\mu g/m^3$ 1hr = Trace
Office Building II 5/20-22/2003	High Concentration $\mu g/m^3$ within smoking area 1hr = .2824 Low Concentration $\mu g/m^3$ within smoking area 1hr = .1020 Background High Concentration $\mu g/m^3$ 8hr = .1240 Background Low Concentration $\mu g/m^3$ 1hr = .0321

Amusement park
6/17-19/2003

High Concentration µg/m³ within smoking area 1hr= **4.6073**
Low Concentration µg/m³ within smoking area 1hr = .6602
Background High Concentration µg/m³ 1hr= .2630
Background Low Concentration µg/m³ 8hr = .1216

VII. Field Quality Assurance

Field quality assurance for the monitoring included the following:

A. Trip Blank

Trip blanks for nicotine were obtained from UCD, labeled, recorded on the filter media transfer sheets, and transported along with the samples for every study.

B. Collocated Samples

Collocated samples were taken at the community college.

C. Trip Spikes

Trip spikes for nicotine were obtained from UCD, labeled, recorded on the filter media transfer sheets, and transported along with the samples for every study.

D. Field Spikes

Field spikes for nicotine were obtained, labeled, recorded on the filter media transfer sheets, and transported along with the samples for every study.

VIII. Quality Assurance Results

A. Trip Blank Sampling Results

The trip blanks results were from trace level to ND, corresponding to a concentration between 0.0058 and 0.029 µg/m^3 for 1-hour samples and between 0.00073 and 0.0036 µg/m^3 for 8-hour samples.

B. Background Sampling Results

Background samples were collected for every study and located in predominant upwind direction. The background levels ranged from .2630 µg/m³ to trace levels. The same concentration limits above were used for all filter media. See page 4 for summary of monitoring results.

C. Collocated Sampling Results

There were two 8-hour and two 1-hour collocated sampling periods with quantifiable levels of nicotine. The comparison of collocated samples (calculated as the difference between the two collocated samples divided by the mean of the two samples) ranged from 32-58% for the 8-hour samples and was 42-54% for the 1-hour sample, as shown in Table 1.

Nicotine Collocated Results (µg/m³)

Sampling Period	Sampler #1	Sampler #2 col	Mean	RPD %
4/1/2003 8-hrs.	0.0316	0.0437	0.0377	32%
4/2/2003 8-hrs.	0.0273	0.0151	0.0212	58%
4/7/2003 1st hr.	Trace	Trace	NA	NA
4/7/2003 2nd hr.	Trace	Trace	NA	NA
4/7/2003 3rd hr.	0.0434	0.0752	0.0593	54%
4/7/2003 4th hr.	INVALID	0.0484	NA	NA
4/7/2003 5th hr.	Trace	INVALID	NA	NA
4/7/2003 6th hr.	0.096	0.1463	0.1212	42%

Table 1

D. Trip and Field Spike Results

Trip and field spikes are prepared for each individual study ranging from 400 µg to 10 µg of nicotine. The spikes are prepared in sets to allow statistics to be applied if necessary to evaluate differences in the results of the sets.

Once the spikes are received they are immediately placed on dry ice and kept there until extraction and analysis. The trip spike samples are kept on dry ice in an ice chest (the same one used for samples) during transportation to the field and at all times while in the field.

The field spike samples are kept on dry ice in an ice chest (the same one used for samples) during transportation to and from the field and at all times while in the field except for the sampling period. Field spikes were subjected to the same environment and experimental conditions (i.e. flow rates) as those occurring at the time of ambient sampling. The field spikes were obtained by sampling ambient air through the previously spiked cartridge and were collocated with a background sample. The percent recovery for the trip spikes ranged from 72% to 89% and the field spikes ranged from 76% to 87%.

All trip and field spikes for the studies were prepared by UC Davis Department of Environmental Toxicology's Trace Analytical Lab (TAL).

ETS Sampling Scenarios

Normal Sampling

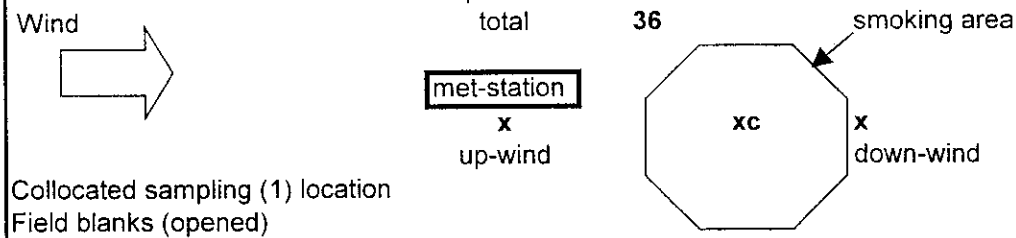
1st Day	2nd Day	3rd Day		
3	3	18	samples	24
		2	spares	2
		1	field blanks	1
		1	trip blanks	1
			total	**28**

note: **x** represents samplers/locations
note: **xc** represents collocated sampler
note: **xs** represents spiked sampler

Normal sampling (0-3) locations
Field blanks (opened)
Trip blanks (not opened)

Collocated Sampling

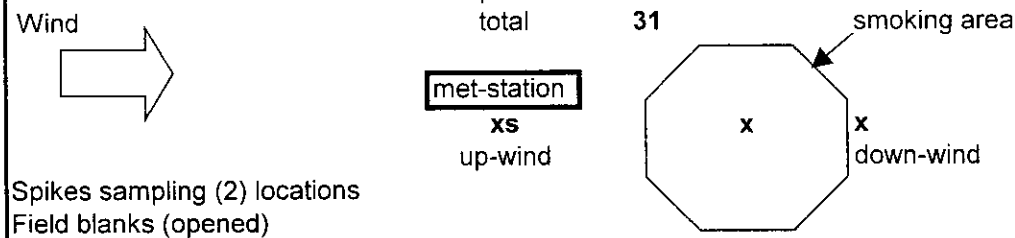
1st Day	2nd Day	3rd Day		
3	3	18	samples	24
1	1	6	collocated	8
		2	spares	2
		1	field blanks	1
		1	trip blanks	1
			total	**36**



Collocated sampling (1) location
Field blanks (opened)
Trip blanks (not opened)

Spiked Sampling

1st Day	2nd Day	3rd Day		
3	3	18	samples	24
		2	spares	2
		1	field blanks	1
		1	trip blanks	1
1	1	1	spikes	3
			total	**31**



Spikes sampling (2) locations
Field blanks (opened)
Trip blanks (not opened)

Figure 1

ETS AMBIENT AIR MONITOR

Figure 2

ARB/Nicotine Samples Set #1 AIRPORT
Samples received on 1/17/2003
Date Extracted: 1/21/2003
Date Analyzed: 1/24/2003

Limits Used for µg/m3:
1 hour 0.0058 µg/m3 £ Trace < 0.029 µg/m3
8 hours 0.00073 µg/m3 £ Trace < 0.0036 µg/m3

Nicotine

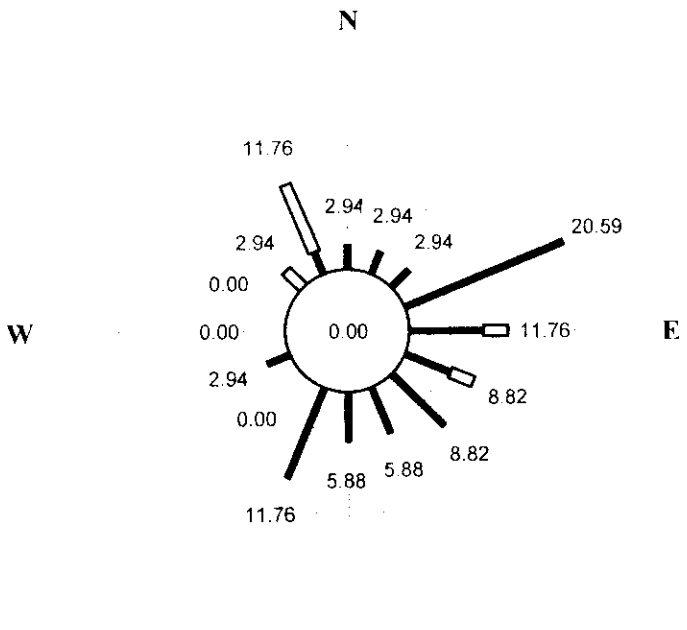
Sample ID	Fortification Level (µg)	µg found	%Rec	Ave %Rec	Stdev	Run Time (min.)	Flow (L/min.)	Vol (L)	Vol (m^3)	µg/m^3
NIC133C		Trace								
NIC102TB		Trace								
NIC103FB		Trace								
NIC134CR400R1	400.0	366.164	92%							
NIC135CR400R2	400.0	378.494	95%							
NIC136CR400R3	400.0	353.990	88%	92%	3%					
										sampler
NIC104FS400R1	400.0	341.906	85%			480	15	7200	7.20	4
NIC105FS400R2	400.0	349.841	87%			480	15	7200	7.20	4
NIC106FS400R3	400.0	344.127	86%	86%	1%	60	15	900	0.90	4
				sampler	date					
NIC132		3.4598		1	15th	480	15	7200	7.20	0.4805
NIC131		5.2664		2	15th	480	15	7200	7.20	0.7314
NIC130		0.1641		3	15th	480	15	7200	7.20	0.0228
NIC129		3.5275		1	16th	480	15	7200	7.20	0.4899
NIC128		7.1266		2	16th	480	15	7200	7.20	0.9898
NIC127		0.1334		3	16th	480	15	7200	7.20	0.0185
NIC125		0.3260		1	17th-1hr	60	15	900	0.90	0.3622
NIC126		0.3949		2	17th-1hr	60	15	900	0.90	0.4388
NIC124		0.0320		3	17th-1hr	60	15	900	0.90	0.0355
NIC123		0.5972		1	17th-2hr	60	15	900	0.90	0.6636
NIC122		1.3484		2	17th-2hr	60	15	900	0.90	1.4982
NIC121		0.0375		3	17th-2hr	60	15	900	0.90	0.0417
NIC119		0.5578		1	17th-3hr	60	15	900	0.90	0.6198
NIC120		0.4978		2	17th-3hr	60	15	900	0.90	0.5531
NIC118		0.0473		3	17th-3hr	60	15	900	0.90	0.0525
NIC116		0.9290		1	17th-4hr	60	15	900	0.90	1.0323
NIC117		0.4655		2	17th-4hr	60	15	900	0.90	0.5172
NIC115		0.0409		3	17th-4hr	60	15	900	0.90	0.0454
NIC113		0.5843		1	17th-5hr	60	15	900	0.90	0.6492
NIC114		0.5294		2	17th-5hr	60	15	900	0.90	0.5882
NIC112		0.0425		3	17th-5hr	60	15	900	0.90	0.0472
NIC110		0.9857		1	17th-6hr	59	15	885	0.89	1.1137
NIC111		0.5001		2	17th-6hr	60	15	900	0.90	0.5557
NIC109		0.0508		3	17th-6hr	60	15	900	0.90	0.0565

C = Resin Blank
TB = Trip Blank
FB = Field Blank
CR = Concurrent Recovery
FS = Field Spike

Sampler #1 northside (closest to terminal B)
Sampler #2 southside (closest to terminal A)
Sampler #3 Background grass area between terminal A and B

Table 2

WIND ROSE
AIRPORT ETS STUDY
JANUARY 15, 08:45 TO 17:00 HRS. (15 MIN. AVG.)



Calms included at center.
Rings drawn at 5% intervals.
Wind flow is FROM the directions shown.
No observations were missing.

PERCENT OCCURRENCE Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
N	2.94	0.00	0.00	0.00	0.00	0.00
NNE	2.94	0.00	0.00	0.00	0.00	0.00
NE	2.94	0.00	0.00	0.00	0.00	0.00
ENE	20.59	0.00	0.00	0.00	0.00	0.00
E	8.82	2.94	0.00	0.00	0.00	0.00
ESE	5.88	2.94	0.00	0.00	0.00	0.00
SE	8.82	0.00	0.00	0.00	0.00	0.00
SSE	5.88	0.00	0.00	0.00	0.00	0.00

TOTAL OBS = 34 MISSING OBS = 0

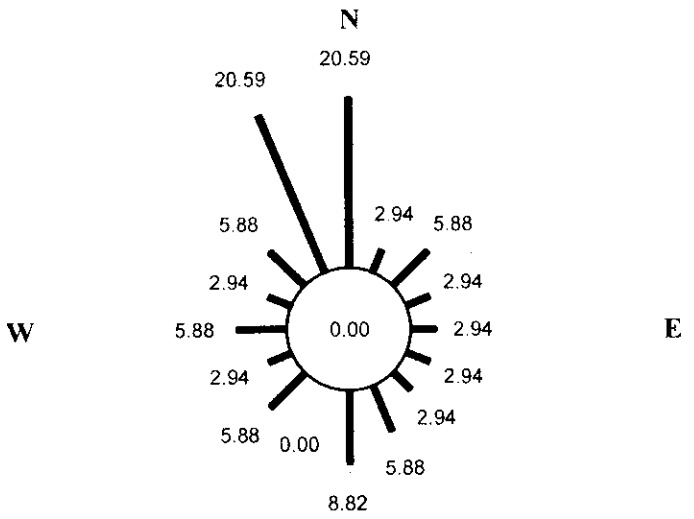
PERCENT OCCURRENCE Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
S	5.88	0.00	0.00	0.00	0.00	0.00
SSW	11.76	0.00	0.00	0.00	0.00	0.00
SW	0.00	0.00	0.00	0.00	0.00	0.00
WSW	2.94	0.00	0.00	0.00	0.00	0.00
W	0.00	0.00	0.00	0.00	0.00	0.00
WNW	0.00	0.00	0.00	0.00	0.00	0.00
NW	0.00	2.94	0.00	0.00	0.00	0.00
NNW	2.94	8.82	0.00	0.00	0.00	0.00

CALM OBS = 0 PERCENT CALM = 0.00

Figure 3

WIND ROSE
AIRPORT ETS STUDY
JANUARY 16, 2003 FROM 08:45 TO 17:00 HRS. (15 MIN. AVG.)



PERCENT OCCURRENCE: Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
N	20.59	0.00	0.00	0.00	0.00	0.00
NNE	2.94	0.00	0.00	0.00	0.00	0.00
NE	5.88	0.00	0.00	0.00	0.00	0.00
ENE	2.94	0.00	0.00	0.00	0.00	0.00
E	2.94	0.00	0.00	0.00	0.00	0.00
ESE	2.94	0.00	0.00	0.00	0.00	0.00
SE	2.94	0.00	0.00	0.00	0.00	0.00
SSE	5.88	0.00	0.00	0.00	0.00	0.00

TOTAL OBS = 34 MISSING OBS = 0

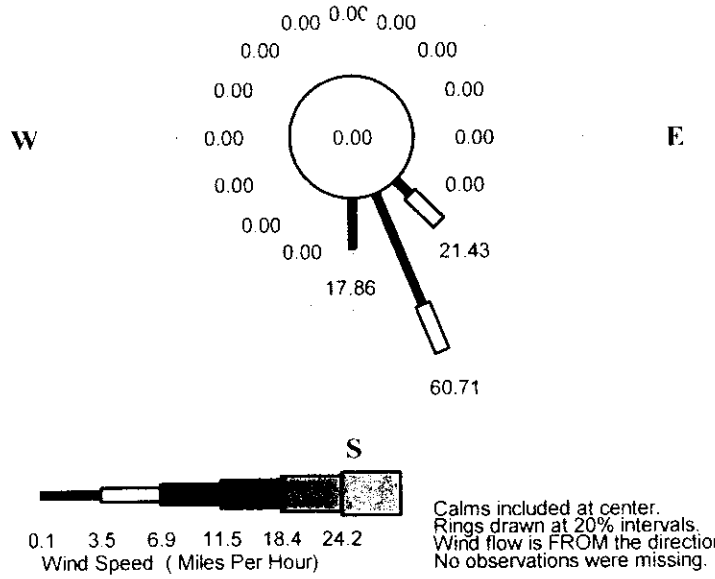
PERCENT OCCURRENCE: Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
S	8.82	0.00	0.00	0.00	0.00	0.00
SSW	0.00	0.00	0.00	0.00	0.00	0.00
SW	5.88	0.00	0.00	0.00	0.00	0.00
WSW	2.94	0.00	0.00	0.00	0.00	0.00
W	5.88	0.00	0.00	0.00	0.00	0.00
WNW	2.94	0.00	0.00	0.00	0.00	0.00
NW	5.88	0.00	0.00	0.00	0.00	0.00
NNW	20.59	0.00	0.00	0.00	0.00	0.00

CALM OBS = 0 PERCENT CALM = 0.00

Figure 4

WIND ROSE
AIRPORT ETS STUDY
JANUARY 17, 2003 FROM 08:45 TO 15:30 HRS. (15 MIN. AVG.)



Calms included at center.
Rings drawn at 20% intervals.
Wind flow is FROM the directions shown.
No observations were missing.

PERCENT OCCURRENCE Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
N	0.00	0.00	0.00	0.00	0.00	0.00
NNE	0.00	0.00	0.00	0.00	0.00	0.00
NE	0.00	0.00	0.00	0.00	0.00	0.00
ENE	0.00	0.00	0.00	0.00	0.00	0.00
E	0.00	0.00	0.00	0.00	0.00	0.00
ESE	0.00	0.00	0.00	0.00	0.00	0.00
SE	7.14	14.29	0.00	0.00	0.00	0.00
SSE	42.86	17.86	0.00	0.00	0.00	0.00

TOTAL OBS = 28 MISSING OBS = 0

PERCENT OCCURRENCE Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
S	17.86	0.00	0.00	0.00	0.00	0.00
SSW	0.00	0.00	0.00	0.00	0.00	0.00
SW	0.00	0.00	0.00	0.00	0.00	0.00
WSW	0.00	0.00	0.00	0.00	0.00	0.00
W	0.00	0.00	0.00	0.00	0.00	0.00
WNW	0.00	0.00	0.00	0.00	0.00	0.00
NW	0.00	0.00	0.00	0.00	0.00	0.00
NNW	0.00	0.00	0.00	0.00	0.00	0.00

CALM OBS = 0 PERCENT CALM = 0.00

Figure 5

ARB/Nicotine Samples Set #2 Community College
Samples received on 4/8/2003
Date Extracted: 4/8/2003
Date Analyzed: 4/9/2003

Limits Used for µg/m3:
1 hour 0.0058 µg/m3 £ Trace < 0.029 µg/m3
8 hours 0.00073 µg/m3 £ Trace < 0.0036 µg/m3

Nicotine

Sample ID	Fortification Level (µg)	µg found	% Rec	Ave % Rec	Stdev	Run Time (min.)	Flow (L/min.)	Vol (L)	Vol (m^3)	µg/m^3
NIC176C		Trace								
NIC137TB		Trace								
NIC177CR100R1	100.0	79.1105	79%							
NIC178CR100R2	100.0	74.4450	74%							
NIC179CR100R3	100.0	81.5101	82%	78%	4%					
NIC175FS100R1	100.0	83.3829	83%							
NIC138TS100R1	100.0	82.2737	82%							
				sampler	date					
NIC139		0.2279		1	1st	480	15	7200	7.20	0.0316
NIC140		0.3147		2	1st	480	15	7200	7.20	0.0437
NIC141		0.2131		3	1st	480	15	7200	7.20	0.0296
NIC142		0.1320		4	1st	480	15	7200	7.20	0.0183
NIC143		0.1962		1	2nd	480	15	7200	7.20	0.0273
NIC144		0.1090		2	2nd	480	15	7200	7.20	0.0151
NIC145		0.0930		3	2nd	480	15	7200	7.20	0.0129
NIC146		0.0341		4	2nd	480	15	7200	7.20	0.0047
NIC147		0.0222		1	7th-1hr	60	15	900	0.90	Trace
NIC148		0.0209		2	7th-1hr	60	15	900	0.90	Trace
NIC149		0.0323		3	7th-1hr	60	15	900	0.90	0.0359
NIC150		0.0127		4	7th-1hr	60	15	900	0.90	Trace
NIC151		0.0156		1	7th-2hr	60	15	900	0.90	Trace
NIC152		0.0169		2	7th-2hr	60	15	900	0.90	Trace
NIC153		0.0339		3	7th-2hr	60	15	900	0.90	0.0377
NIC154		0.0188		4	7th-2hr	60	15	900	0.90	Trace
NIC155		0.0391		1	7th-3hr	60	15	900	0.90	0.0434
NIC156		0.0676		2	7th-3hr	60	15	900	0.90	0.0752
NIC171		0.0500		3	7th-3hr	60	15	900	0.90	0.0556
NIC158		0.0235		4	7th-3hr	60	15	900	0.90	Trace
NIC160		0.0436		2	7th-4hr	60	15	900	0.90	0.0484
NIC161		0.0416		3	7th-4hr	60	15	900	0.90	0.0462
NIC162		0.0179		4	7th-4hr	60	15	900	0.90	Trace
NIC163		0.0399		1	7th-5hr	60	15	900	0.90	0.0444
NIC165		0.0585		3	7th-5hr	60	15	900	0.90	0.0650
NIC166		0.0198		4	7th-5hr	60	15	900	0.90	Trace
NIC167		0.0864		1	7th-6hr	60	15	900	0.90	0.0960
NIC168		0.1316		2	7th-6hr	60	15	900	0.90	0.1463
NIC169		0.0473		3	7th-6hr	60	15	900	0.90	0.0525
NIC170		0.0245		4	7th-6hr	60	15	900	0.90	Trace

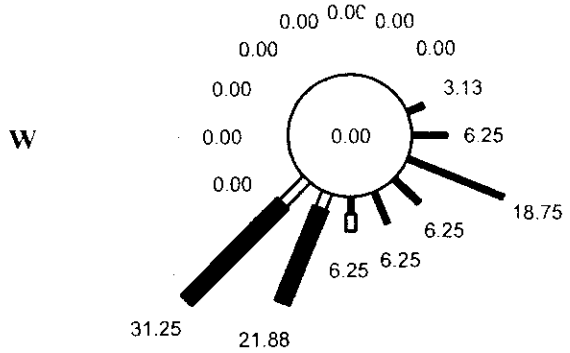
C = Resin Blank
TB = Trip Blank
CR = Concurrent Recovery
FS = Field Spike

sampler #1 Eastside of smoking area near BBQ pit
sampler #2 Collocated next to sampler #1
sampler #3 Westside of smoking area
sampler #4 Background Southside of smoking area

sampler #4 Background Southside of smoking area

Table 3

WIND ROSE
COMMUNITY COLLEGE ETS STUDY
APRIL 1, 2003 FROM 09:00 TO 17:00 HRS. (15 MIN. AVG.)



Calms included at center.
Rings drawn at 10% intervals.
Wind flow is FROM the directions shown.
No observations were missing.

PERCENT OCCURRENCE Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
N	0.00	0.00	0.00	0.00	0.00	0.00
NNE	0.00	0.00	0.00	0.00	0.00	0.00
NE	0.00	0.00	0.00	0.00	0.00	0.00
ENE	3.13	0.00	0.00	0.00	0.00	0.00
E	6.25	0.00	0.00	0.00	0.00	0.00
ESE	18.75	0.00	0.00	0.00	0.00	0.00
SE	6.25	0.00	0.00	0.00	0.00	0.00
SSE	6.25	0.00	0.00	0.00	0.00	0.00

TOTAL OBS = 32 MISSING OBS = 0

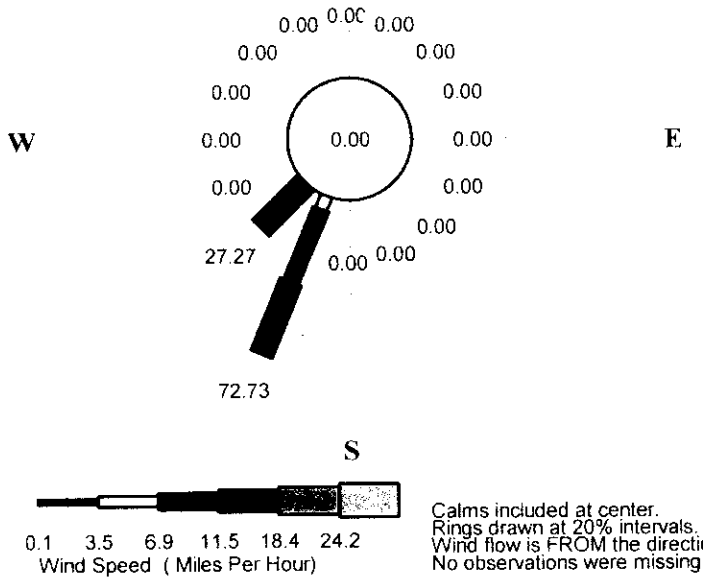
PERCENT OCCURRENCE Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
S	3.13	3.13	0.00	0.00	0.00	0.00
SSW	0.00	3.13	18.75	0.00	0.00	0.00
SW	0.00	6.25	25.00	0.00	0.00	0.00
WSW	0.00	0.00	0.00	0.00	0.00	0.00
W	0.00	0.00	0.00	0.00	0.00	0.00
WNW	0.00	0.00	0.00	0.00	0.00	0.00
NW	0.00	0.00	0.00	0.00	0.00	0.00
NNW	0.00	0.00	0.00	0.00	0.00	0.00

CALM OBS = 0 PERCENT CALM = 0.00

Figure 6

WIND ROSE
COMMUNITY COLLEGE ETS STUDY
APRIL 2, 2003 FROM 09:00 TO 17:00 HRS. (15 MIN. AVG.)



PERCENT OCCURRENCE Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
N	0.00	0.00	0.00	0.00	0.00	0.00
NNE	0.00	0.00	0.00	0.00	0.00	0.00
NE	0.00	0.00	0.00	0.00	0.00	0.00
ENE	0.00	0.00	0.00	0.00	0.00	0.00
E	0.00	0.00	0.00	0.00	0.00	0.00
ESE	0.00	0.00	0.00	0.00	0.00	0.00
SE	0.00	0.00	0.00	0.00	0.00	0.00
SSE	0.00	0.00	0.00	0.00	0.00	0.00

TOTAL OBS = 33 MISSING OBS = 0

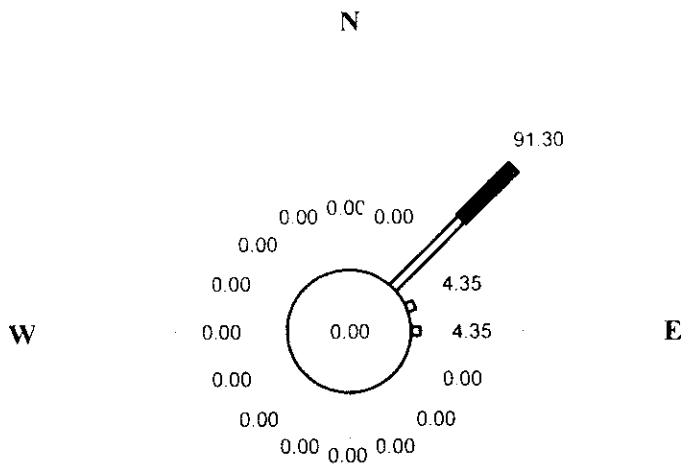
PERCENT OCCURRENCE Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
S	0.00	0.00	0.00	0.00	0.00	0.00
SSW	0.00	6.06	33.33	33.33	0.00	0.00
SW	0.00	0.00	0.00	27.27	0.00	0.00
WSW	0.00	0.00	0.00	0.00	0.00	0.00
W	0.00	0.00	0.00	0.00	0.00	0.00
WNW	0.00	0.00	0.00	0.00	0.00	0.00
NW	0.00	0.00	0.00	0.00	0.00	0.00
NNW	0.00	0.00	0.00	0.00	0.00	0.00

CALM OBS = 0 PERCENT CALM = 0.00

Figure 7

WIND ROSE
COMMUNITY COLLEGE ETS STUDY
APRIL 7, 2003 FROM 09:00 TO 14:30 HRS. (15 MIN. AVG.)



Calms included at center.
Rings drawn at 20% intervals.
Wind flow is FROM the directions shown.
No observations were missing.

PERCENT OCCURRENCE Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
N	0.00	0.00	0.00	0.00	0.00	0.00
NNE	0.00	0.00	0.00	0.00	0.00	0.00
NE	0.00	52.17	39.13	0.00	0.00	0.00
ENE	0.00	4.35	0.00	0.00	0.00	0.00
E	0.00	4.35	0.00	0.00	0.00	0.00
ESE	0.00	0.00	0.00	0.00	0.00	0.00
SE	0.00	0.00	0.00	0.00	0.00	0.00
SSE	0.00	0.00	0.00	0.00	0.00	0.00

TOTAL OBS = 23 MISSING OBS = 0

PERCENT OCCURRENCE Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
S	0.00	0.00	0.00	0.00	0.00	0.00
SSW	0.00	0.00	0.00	0.00	0.00	0.00
SW	0.00	0.00	0.00	0.00	0.00	0.00
WSW	0.00	0.00	0.00	0.00	0.00	0.00
W	0.00	0.00	0.00	0.00	0.00	0.00
WNW	0.00	0.00	0.00	0.00	0.00	0.00
NW	0.00	0.00	0.00	0.00	0.00	0.00
NNW	0.00	0.00	0.00	0.00	0.00	0.00

CALM OBS = 0 PERCENT CALM = 0.00

Figure 8

ARB/Nicotine Samples Set #3 Office Building
Samples received on 4/14/2003
Date Extracted: 4/14/2003
Date Analyzed: 4/14/2003

Limits Used for µg/m3:
1 hour 0.0058 µg/m3 £ Trace < 0.029 µg/m3
8 hours 0.00073 µg/m3 £ Trace < 0.0036 µg/m3

Nicotine

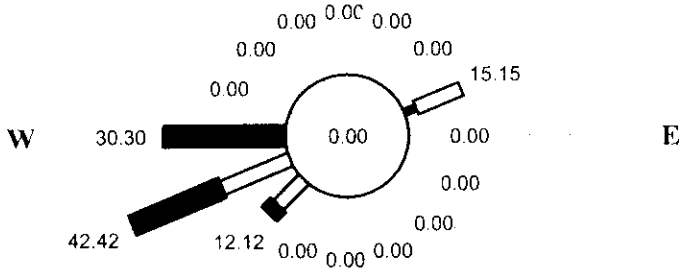
Sample ID	Fortification Level (µg)	µg found	% Rec	Ave % Rec	Stdev	Run Time (min.)	Flow (L/min.)	Vol (L)	Vol (m^3)	µg/m^3
NIC206C		ND								
NIC102TB		ND								
NIC207CR50R1	50.0	47.5140	95%							
NIC208CR50R2	50.0	44.7014	89%							
NIC209CR50R3	50.0	47.0994	94%	93%	3%					
NIC182FS50R1	50.0	38.0002	76%							
NIC181TS50R1	50.0	44.6704	89%							
				sampler	date					
NIC172		0.4262		1	9th	480	15	7200	7.20	0.0592
NIC173		0.5261		2	9th	480	15	7200	7.20	0.0731
NIC174		0.0734		3	9th	480	15	7200	7.20	0.0102
NIC183		0.3006		1	10th	480	15	7200	7.20	0.0417
NIC184		0.4856		2	10th	480	15	7200	7.20	0.0674
NIC185		0.0555		3	10th	480	15	7200	7.20	0.0077
NIC186		0.1581		1	11th-1hr	60	15	900	0.90	0.1756
NIC187		0.1302		2	11th-1hr	60	15	900	0.90	0.1447
NIC188		0.0249		3	11th-1hr	60	15	900	0.90	Trace
NIC189		0.1551		1	11th-2hr	60	15	900	0.90	0.1724
NIC190		0.0717		2	11th-2hr	60	15	900	0.90	0.0797
NIC191		0.0199		3	11th-2hr	60	15	900	0.90	Trace
NIC192		0.0632		1	11th-3hr	60	15	900	0.90	0.0702
NIC193		0.0541		2	11th-3hr	60	15	900	0.90	0.0601
NIC194		0.0187		3	11th-3hr	60	15	900	0.9	Trace
NIC195		ND		1	ND	NA	NA	NA	NA	NA
NIC196		0.1178		2	11th-4hr	60	15	900	0.90	0.1309
NIC197		0.0150		3	11th-4hr	60	15	900	0.90	Trace
NIC198		0.0348		1	11th-5hr	60	15	900	0.90	0.0387
NIC199		0.0740		2	11th-5hr	60	15	900	0.90	0.0822
NIC200		ND		3	11th-5hr	60	15	900	0.90	ND
NIC201		0.0417		1	11th-6hr	60	15	900	0.90	0.0463
NIC202		0.0632		2	11th-6hr	60	15	900	0.90	0.0702
NIC203		0.025		3	11th-6hr	60	15	900	0.90	Trace

C = Resin Blank
TB = Trip Blank
CR = Concurrent Recovery
FS = Field Spike

sampler #1 Eastside of smoking area next to tree
sampler #2 Westside of smoking area in between benches
sampler #3 Background west of smoking area on lawn

Table 4

WIND ROSE
OFFICE BUILDING ETS STUDY
APRIL 9, 2003 FROM 09:00 TO 17:00 HRS. (15 MIN. AVG.)



0.1 3.5 6.9 11.5 18.4 24.2
Wind Speed (Miles Per Hour)

Calms included at center.
Rings drawn at 10% intervals.
Wind flow is FROM the directions shown.
No observations were missing.

PERCENT OCCURRENCE. Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
N	0.00	0.00	0.00	0.00	0.00	0.00
NNE	0.00	0.00	0.00	0.00	0.00	0.00
NE	0.00	0.00	0.00	0.00	0.00	0.00
ENE	3.03	12.12	0.00	0.00	0.00	0.00
E	0.00	0.00	0.00	0.00	0.00	0.00
ESE	0.00	0.00	0.00	0.00	0.00	0.00
SE	0.00	0.00	0.00	0.00	0.00	0.00
SSE	0.00	0.00	0.00	0.00	0.00	0.00

TOTAL OBS = 33 MISSING OBS = 0

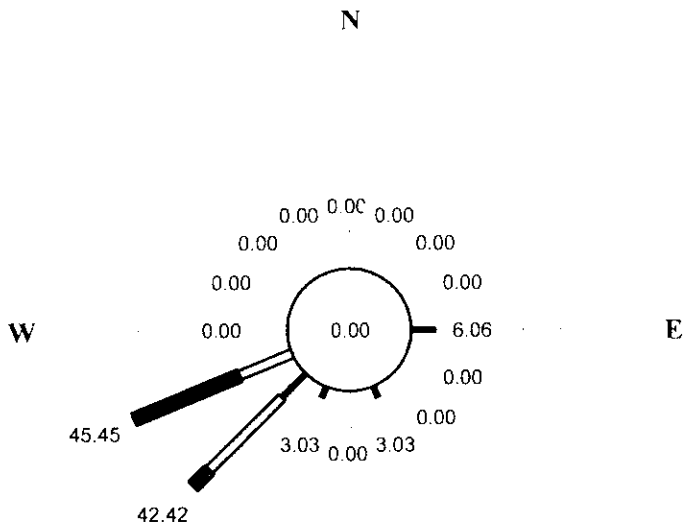
PERCENT OCCURRENCE. Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
S	0.00	0.00	0.00	0.00	0.00	0.00
SSW	0.00	0.00	0.00	0.00	0.00	0.00
SW	0.00	9.09	3.03	0.00	0.00	0.00
WSW	0.00	18.18	24.24	0.00	0.00	0.00
W	0.00	0.00	30.30	0.00	0.00	0.00
WNW	0.00	0.00	0.00	0.00	0.00	0.00
NW	0.00	0.00	0.00	0.00	0.00	0.00
NNW	0.00	0.00	0.00	0.00	0.00	0.00

CALM OBS = 0 PERCENT CALM = 0.00

Figure 9

WIND ROSE
OFFICE BUILDING ETS STUDY
APRIL 10, 2003 FROM 09:00 TO 17:00 HRS. (15 MIN. AVG.)



Calms included at center.
Rings drawn at 10% intervals.
Wind flow is FROM the directions shown.
No observations were missing.

PERCENT OCCURRENCE Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
N	0.00	0.00	0.00	0.00	0.00	0.00
NNE	0.00	0.00	0.00	0.00	0.00	0.00
NE	0.00	0.00	0.00	0.00	0.00	0.00
ENE	0.00	0.00	0.00	0.00	0.00	0.00
E	6.06	0.00	0.00	0.00	0.00	0.00
ESE	0.00	0.00	0.00	0.00	0.00	0.00
SE	0.00	0.00	0.00	0.00	0.00	0.00
SSE	3.03	0.00	0.00	0.00	0.00	0.00

TOTAL OBS = 33 MISSING OBS = 0

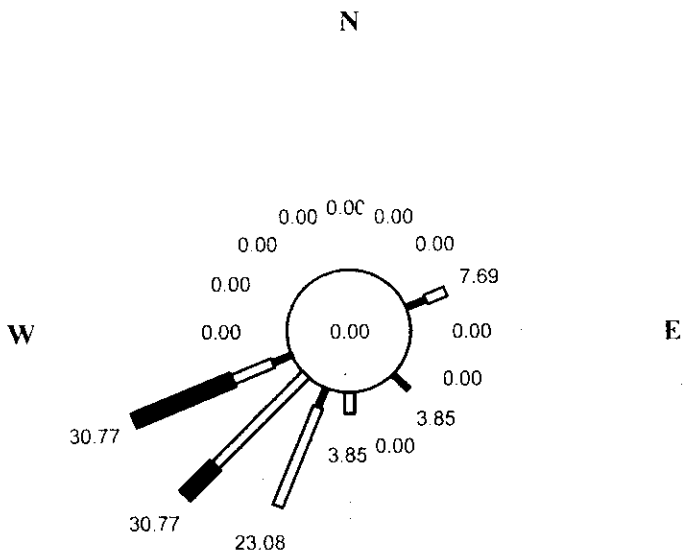
PERCENT OCCURRENCE Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
S	0.00	0.00	0.00	0.00	0.00	0.00
SSW	3.03	0.00	0.00	0.00	0.00	0.00
SW	9.09	27.27	6.06	0.00	0.00	0.00
WSW	0.00	15.15	30.30	0.00	0.00	0.00
W	0.00	0.00	0.00	0.00	0.00	0.00
WNW	0.00	0.00	0.00	0.00	0.00	0.00
NW	0.00	0.00	0.00	0.00	0.00	0.00
NNW	0.00	0.00	0.00	0.00	0.00	0.00

CALM OBS = 0 PERCENT CALM = 0.00

Figure 10

WIND ROSE
OFFICE BUILDING ETS STUDY
APRIL 11, 2003 FROM 09:00 TO 15:15 HRS. (15 MIN. AVG.)



Calms included at center.
Rings drawn at 10% intervals.
Wind flow is FROM the directions shown.
No observations were missing.

PERCENT OCCURRENCE Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
N	0.00	0.00	0.00	0.00	0.00	0.00
NNE	0.00	0.00	0.00	0.00	0.00	0.00
NE	0.00	0.00	0.00	0.00	0.00	0.00
ENE	3.85	3.85	0.00	0.00	0.00	0.00
E	0.00	0.00	0.00	0.00	0.00	0.00
ESE	0.00	0.00	0.00	0.00	0.00	0.00
SE	3.85	0.00	0.00	0.00	0.00	0.00
SSE	0.00	0.00	0.00	0.00	0.00	0.00

TOTAL OBS = 26 MISSING OBS = 0

PERCENT OCCURRENCE Wind Speed (Miles Per Hour)
LOWER BOUND OF CATEGORY

DIR	0.1	3.5	6.9	11.5	18.4	24.2
S	0.00	3.85	0.00	0.00	0.00	0.00
SSW	3.85	19.23	0.00	0.00	0.00	0.00
SW	0.00	23.08	7.69	0.00	0.00	0.00
WSW	3.85	7.69	19.23	0.00	0.00	0.00
W	0.00	0.00	0.00	0.00	0.00	0.00
WNW	0.00	0.00	0.00	0.00	0.00	0.00
NW	0.00	0.00	0.00	0.00	0.00	0.00
NNW	0.00	0.00	0.00	0.00	0.00	0.00

CALM OBS = 0 PERCENT CALM = 0.00

Figure 11

ARB/Nicotine Samples Set #4 Office Building #2
Samples received on 5/23/2003
Date Extracted: 5/27/2003
Date Analyzed: 5/27/2003
Nicotine

Limits Used for µg/m3:
1 hour 0.0058 µg/m3 £ Trace < 0.029 µg/m3
8 hours 0.00073 µg/m3 £ Trace < 0.0036 µg/m3
* Questionable Data

Sample ID	Fortification Level (µg)	µg found	% Rec	Ave % Rec	Stdev	Run Time (min.)	Flow (L/min.)	Vol (L)	Vol (m^3)	µg/m^3
NIC242C		ND								
NIC210TB		ND								
NIC211FB		ND								
NIC243CR25R1	25.0	21.9654	88%							
NIC244CR25R2	25.0	22.1027	88%							
NIC245CR25R3	25.0	21.5859	86%	88%	1%					
NIC212FS25R2	25.0	20.0483	*80%							
NIC213FS25R1	25.0	14.1321	*57%							
NIC214FS25R3	25.0	19.9731	80%							
NIC215TS25R1	25.0	20.6914	83%							
				sampler	date					
NIC216		0.7533		1	20th	480	15	7200	7.20	0.1046
NIC217		0.9167		2	20th	480	15	7200	7.20	0.1273
NIC218		0.8927		3	20th	480	15	7200	7.20	0.1240
NIC219		0.8682		1	21st	480	15	7200	7.20	0.1206
NIC220		1.0956		2	21st	480	15	7200	7.20	0.1522
NIC221		0.3562		3	21st	480	15	7200	7.20	0.0495
NIC222		0.1913		1	22nd-1hr	60	15	900	0.90	0.2126
NIC223		0.1362		2	22nd-1hr	60	15	900	0.90	0.1513
NIC224		0.0353		3	22nd-1hr	60	15	900	0.90	0.0392
NIC225		0.2120		1	22nd-2hr	60	15	900	0.90	0.2356
NIC226		0.2541		2	22nd-2hr	60	15	900	0.90	0.2824
NIC227		0.0643		3	22nd-2hr	60	15	900	0.90	0.0715
NIC228		0.1253		1	22nd-3hr	60	15	900	0.90	0.1392
NIC229		0.2183		2	22nd-3hr	60	15	900	0.90	0.2426
NIC230		0.0651		3	22nd-3hr	60	15	900	0.90	0.0724
NIC231		0.1088		1	22nd-4hr	60	15	900	0.90	0.1209
NIC232		0.1747		2	22nd-4hr	60	15	900	0.90	0.1941
NIC233		0.0655		3	22nd-4hr	60	15	900	0.90	0.0728
NIC234		0.1118		1	22nd-5hr	60	15	900	0.90	0.1242
NIC235		0.1687		2	22nd-5hr	60	15	900	0.90	0.1874
NIC236		0.0686		3	22nd-5hr	60	15	900	0.90	0.0762
NIC237		0.0918		1	22nd-6hr	60	15	900	0.90	0.1020
NIC238		0.2176		2	22nd-6hr	60	15	900	0.90	0.2417
NIC239		0.0289		3	22nd-6hr	60	15	900	0.90	0.0321

C = Resin Blank
TB = Trip Blank
CR = Concurrent Recovery
FS = Field Spike

sampler #1 Next to the west Bank
sampler #2 Next to the east Bank
sampler #3 Background Behind Planter corner of quad area next to alleyway

Table 5

ARB/Nicotine Samples Set #5 Amusement Park
Samples received on 6/20/2003
Date Extracted: 6/23/2003
Date Analyzed: 6/24/2003

Limits Used for µg/m3:
1 hour 0.0058 µg/m3 £ Trace < 0.029 µg/m3
8 hours 0.00073 µg/m3 £ Trace < 0.0036 µg/m3

Nicotine

Sample ID	Fortification Level (µg)	µg found	% Rec	Ave % Rec	Stdev	Run Time (min.)	Flow (L/min.)	Vol (L)	Vol (m^3)	µg/m^3
NIC275C		ND								
NIC246TB		ND								
NIC276CR10R1	10.0	7.7992	78%							
NIC277CR10R2	10.0	7.6141	76%							
NIC278CR10R3	10.0	7.7762	78%	77%	1%					
NIC247TS10R1	10.0	7.1726	72%							
				sampler	**date**					
NIC248		16.9996		1	17th	480	15	7200	7.20	2.3611
NIC249		20.1031		2	17th	480	15	7200	7.20	2.7921
NIC250		0.8938		3	17th	480	15	7200	7.20	0.1241
NIC252		22.2234		1	18th	480	15	7200	7.20	3.0866
NIC253		18.3371		2	18th	480	15	7200	7.20	2.5468
NIC254		0.8754		3	18th	480	15	7200	7.20	0.1216
NIC255		0.7734		1	19th-1hr	60	15	900	0.90	0.8593
NIC256		0.5942		2	19th-1hr	60	15	900	0.90	0.6602
NIC257		0.1399		3	19th-1hr	60	15	900	0.90	0.1554
NIC258		1.7816		1	19th-2hr	60	15	900	0.90	1.9796
NIC259		1.0900		2	19th-2hr	60	15	900	0.90	1.2111
NIC260		0.1262		3	19th-2hr	60	15	900	0.90	0.1403
NIC261		2.8501		1	19th-3hr	60	15	900	0.90	3.1668
NIC262		0.7930		2	19th-3hr	60	15	900	0.90	0.8812
NIC263		0.1524		3	19th-3hr	60	15	900	0.90	0.1694
NIC264		2.7863		1	19th-4hr	60	15	900	0.90	3.0959
NIC265		2.6020		2	19th-4hr	60	15	900	0.90	2.8911
NIC266		0.1210		3	19th-4hr	60	15	900	0.90	0.1344
NIC267		4.1466		1	19th-5hr	60	15	900	0.90	4.6073
NIC268		3.2723		2	19th-5hr	60	15	900	0.90	3.6359
NIC269		0.2367		3	19th-5hr	60	15	900	0.90	0.2630
NIC270		2.7862		1	19th-6hr	60	15	900	0.90	3.0958
NIC271		1.9208		2	19th-6hr	60	15	900	0.90	2.1342
NIC272		0.1407		3	19th-6hr	60	15	900	0.90	0.1564

C = Resin Blank
TB = Trip Blank
CR = Concurrent Recovery
TS = Trip Spike

sampler #1 Lamppost next to water
sampler #2 Entry sign to smoking area
sampler #3 Background Lamppost near fenceline

Table 6

ATTACHMENT I

SAMPLING PROTOCOL

MONITORING PROTOCOL FOR NEAR-SOURCE AMBIENT AIR MONITORING OF NICOTINE AS A MARKER FOR ENVIRONMENTAL TOBACCO SMOKE

Monitoring and Laboratory Division

October 1, 2002

The following protocol has been reviewed and approved by the Air Resources Board (ARB) staff. Approval of this protocol does not necessarily reflect the views and policies of ARB, nor does the mention of trade names or commercial products constitute endorsement of recommendation for use.

Sampling Plan Identification and Approval

Title: Near-Source Ambient Air Monitoring of Nicotine as a Marker for Environmental Tobacco Smoke

Approval: The following sampling plan for monitoring nicotine as a surrogate for environmental tobacco smoke (ETS) in several locations in California is recommended for approval.

Signatures:

9/30/02

Kenneth R. Stroud, Chief — Date
Air Quality Surveillance Branch
Air Resources Board

10/11/02

Janette Brooks, Chief — Date
Air Quality Measures Branch
Air Resources Board

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2.0 Project Objective

Stationary Source Division (SSD) of the Air Resources Board (ARB) has requested that the Monitoring and Laboratory Division (MLD) conduct near-source ambient air sampling for environmental tobacco smoke (ETS). The ARB has contracted with the Trace Analytical Laboratory in the Department of Environmental Toxicology at UC Davis to perform the analytical work. The purpose is to gather data from potential near-source or hot spot areas to which the public is exposed to evaluate ETS ambient air exposure. The information gained in this study will help in ARB's assessment of ETS as a TAC. To do this, the ARB is planning to use nicotine as a marker for the constituents of ETS. Nicotine is a unique marker for the presence of ETS and has been monitored in numerous indoor air studies related to ETS.

3.0 Location

Several potential outdoor smoking areas are being considered for the near-source ETS monitoring study. Some of these potential areas include:

- office building with outdoor smoking area
- amusement park smoking area where children are present
- high school or college outdoor eating/smoking area
- stadium during a sporting event
- apartment or condominium complex

4.0 Sampling Design and Method

The air monitoring for this project will include three samplers per smoking area. Siting criteria permitting, priority will be given to placing at least one sampler within the smoking area and at least one sampler on the perimeter of the smoking area, in the expected downwind direction. The third sampler will be placed away from the smoking area in what is expected to be an upwind location. The samplers, BGI PQ100s, will run at 15 lpm using XAD-4 absorbent sampling media with quantitation limits of 0.01- 0.05 μg/m^3. There will be one collocated sampler at one of the sampling locations chosen by SSD. The sites need not comply with the siting criteria for ambient air monitoring, but every effort will be made to meet the micro-scale monitoring siting criteria in 40 CFR Part 58, Appendix E, and Volume II of ARB's Quality Assurance Manual (Table 1). The flow rates will be checked pre and post following each sample day (Table 2).

Table 1

Item/ Influence	Requirements
Height of Inlet	2 to 7 meters above the ground
Spacing Between Inlets (Collocated)	Within 4 meters, but at least 1 meter apart.
Obstacles	Distance between samplers and obstacles must be at least 2 times the height the obstacle protrudes above the sampler.
Tree Dripline	Inlet must be 10 meters from dripline if tree represents an obstruction.
Walls, Parapets, etc.	Inlet must be 2 meters from the walls, parapets, etc.
Air Flow Arc	Unrestricted 270 degree arc that must include predominant wind direction for seasonal high pollutant; samplers located on the side of a building require 180-degree clearance.
Traffic	5 to 15 meters from roadway

**From 40 CFR Part 58, Appendix E, and ARB Quality Assurance Manual, Vol. II, section 2.0.4 (Feb. 2000)*

A portable meteorological station will be placed at or near the samplers. Ambient temperature, scalar wind speed, scalar wind direction, and relative humidity will be measured continuously and reported in hourly averages. The height above ground of the sensors on the portable meteorological station is approximately 2.5 meters. Data will be presented using wind rose graphs to shows wind patterns and speeds.

Samples will not be collected if rain or strong wind is occurring or expected before the sampling period would end. Also, sampling will not be conducted near greenhouses, due to potential use of nicotine as an insecticide in greenhouses.

5.0 Frequency and Duration of Monitoring

For each ETS study location, a minimum of 3 days, with sampling periods as follows:

- On two sampling days, collect 8-hour samples at the three monitoring locations (approximately 8:30 a.m. to 4:30 p.m. for a total of 6 samples).
- On one sampling day, collect approximately consecutive 1-hour samples for 6-hours at the three monitoring locations between 9:00 a.m. and 4:00 p.m. (total of 18 samples).

6.0 Analysis

The Trace Analytical Laboratory in the Department of Environmental Toxicology at UC Davis will analyze the XAD-4 samples for nicotine. Analysis will be by gas chromatography with mass selective detector.

7.0 Quality Control

Field QC procedures are critical to ensuring the data collected is accurate, relevant, and defensible. So to ensure these measures a National Institute of Standard Technology (NIST)-traceable transfer standard will be used to, calibrate, and verify flow rates, for the BGI PQ-100s and the Meteorological equipment (Auto-Met). Following the end of the study a pre and post calibration form will be used to verify the accuracy of the equipment used in the study (Table 2,3).

Each XAD-4 sampling cartridge will be assigned a sample report which consists of site name, sampler ID, field operator, filter ID/Code, start/end date and time, elapsed time, target flow rate, volume, observed conditions, operator comments, and filter/sample transfer information (Table 4).

Following sampling, XAD-4 samples will be placed with the sample report in an ice chest containing dry ice, until transfer to the UC Davis lab for analysis.

In addition, three field cartridge spikes will be used at two locations, and one trip blank cartridge and one field blank cartridge will be carried to and from the field at all locations. The field blank will be opened in the field, but the trip blank will not be opened.

8.0 Schedule

The near-source monitoring is tentatively scheduled for the fall and winter of 2002.

9.0 Roles and Responsibilities

SSD will pick the sampling sites and obtain approval for site access. SSD will also collect and monitor smoking activity and frequency during each sampling day at each location. All data analysis will be done by SSD.

The UC Davis lab will provide sampling media and media transfer documentation. The lab will perform analysis of sample media and report findings.

The Special Purpose Monitoring Section (SPM) will write the ETS sampling protocol. SPM will also, work with the client on site logistics, pick-up and transport sample media to and from the field, perform air sampling, and write the final ETS report. The final ETS report will include maps, and photos of the smoking areas with sampler locations and meteorological data, and include the UC Davis analytical report as an attachment.

Sampler Flow Check Log

Transfer Standard Information:

*MFM Make & Model:*_____ *Flow Range:*_____

*I.D.(Bar Code):*_____ *Calibration Date:*_____

Flow Check Data:

		Pre-Sampling Flow Check		**Post-Sampling Flow Check**		
Date	Sampler I.D.	Transfer Standard Flow Rate (slpm)	Sampler Flow Rate (slpm)	Transfer Standard Flow Rate (slpm)	Sampler Flow Rate (slpm)	Comments

Table 2

Auto Met X1042 ›sultant Wind Direction

Station Number: Sensor Number: A6978 Cal. Date: 05/17/02

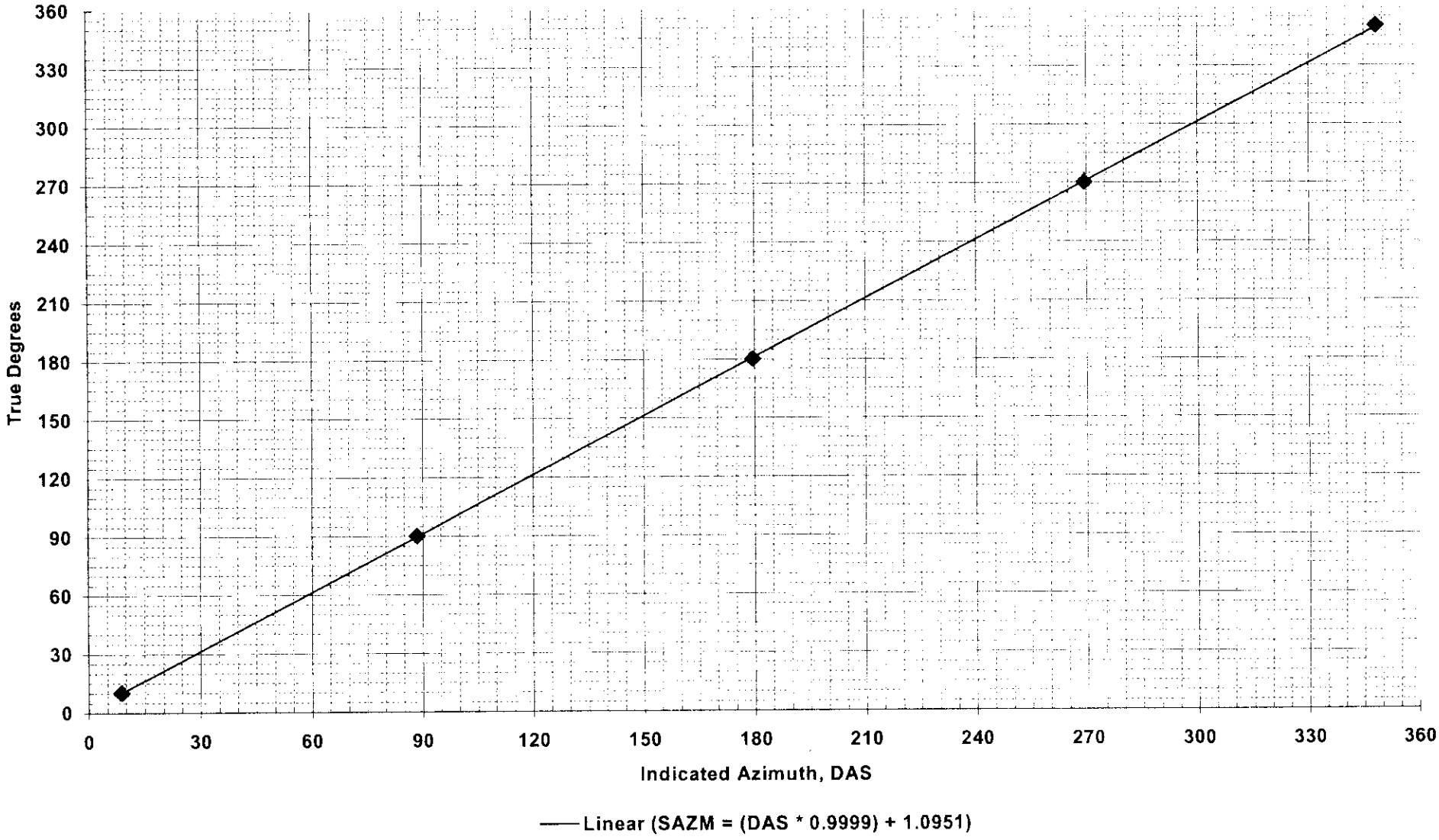

Table 3

Media Sample Report

Project Name: _____

Site Name: _____

Sampler I.D.: _____

Field Operator _____

Filter I.D. or Code: _____

Start Date / Time: _____

End Date / Time: _____

Sample Summary:

Elapsed Time (min)	Target Flow Rate (lpm)	Total Volume Sampled (liters)	Observed Conditions

Observed Conditions

A. No Unusual Conditions
B. Rain / Snow
C. Fog
D. Nearby Smoke / Fire
E. Construction
F. Sampler Malfunction
G. Other (state)

Operator Comments:

_____

_____

_____

Filter / Sample Transfer:

Action	Date	Time	Name		Comments
			Released	Taken	
(Media transferred from lab to site operator					
(Media transfer)					
(Media transfer)					
(Media transfer)					
(Media transferred from site operator to lab					

Table 4

ATTACHMENT II

UCD ANALYTICAL REPORT

FINAL REPORT

September 22, 2003

ANALYSIS OF NICOTINE IN CALIFORNIA
AIR SAMPLES FROM XAD-4 RESIN

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INTRODUCTION

Nicotine is unique to tobacco, is a major constituent of its smoke, and has been used as a marker for environmental tobacco smoke (ETS), also known as second-hand smoke [1,2]. Staff of the Air Resources Board of California (ARB), in an effort to improve estimates of Californians' exposures to ETS, conducted an air sampling study with analysis of nicotine. The Trace Analytical Laboratory (TAL) in the Department of Environmental Toxicology, University of California, Davis was selected to provide air sampler cartridges and analysis of nicotine in air samples. Nicotine is present at >95% vapor phase in ETS [3]. Filter capture, problems with nicotine degradation, extraction, and analysis need to be overcome for efficient estimates of nicotine in air. Several methods of capturing nicotine for analysis from air sampling have been proposed such as filter packs, sorbent beds, annular denuders and passive samplers [2]. The California ARB staff collected air-samples using XAD-4 resin in five different locations in California. Samplers were run at 1 and 8 hour-intervals with 3 or 4 samplers at each location. Sample cartridges were placed on dry ice and transported back to TAL for quantitative nicotine analysis by gas chromatography/mass spectrometry (GC/MS). Trapping efficiencies, method detection limits, concurrent recoveries and storage stabilities were determined to aid the ARB investigation.

MATERIALS AND METHODS

Standard Preparation. Nicotine (98.5%, Lot#267-54A) reference standard, was obtained from Chem Service (Cat. #PS-85, 660 Tower Lane, West Chester, PA), and stored frozen at approx. -20°C except when in use. All solvents and reagents were residue grade or better. Stock solution (1mg/mL) was prepared by dissolving 0.0509 g nicotine into 50 mL acetone. Stock solution was stored frozen.

Calibration standards for GC/MS analysis were prepared in the presence of matrix. For each calibration standard solution, solvent was prepared through the method and the resin extract used for final dilution with 40/40/20 acetone/ethyl acetate/methanol. Dilution of 2, 1, 0.5, 0.2, 0.1 and 0.05 mL of stock solution (1 mg/mL) in 10 mL of 40/40/20 acetone/ethyl acetate/methanol resin extract produced 200, 100, 50, 20, 10 and 5 pg/μL calibration standard solutions. Calibrations standards were stored in the refrigerator (<5°C) and were prepared fresh every two weeks for the course of the study.

XAD-4 Resin Preparation. XAD-4 (Rohm and Haas, Amberlite, Philadelphia, PA, 100-120 mesh), a macro reticular resin, was employed as the trapping medium. XAD-4 resin was prepared prior to use according to Seiber et al. [4] with modifications described below. Resin (~18.7 L) was initially rinsed with methanol in an ~ 40 L container. The fines were removed by placing a hose at the bottom of the container, overfilling with deionized water and stirring vigorously. Two liters of 0.25 *N* hydrochloric acid were added and the resin was stirred for 30 min. Again, water was added and fines with excess water was decanted. The water steps above were repeated until the water above the resin was clear and the pH was that of the deionized water. The resin was then transferred with methanol to gallon bottles. The resin was Soxhlet

extracted once with fresh methanol and ~ 100mL of pyridine for 24 hours, then extracted again with fresh methanol for 24 hours, then extracted twice with fresh ethyl acetate for 24 hours. This pyridine clean up step was added to the original method to clean resin for nicotine capture and subsequent extraction with 0.01% pyridine in acetone/ethyl acetate/methanol. The resin was dried in a vacuum oven (25 in. Hg) until thoroughly dried. Resin batches were numbered and stored at room temperature in clean, dry jars with Teflon®-lined lids. Each solvent step is important for thorough cleanup of the XAD-4 resin.

Trapping Efficiency Test. Preparation of cartridges is described in Hall et al., 1997 [5]. The resin cartridge consisted of a resin bed (~30 mL) held in position with a stainless steel mesh screen. The cartridges were connected in tandem with Teflon® tubing. Tygon® tubing was connected to a Staplex high-volume air pump fitted with a manifold that allowed a flow rate of 15 L per min (Lpm).

Nicotine standard solution was applied directly to the resin bed at 50 μg. Air samples were collected for 1 and 8-hr intervals, (n=4). Resin was extracted as described below and analyzed as described in the quantitation section.

Determination of Method Detection Limit (MDL) and Estimated Quantitation Limit (EQL). Eight samples each were fortified with 0.10μg nicotine for a 1-hr and an 8-hr air sampling interval. Samples were extracted and quantified as below in a final volume of 10 mL. For quantitation at pg/μL, values below MDL were non-detect (ND), values above or equal to MDL but below EQL were trace (TR), and values above EQL were reported at two significant figures. EQL was determined using 10 mL

samples while actual samples were analyzed in 5 mL, so the pg/μL level was the deciding point for non-detect and trace amounts reported.

Storage Stability. Jars with 30 mL of pyridine washed XAD-4 resin were each fortified with 1 μg of nicotine. Six jars were analyzed through the method below on Day 0 and the remaining jars were stored at –20±6°C. Six jars were removed and analyzed as below on Day 29.

Collection of Air Samples. Air samplers were placed at various sites in California and samples were taken for 1-hr and 8-hr periods. Usually 3 air pumps were used at a site, and cartridges changed for the appropriate time interval. Cartridges were stored in freezer boxes after collection and delivered to the TAL facility. Samples were assigned unique numbers and analyzed as below.

Extraction of Air Samples. The cap and screen were removed from the resin cartridge and poured into a 4 oz. jar. The remaining resin was transferred by carefully rinsing the cartridge using 75 mL of 0.01% of pyridine in 40/40/20 acetone/ethyl acetate/methanol (extraction solvent) into the jar and capping with a Teflon®-lined lid. Concurrent fortifications were prepared at this point by adding clean resin to a jar, then adding appropriate standard and 75 mL of extraction solvent. Jars are mechanically swirled, on a rotary platform shaker for one hour at a moderate speed. The extraction solvent was decanted into a 500-mL round bottom flask (RBF) through a funnel with glass wool. The resin was re-extracted with an additional 75 mL of extraction solvent and swirled for 30 min. Pooling the decanted extraction solvent from the jar, resin was re-extracted a final time with 75 mL extraction solvent for 30 min. The pooled extract in the RBF was concentrated to 1-2 mL using a rotary evaporator with an ~35°C water bath.

The sample was diluted to an appropriate volume for GC/MS analysis with 40/40/20 acetone/ethyl acetate/methanol.

Instrumentation and Quantitation. Nicotine analysis was performed using a Hewlett Packard (HP, Avondale, PA) 6890, equipped with a 30 m x 0.25 mm DB-17ms column (0.25 µm film thickness, J&W Scientific, Folsom, CA) and Mass Selective Detector (MSD) Model 5973, in Selective Ion Monitoring (SIM) Mode. The inlet was in pulsed splitless mode with the injection pressure pulse 50 psi for 1 min and the injector purge at 50 mL/min for 0.95 min. A HP 6890 autosampler was used to make 3 µl injections. The injector was heated at 250°C, the MSD interface at 280°C, MSD source at 230°C, the MSD quadrapole at 150°C and the column at 80°C for 1 min then ramped up by 20°C/min to 280°C. The retention time was 6.44 min for nicotine. The quantitation ion (m/z, mass/charge ratio) was 84 (Dwell time = 50 milliseconds x 2) and the confirmation ion (m/z) was 162. Prior to each analytical set, the analyst performed an autotune and a tune evaluation of the MSD to insure proper function. In addition, calibration standards were injected with each run to check GC/MS performance.

The data system was HP ChemStation® G1701BA version B.01.00. Peak areas from calibration standards were used to generate a linear standard curve (nicotine response vs. concentration pg/µL). The average of replicate injections of each sample was reported. Average peak areas from samples were converted to pg/µL by using the linear regression from the standard curve. The sample concentration was multiplied by the final sample volume resulting in µg/sample. Fortified samples yielded a percent recovery by dividing the µg/sample by the fortification amount. If the peak area for nicotine was 10% larger than the highest standard value, the sample was diluted and

reinjected. For sample values above EQL, 10% of the samples were assessed by ion ratio comparison for nicotine.

RESULTS AND DISCUSSION

Difficulties with trapping efficiencies for nicotine were curtailed by addition of pyridine to the extraction solvent and pre-washing the XAD-4 collection resin with dilute pyridine. Trapping efficiencies were 82 ± 6% for the 1-hour study and 69 ± 3% for the 8-hour study, with 90 ± 1% concurrent recoveries (fortified at extraction). These trapping efficiencies were sufficient and comparable to other XAD-4 studies [2,6].

The method detection limit (MDL) is considered to be the t-value (2.998 for n=8) times the standard deviation. Results for the 1-hour air sampling study showed an average of 9.14 ± 0.35 pg/μL recovered. MDL was calculated as 1.05 pg/μL (0.35 × 2.998). Estimated quantitation limit (EQL) was calculated as 5.25 pg/μL (MDL × 5): thus, non detect (ND) < 1.05 < Trace <5.25 pg/μL. When calculated for 5 mL samples, the limits used for 1-hour were ND < 0.0058 ≤ Trace <0.029 $\mu g/m^3$ and for 8-hour ND < 0.00073 ≤ Trace <0.0036μg/m3. Results for the 8-hour air sampling study showed an average recovery of 10.22 ± 0.18 pg/μL, an MDL of 0.53 pg/μL and an EQL of 2.66 pg/μL. Because the standard deviation for the 8-hour set was significantly less than the 1-hour set, and when calculated was well below our actual instrument sensitivity, the MDL and EQL at pg/μL for the 1-hour set was used throughout the study.

Storage stability samples were analyzed 29 days after fortification. Recovery averaged 103 ± 5% for n=6. Concurrent recoveries run with those samples averaged 89 ± 4% for n=6. No apparent degradation of nicotine occurred in spiked frozen cartridges over 29 days.

For each set of samples received from ARB, concurrent recoveries were run. For the entire study concurrent recoveries averaged 85 ± 7%. Trip/field spikes, which were fortified by TAL, averaged 80 ± 9% recovery.

Results from samples collected by staff of the California ARB are presented in Table 1, for 1-hour and 8-hour sampling intervals. (The raw data sheets from each of the monitoring locations are presented in Attachment A.) General locations were an airport, a junior college, a local government building, an office building, and an amusement park. Samples taken at the amusement park had the highest levels of nicotine. The highest 1-hr sample was 4.607 $\mu g/m^3$ nicotine; the highest 8-hr sample was 3.096 $\mu g/m^3$. The lowest values were found at junior college.

References

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2. Caka, M.; Eatough, D.; Lewis, E.; Tang, H.; Hammond, S.;Leaderer, B.; Kourakis, P.; Spengler, J.; Fasano, A.; McCarthy, J.; Ogden, M.; Lewtas, J. An Intercomparison of Sampling Techniques for Nicotine in Indoor Environments. *Environ. Sci. Technol.* **1990**, *24*, 1196-1203.

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5. Hall, G.; Mourer, C.; Shibamoto, T.; Fitzell, D. Development and validation of an analytical method for naled and dichlorvos in air. *J. Agric. Food Chem.* **1997**, *45*, 145-148.

6. Ogen, M.; Eudy, L.; Heaver, D.; Conrad, F.; Green, C. Improved Gas Chromatographic Determination of Nicotine in Environmental Tobacco Smoke. *Analyst.* **1989**, *114*, 1005-1008.

Table 1. Results of analysis of nicotine in air samples from 5 different California locations in µg/m^3.

ND = non detect , TR = Trace, NP = No Pump (malfunction).
Limits Used for µg/m^3:

1 Hour ND < 0.0058≤ Trace< 0.029 µg/m^3

8 Hour ND < 0.00073≤ Trace< 0.0036 µg/m^3

	Airport			Junior College				Local Government Center			Office Complex			Amusement Park		
Site/Pump	1	2	3	1	2	3	4	1	2	3	1	2	3	1	2	3
1 Hour	1.114	0.556	0.0565	TR	TR	0.0359	TR	0.176	0.145	TR	0.213	0.151	0.0392	0.859	0.660	0.155
	0.649	0.588	0.0472	TR	TR	0.0377	TR	0.172	0.0797	TR	0.236	0.282	0.0715	1.980	1.211	0.140
	1.032	0.517	0.0454	0.0434	0.0752	0.0556	TR	0.0702	0.0601	TR	0.139	0.243	0.0724	3.167	0.881	0.169
	0.620	0.553	0.0525	NP	0.0484	0.0462	TR	NP	0.131	TR	0.121	0.194	0.0728	3.096	2.891	0.134
	0.663	1.498	0.0417	0.0444	NP	0.0650	TR	0.0387	0.0822	ND	0.124	0.187	0.0762	4.607	3.636	0.263
	0.362	0.439	0.0355	0.0960	0.146	0.0525	TR	0.0463	0.0702	TR	0.102	0.242	0.0321	3.096	2.134	0.156
8 Hour	0.490	0.990	0.0185	0.0316	0.0437	0.0296	0.0183	0.0592	0.0731	0.0102	0.105	0.127	0.124	2.361	2.792	0.124
	0.480	0.731	0.0228	0.0273	0.0151	0.0129	0.00474	0.0417	0.0674	0.00771	0.121	0.152	0.0495	3.087	2.547	0.122

Attachment A

ARB/Nicotine Samples Set #1
Samples received on 1/17/2003 — Airport
Date Extracted: 1/21/2003
Date Analyzed: 1/24/2003
Nicotine

Sample ID	Sampler #	Fortification Level (µg)	µg found	% Rec	Ave % Rec	Stdev	Run Time (min.)	Flow (L/min.)	Vol (L)	Vol (m^3)	µg/m^3
NIC133C			Trace								
NIC102TB			Trace								
NIC103FB			Trace								
NIC134CR400R1		400.0	366.164	92%							
NIC135CR400R2		400.0	378.494	95%							
NIC136CR400R3		400.0	353.990	88%	92%	3%					
NIC104FS400R1		400.0	341.906	85%			480	15	7200	7.20	
NIC105FS400R2		400.0	349.841	87%			480	15	7200	7.20	
NIC106FS400R3		400.0	344.127	86%	86%	1%	60	15	900	0.90	
NIC109	3		0.0508				60	15	900	0.90	0.0565
NIC110	1		0.9857				59	15	885	0.89	1.1137
NIC111	2		0.5001				60	15	900	0.90	0.5557
NIC112	3		0.0425				60	15	900	0.90	0.0472
NIC113	1		0.5843				60	15	900	0.90	0.6492
NIC114	2		0.5294				60	15	900	0.90	0.5882
NIC115	3		0.0409				60	15	900	0.90	0.0454
NIC116	1		0.9290				60	15	900	0.90	1.0323
NIC117	2		0.4655				60	15	900	0.90	0.5172
NIC118	3		0.0473				60	15	900	0.90	0.0525
NIC119	1		0.5578				60	15	900	0.90	0.6198
NIC120	2		0.4978				60	15	900	0.90	0.5531
NIC121	3		0.0375				60	15	900	0.90	0.0417
NIC122	2		1.3484				60	15	900	0.90	1.4982
NIC123	1		0.5972				60	15	900	0.90	0.6636
NIC124	3		0.0320				60	15	900	0.90	0.0355
NIC125	1		0.3260				60	15	900	0.90	0.3622
NIC126	2		0.3949				60	15	900	0.90	0.4388
NIC127	3		0.1334				480	15	7200	7.20	0.0185
NIC128	2		7.1266				480	15	7200	7.20	0.9898
NIC129	1		3.5275				480	15	7200	7.20	0.4899
NIC130	3		0.1641				480	15	7200	7.20	0.0228
NIC131	2		5.2664				480	15	7200	7.20	0.7314
NIC132	1		3.4598				480	15	7200	7.20	0.4805

C = Resin Blank
TB = Trip Blank
FB = Field Blank
CR = Concurrent Recovery
FS = Field Spike

Limits Used for µg/m^3:

1 hour	0.0058 µg/m3 ≤ Trace < 0.029 µg/m3
8 hours	0.00073 µg/m3 ≤ Trace < 0.0036 µg/m3

ARB/Nicotine Samples Set #2
Samples received on 4/8/2003
Date Extracted: 4/8/2003
Date Analyzed: 4/9/2003
Nicotine

Junior College

Sample ID	Sampler #	Fortification Level (µg)	µg found	% Rec	Ave % Rec	Stdev	Run Time (min.)	Flow (L/min.)	Vol (L)	Vol (m^3)	µg/m^3
NIC176C			Trace								
NIC137TB			Trace								
NIC177CR100R1		100.0	79.110	79%							
NIC178CR100R2		100.0	74.445	74%							
NIC179CR100R3		100.0	81.510	82%	78%	4%					
NIC175FS100R1		100.0	83.383	83%							
NIC138TS100R1		100.0	82.274	82%							
NIC139	1		0.2279				480	15	7200	7.20	0.0316
NIC140	2*		0.3147				480	15	7200	7.20	0.0437
NIC141	3		0.2131				480	15	7200	7.20	0.0296
NIC142	4		0.1320				480	15	7200	7.20	0.0183
NIC143	1		0.1962				480	15	7200	7.20	0.0273
NIC144	2*		0.1090				480	15	7200	7.20	0.0151
NIC145	3		0.0930				480	15	7200	7.20	0.0129
NIC146	4		0.0341				480	15	7200	7.20	0.00474
NIC147	1		0.0222				60	15	900	0.90	Trace
NIC148	2*		0.0209				60	15	900	0.90	Trace
NIC149	3		0.0323				60	15	900	0.90	0.0359
NIC150	4		0.0127				60	15	900	0.90	Trace
NIC151	1		0.0156				60	15	900	0.90	Trace
NIC152	2*		0.0169				60	15	900	0.90	Trace
NIC153	3		0.0339				60	15	900	0.90	0.0377
NIC154	4		0.0188				60	15	900	0.90	Trace
NIC155	1		0.0391				60	15	900	0.90	0.0434
NIC156	2*		0.0676				60	15	900	0.90	0.0752
NIC158	4		0.0235				60	15	900	0.90	Trace
NIC160	2		0.0436				60	15	900	0.90	0.0484
NIC161	3		0.0416				60	15	900	0.90	0.0462
NIC162	4		0.0179				60	15	900	0.90	Trace
NIC163	1		0.0399				60	15	900	0.90	0.0444
NIC165	3		0.0585				60	15	900	0.90	0.0650
NIC166	4		0.0198				60	15	900	0.90	Trace
NIC167	1		0.0864				60	15	900	0.90	0.0960
NIC168	2*		0.1316				60	15	900	0.90	0.1463
NIC169	3		0.0473				60	15	900	0.90	0.0525
NIC170	4		0.0245				60	15	900	0.90	Trace
NIC171	3		0.0500				60	15	900	0.90	0.0556

C = Resin Blank
TB = Trip Blank
CR = Concurrent Recovery
FS = Field Spike
* = Collocated Sample

Limits Used for µg/m^3:

1 hour	0.0058 µg/m3 ≤ Trace < 0.029 µg/m3
8 hours	0.00073 µg/m3 ≤ Trace < 0.0036 µg/m3

ARB/Nicotine Samples Set #3
Samples received on 4/14/2003
Date Extracted: 4/14/2003
Date Analyzed: 4/14/2003
Nicotine

Govt Center

Sample ID	Sampler #	Fortification Level (µg)	µg found	% Rec	Ave % Rec	Stdev	Run Time (min.)	Flow (L/min.)	Vol (L)	Vol (m^3)	µg/m^3
NIC206C			ND								
NIC102TB			ND								
NIC207CR50R1		50.0	47.51	95%							
NIC208CR50R2		50.0	44.70	89%							
NIC209CR50R3		50.0	47.10	94%	93%	3%					
NIC182FS50R1		50.0	38.00	76%							
NIC181TS50R1		50.0	44.67	89%							
NIC172	1		0.4262				480	15	7200	7.20	0.0592
NIC173	2		0.5261				480	15	7200	7.20	0.0731
NIC174	3		0.0734				480	15	7200	7.20	0.0102
NIC183	1		0.3006				480	15	7200	7.20	0.0417
NIC184	2		0.4856				480	15	7200	7.20	0.0674
NIC185	3		0.0555				480	15	7200	7.20	0.00771
NIC186	1		0.1581				60	15	900	0.90	0.1756
NIC187	2		0.1302				60	15	900	0.90	0.1447
NIC188	3		0.0249				60	15	900	0.90	Trace
NIC189	1		0.1551				60	15	900	0.90	0.1724
NIC190	2		0.0717				60	15	900	0.90	0.0797
NIC191	3		0.0199				60	15	900	0.90	Trace
NIC192	1		0.0632				60	15	900	0.90	0.0702
NIC193	2		0.0541				60	15	900	0.90	0.0601
NIC194	3		0.0187				60	15	900	0.90	Trace
NIC195	1		ND				NA	NA	NA	NA	NA
NIC196	2		0.1178				60	15	900	0.90	0.1309
NIC197	3		0.0150				60	15	900	0.90	Trace
NIC198	1		0.0348				60	15	900	0.90	0.0387
NIC199	2		0.0740				60	15	900	0.90	0.0822
NIC200	3		ND				60	15	900	0.90	ND
NIC201	1		0.0417				60	15	900	0.90	0.0463
NIC202	2		0.0632				60	15	900	0.90	0.0702
NIC203	3		0.0249				60	15	900	0.90	Trace

C = Resin Blank
TB = Trip Blank
CR = Concurrent Recovery
FS = Field Spike

Limits Used for µg/m^3:

1 hour	0.0058 µg/m3 ≤ Trace < 0.029 µg/m3
8 hours	0.00073 µg/m3 ≤ Trace < 0.0036 µg/m3

ARB/Nicotine Samples Set #4
Samples received on 5/23/2003
Date Extracted: 5/27/2003
Date Analyzed: 5/27/2003
Nicotine

Office Complex

Sample ID	Sampler #	Fortification Level (µg)	µg found	% Rec	Ave % Rec	Stdev	Run Time (min.)	Flow (L/min.)	Vol (L)	Vol (m^3)	µg/m^3
NIC242C			ND								
NIC210TB			ND								
NIC211FB			ND								
NIC243CR25R1		25.0	21.97	88%							
NIC244CR25R2		25.0	22.10	88%							
NIC245CR25R3		25.0	21.59	86%	88%	1%					
NIC212FS25R2		25.0	20.05	80%							
NIC213FS25R1		25.0	14.13	57%							
NIC214FS25R3		25.0	19.97	80%							
NIC215TS25R1		25.0	20.69	83%							
NIC216	341		0.7533				480	15	7200	7.20	0.1046
NIC217	353		0.9167				480	15	7200	7.20	0.1273
NIC218	347		0.8927				480	15	7200	7.20	0.1240
NIC219	341		0.8682				480	15	7200	7.20	0.1206
NIC220	353		1.0956				480	15	7200	7.20	0.1522
NIC221	347		0.3562				480	15	7200	7.20	0.0495
NIC222	341		0.1913				60	15	900	0.90	0.2126
NIC223	353		0.1362				60	15	900	0.90	0.1513
NIC224	347		0.0353				60	15	900	0.90	0.0392
NIC225	341		0.2120				60	15	900	0.90	0.2356
NIC226	353		0.2541				60	15	900	0.90	0.2824
NIC227	347		0.0643				60	15	900	0.90	0.0715
NIC228	341		0.1253				60	15	900	0.90	0.1392
NIC229	353		0.2183				60	15	900	0.90	0.2426
NIC230	347		0.0651				60	15	900	0.90	0.0724
NIC231	341		0.1088				60	15	900	0.90	0.1209
NIC232	353		0.1747				60	15	900	0.90	0.1941
NIC233	347		0.0655				60	15	900	0.90	0.0728
NIC234	341		0.1118				60	15	900	0.90	0.1242
NIC235	353		0.1687				60	15	900	0.90	0.1874
NIC236	347		0.0686				60	15	900	0.90	0.0762
NIC237	341		0.0918				60	15	900	0.90	0.1020
NIC238	353		0.2176				60	15	900	0.90	0.2417
NIC239	347		0.0289				60	15	900	0.90	0.0321

C = Resin Blank
TB = Trip Blank
CR = Concurrent Recovery
FS = Field Spike
TS = Trip Spike
FB = Field Blank

Limits Used for µg/m^3:

1 hour	0.0058 µg/m3 ≤ Trace < 0.029 µg/m3
8 hours	0.00073 µg/m3 ≤ Trace < 0.0036 µg/m3

ARB/Nicotine Samples Set #5
Samples received on 6/20/2003
Date Extracted: 6/23/2003
Date Analyzed: 6/24/2003
Nicotine

Amusement Park

Sample ID	Sampler #	Fortification Level (µg)	µg found	% Rec	Ave % Rec	Stdev	Run Time (min.)	Flow (L/min.)	Vol (L)	Vol (m^3)	µg/m^3
NIC275C			ND								
NIC246TB			ND								
NIC276CR10R1		10.0	7.80	78%							
NIC277CR10R2		10.0	7.61	76%							
NIC278CR10R3		10.0	7.78	78%	77%	1%					
NIC247TS10R1		10.0	7.17	72%							
NIC248	1		16.9996				480	15	7200	7.20	2.3611
NIC249	2		20.1031				480	15	7200	7.20	2.7921
NIC250	3		0.8938				480	15	7200	7.20	0.1241
NIC252	1		22.2234				480	15	7200	7.20	3.0866
NIC253	2		18.3371				480	15	7200	7.20	2.5468
NIC254	3		0.8754				480	15	7200	7.20	0.1216
NIC255	1		0.7734				60	15	900	0.90	0.8593
NIC256	2		0.5942				60	15	900	0.90	0.6602
NIC257	3		0.1399				60	15	900	0.90	0.1554
NIC258	1		1.7816				60	15	900	0.90	1.9796
NIC259	2		1.0900				60	15	900	0.90	1.2111
NIC260	3		0.1262				60	15	900	0.90	0.1403
NIC261	1		2.8501				60	15	900	0.90	3.1668
NIC262	2		0.7930				60	15	900	0.90	0.8812
NIC263	3		0.1524				60	15	900	0.90	0.1694
NIC264	1		2.7863				60	15	900	0.90	3.0959
NIC265	2		2.6020				60	15	900	0.90	2.8911
NIC266	3		0.1210				60	15	900	0.90	0.1344
NIC267	1		4.1466				60	15	900	0.90	4.6073
NIC268	2		3.2723				60	15	900	0.90	3.6359
NIC269	3		0.2367				60	15	900	0.90	0.2630
NIC270	1		2.7862				60	15	900	0.90	3.0958
NIC271	2		1.9208				60	15	900	0.90	2.1342
NIC272	3		0.1407				60	15	900	0.90	0.1564

C = Resin Blank
TB = Trip Blank
CR = Concurrent Recovery
TS = Trip Spike

Limits Used for µg/m^3:

1 hour	0.0058 µg/m3 ≤ Trace < 0.029 µg/m3
8 hours	0.00073 µg/m3 ≤ Trace < 0.0036 µg/m3

APPENDIX D

Estimated Los Angeles Background Ambient ETS Fine PM Concentrations

As Approved
by the Scientific Review Panel
on June 24, 2005

APPENDIX D

Estimated Los Angeles Background Ambient ETS Fine PM Concentration

Introduction

In this report, staff presents an exposure assessment based in part on quantitative estimates of time-weighted exposure for realistic scenarios which illustrate that Californians experience a range of ETS exposures depending upon lifestyle and daily routine. However, 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 the background ETS level which results from the contribution of steady state ETS emissions that routinely occur. The ETS background level in a small rural town may be undetectable due to its smoker population. But, the ETS concentration found in an urban area will be higher due to greater smoker population density and number of tobacco products smoked. Since most of California's population lives and works in urban areas, it would be helpful to ascertain what outdoor ambient ETS levels could be occurring in these areas. There is very limited published information on ambient ETS levels. Therefore, to calculate an urban ETS concentration, ARB staff estimated an outdoor ambient annual average ETS fine particulate matter (PM) concentration (i.e., $PM_{2.5}$ or less) for the Los Angeles area for 2003.

Background

The Los Angeles area estimate is derived from data collected from studies by Schauer *et al*. (1996) and Rogge *et al*. (1994). Both these studies estimated ETS fine particulate concentrations in the Los Angeles area using 1982 data. The Schauer *et al*. (1996) study determined a source apportionment of fine particulate mass concentrations and estimated a 1982 fine particulate annual average concentration of cigarette smoke through a chemical mass balance receptor model based on organic compounds. This model applied atmospheric organic compound concentration data and source emission profile data collected specifically for testing this model (Gray *et al*., 1986; Hildemann *et al*., 1991). The fine PM samples were collected from four sampling sites throughout the Los Angeles area: West Los Angeles, Downtown Los Angeles, Pasadena, and Rubidoux. Schauer *et al*. (1996), estimated the average 1982 fine PM annual average for ETS from these four sampling sites in the Los Angeles area to be 0.21 $\mu g/m^3$ by using the fine PM concentration data and source emission profile data.

The Rogge *et al*. (1994) study found that iso- and anteisoalkanes (C_{29}-C_{34}) are enriched in ETS particles and displays a concentration pattern characteristic of tobacco leaf surface waxes. These iso- and anteisoalkane (C_{29}-C_{34}) concentrations are distinctly different from leaf surface abrasion products shed from plant leaves that grow in the Los Angeles area and contain 40-times more in tobacco and ETS particles than leaf surface waxes from Los Angeles area plants. Four different cigarette categories – nonfilter, filter, light, and menthol were used. For each cigarette category, one of the five most popular cigarette brands was tested to determine an average emission rate for ETS fine

PM. Exhaled mainstream and sidestream smoke generated by human smokers were collected. Isoalkane and anteisoalkane emission rates were then determined from fine particulate ETS per cigarette (Table 1, Rogge *et al*., 1994). Rogge *et al*. (1994) then utilized 1982 ambient isoalkane and anteisoalkane monitoring data for the Los Angeles area (West Los Angeles, downtown Los Angeles, and Pasadena monitors) to estimate an isoalkane/anteisoalkane concentration. By using a fine particulate mass emission rate per cigarette from Hildemann *et al*. (1991), Rogge *et al*. (1994) estimated ambient ETS marker concentrations by the emission rate ratio of fine PM to isoalkanes, multiplied by the 1982 ambient isoalkane and anteisoalkane concentrations. The average 1982 Los Angeles outdoor ambient fine particulate cigarette smoke concentration was found by Rogge, *et al.* (1994) to be approximately 0.28 - 0.36 µg/m^3.

Staff Estimate

The Rogge *et al*. (1994) and Schauer *et al*. (1996) studies estimated annual average ETS fine particulate concentrations in the Los Angeles area for the year 1982. To estimate a 2003 Los Angeles annual average ETS fine particulate concentration, staff applied an adjustment to the 1982 PM estimates to reflect reductions in cigarette consumption and cigarette PM emission rates between 1982 and 2003 (Table D-1).

Table D-1

Estimated Ambient ETS Fine PM Concentration for the Los Angeles Area

		1982	2003	% Difference
Statewide Emissions	Total California Cigarettes	*57.3 billion	*23.5 billion	59
	ETS PM Emission Rate	20.4 mg/cig (1981 data)	13.4 mg/cig (1994-1998 data)	33
	Statewide ETS Fine PM Emissions (tons/year)	1,290	348	73
Estimated Los Angeles Conc.	Modeled ETS PM conc. (µg/m^3) Schauer *et al.* (1996)	0.21	** 0.06	
	Measured ETS PM conc. (µg/m^3) Rogge *et al.* (1994)	0.28 – 0.36	** 0.08 – 0.10	

* CBOE (2004).

** Estimated 2003 ambient ETS fine particulate concentration.

Methodology

We compared the estimated statewide ETS PM emissions for 1982 and 2003 to determine what change had occurred in mass emissions. A mass emission for Los Angeles only was not performed due to the lack of detailed cigarette sales data. ETS emissions were derived by multiplying cigarette sales by the per cigarette PM emission rate. As Table D-1 indicates, estimated ETS PM mass emissions declined from 1,290 tons per year to 348 tons per year (73% difference) between 1982 and 2003. This was due to two major factors.

The main reason for such a dramatic reduction was a significant reduction in cigarette sales over time. Statewide cigarette sales data compiled by the California Board of Equalization (CBOE) between 1982 and 2003 indicated that sales had dropped by about 60% (CBOE, 2004). Secondly, staff believes that our estimated PM inventory for 2003 would be more accurate if the "per cigarette PM emission rate" is updated from the value used for the 1982 estimate. Both the Schauer *et al.* (1996) and Rogge *et al.* (1994) studies use an emission rate (20.4 mg/cigarette) derived by Hildemann *et al.* (1991) for popular brands in 1982. More current studies by Nelson *et al.* (1997), Martin *et al.* (1997), and Repace (2001), result in an emission rate of 13.4 mg/cigarette on average for the popular brands of the 1990's. The Federal Trade Commission (FTC) has also shown that tar content has declined from 1982 to 2000 (FTC, 2000). Since tar is defined as total PM minus moisture and alkaloids (i.e., nicotine), a reduction in tar means a reduction in PM as well. So, we believe it is appropriate to use an updated PM emission rate.

To calculate the 2003 Los Angeles annual average ETS fine particulate concentration, we assumed that: 1) the ratio of fine particulate-emitting sources and fine particulate ambient concentrations from 1982 are comparable to those that exist today, and 2) the decline from 1982 to 2003 in statewide ETS PM emissions (73%) correlates to a linear mass reduction in the outdoor ambient ETS fine PM concentration.

By using the modeled Schauer *et al*. (1996) and the measured Rogge *et al*. (1994) ETS PM concentrations (0.21 $\mu g/m^3$ and 0.28 – 0.36 $\mu g/m^3$, respectively) for 1982, and assuming a 73% reduction in ETS PM concentrations, the Los Angeles area annual average ETS fine particulate concentration range is estimated to be 0.06 - 0.10 $\mu g/m^3$ (Table D-1) using the following equation:

$$\text{2003 ETS PM Concentration } (\mu g/m^3) = C_{1982} \times 0.27$$

Where: C_{1982} = 1982 ETS PM Concentration ($\mu g/m^3$)
0.27 = 73% decrease in ETS emissions from 1982 to 2003

In addition, nicotine emission factor studies (Nelson, 1994; Martin *et al*., 1997) indicated the ratio of ETS-derived-PM to ETS-derived-nicotine is about 8:1.

Thus, the range for ETS nicotine concentrations in Los Angeles is estimated to be about 0.008 - 0.013 µg/m^3 (Table D-2). By comparison, the CARB monitoring study showed 8-hour background nicotine levels in Los Angeles to be 0.009 - 0.12 µg/m^3. The CARB 8-hour monitoring had an estimated quantitation limit of 0.0036 µg/m^3.

Table D-2

Estimated Range of Ambient ETS PM and Nicotine Concentrations for the Los Angeles Area

Urban Location	Year	ETS Fine PM Concentration (µg/m^3)	ETS Nicotine Concentration (µg/m^3)
Los Angeles Area	2003	0.06 – 0.10	0.008 – 0.013
CARB Monitoring Study Los Angeles Area	2003		* 0.009 – 0.12

* Background as measured from two Los Angeles areas.

Conclusion

Since many Californians experience a majority of their personal ETS exposure from a background outdoor ambient level, it is helpful to estimate these levels. The staff used previous Los Angeles area studies, applied an adjustment factor, which included current cigarette sales and emissions data, to estimate an annual average fine PM concentration of 0.06 - 0.10 µg/m^3 in Los Angeles air.

A more accurate assessment of California ambient ETS levels would require additional research to develop more accurate present day concentration data for use in an updated source apportionment study.

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