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California Air Resources Board
Research Division
2020 L Street
Sacramento, CA 95814

Prepared by:

Shelly L. Miller, Steven Branoff, Younhee Lim, Deling Liu,
Michael D. Van Loy, and William W. Nazaroff

Department of Civil and Environmental Engineering
631 Davis Hall
University of California
Berkeley, CA 94720-1710

Department of Mechanical Engineering
Department of Applied Mathematics
Campus Box 427
University of Colorado
Boulder, CO 80309-0427

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Abstract

Little is known about the sources or magnitudes of exposure to more than 190 compounds designated by California legislation as "toxic air contaminants" (TACs). This study estimates the contribution of environmental tobacco smoke (ETS) to the exposure of nonsmoking Californians for 17 of these compounds known to be emitted from burning tobacco. Two distinct approaches were used in the assessment: (a) measured 24-h personal exposures to selected compounds, such as benzene, were compared for individuals who reported ETS exposure against those who reported no exposure; and (b) information on activity patterns of Californians was combined with estimates of ETS concentrations in indoor environments. The first method was applied for nonsmokers (age ≥ 7 y) for the mid-to-late 1980's. The second method was separately applied for adults, adolescents, and children for both the mid-to-late 1980's and for the late 1990's. Averaged over all nonsmoking Californians in the late 1980's, ETS is estimated to have contributed 5-15 $\mu g\ h\ m^{-3}$ to daily benzene exposure, corresponding to 2-5% of the total inhalation exposure of nonsmokers. Among those nonsmokers exposed to ETS, average exposure for adolescents was in the range 65-95% of the average for adults; the corresponding range for children was 80-130%. In the late 1990's, as a result of reduced smoking prevalence among adults and legislation that severely restricts smoking in public buildings, ETS exposures are estimated to be reduced. The fraction of adult nonsmokers exposed to ETS indoors on a given day is predicted to have declined from 52% to 16-19% during the last decade. For adolescents, the corresponding change is from 63% to 33-35%, whereas for children the reduction is from 33% to 21-23% exposed. Among individuals still exposed, the average level of exposure is not predicted to have changed markedly. Using emission factor data, ETS contributions to exposure are estimated for these compounds: acetaldehyde, acetonitrile, acrylonitrile, benzene, 1,3-butadiene, 2-butanone, o-cresol, m,p-cresol, ethyl acrylate (upper bound, only), ethylbenzene, formaldehyde, n-nitrosodimethylamine, phenol, styrene, toluene, o-xylene, and m,p-xylene.

Executive Summary

Background

In California, Assembly Bills 1807 and 2728 established the objective of assessing and controlling exposure to more than 190 compounds designated as "toxic air contaminants" (TACs). The California Health and Safety Code Section 39660.5 requires that indoor exposures to candidate toxic air contaminants (TACs) be considered during exposure and risk assessments. In particular, the Air Resources Board (ARB) Indoor Air Quality and Personal Exposure Assessment Program is charged with generating the data necessary for making realistic exposure assessments to indoor pollutants.

For most of these compounds, little information is available on human exposures, and even less on the relative contributions to exposure of sources. The aim of our study was to estimate the contribution of environmental tobacco smoke (ETS) to the exposure of nonsmoking Californians' to selected TACs.

Objectives

Five specific objectives were pursued in this project:

(1) Determine the frequency distribution of exposure to selected toxic air contaminants from ETS for the California population.
(2) Determine the proportion of Californians' total exposure to air toxics that can be attributed to ETS for selected compounds over the period 1984-1990.
(3) Explore the relative amounts of exposure in the workplace and other public spaces versus exposure in residential settings.
(4) Predict the impact of changes in smoking policy and smoking behavior on exposures to ETS-derived toxic air contaminants for the California population for the second half of the 1990s.
(5) Critique the quality of the resulting distributions, taking into account the methodological limitations and the quality of the input data.

Methods

The research was conducted in three phases. In Phase I, the exposure of nonsmoking Californians (age $\geq$ 7 y) was estimated for the mid to late 1980s. Measurements of personal exposure to volatile organic compounds were compared for individuals reporting ETS exposure to those who reported no exposure. The contribution of ETS to personal exposure was determined in this manner separately for four compounds: benzene, styrene, o-xylene, and m,p-xylene. For these species the fractional contribution of ETS to total exposure was computed. Emission factors were then employed to estimate exposure from ETS to thirteen other compounds. In Phase II, exposures were modeled, again for the mid to late 1980s, for adult and adolescent nonsmokers and for children. Here, exposure was computed by combining activity pattern data for the Californian population with concentrations of ETS constituents determined from steady-state material balance models and from published measurements of ETS tracers such as nicotine or particulate matter. Daily exposures were computed by this approach for individuals with different activity patterns and who visited different groups of microenvironments such as residences, workplaces, and restaurants. In Phase III, we estimated exposure for conditions in the late 1990s, using the same methods as in Phase II, but accounting for changes in smoking behavior and for the effects of new statewide restrictions on smoking in public buildings (AB13). In each phase, key uncertainties in the results were quantified.

Results

Environmental tobacco smoke contributes significantly to the exposure of nonsmokers to toxic air contaminants. Among nonsmoking Californians exposed to ETS, the estimated average contribution of ETS was 3-10% of total benzene exposure in the mid to late 1980s (see Table 5.10). For styrene and xylenes, ETS is also a significant contributor to nonsmokers' exposure,

responsible for 6-18% of average styrene exposure, 1-8% of average o-xylene exposure, and 1-5% of average m,p-xylene exposure (again, among nonsmokers exposed to ETS; see Table 5.10). Averaged over all nonsmoking Californians, the corresponding contributions of ETS to total inhalation exposures were 2-5% for benzene, 3-10% for styrene, 0.4-5% for o-xylene, and 1-3% for m,p-xylene (see Table 5.11). For other toxic air contaminants considered in this study — acetaldehyde, acetonitrile, acrylonitrile, 1,3-butadiene, 2-butanone, o-cresol, m,p-cresol, ethyl acrylate, ethylbenzene, formaldehyde, n-nitrosodimethylamine, phenol, and toluene — the extent of exposure from ETS was quantified (see Tables 5.3-5.5), but total human exposures are unknown. As of the late 1980's, the most important exposure sites for adults were in one's own home and in occupational settings, together contributing about 60% to the total ETS exposure (see Figure 5.3). For adolescents and children, residential exposures dominated, contributing roughly half of the total for adolescents and 70-75% of the total for children (see Figures 5.4 and 5.5). Among those nonsmokers exposed to ETS, average exposure for adolescents was in the range 65-95% of the average for adults; the corresponding range for children was 80-130% (see data in Table 5.10). Again, among those exposed, mean daily exposure to benzene from ETS is estimated to have been in the range 9-31 $\mu g\ h\ m^{-3}$ for adults, 9-20 $\mu g\ h\ m^{-3}$ for adolescents, and 12-24 $\mu g\ h\ m^{-3}$ for children, with the ranges being indicative of the uncertainty in the estimates (see Tables 5.3-5.5).

As of the late 1990's, following implementation of AB13, smoking is no longer permitted in most workplaces in California. Assuming the law is observed, the only significant remaining indoor sites of ETS exposure are in private residences and in vehicles. The prevalence of smoking among adult Californians has also declined during the 1990's (see Figure 6.1), reducing the frequency and intensity of ETS exposure. These factors are estimated to have substantially reduced the exposure to toxic air contaminants from ETS, primarily by reducing the number of people exposed on a given day. For nonsmoking adults in California, the percentage exposed to ETS on a given day (in microenvironments modeled in this study) is estimated to have declined from 52% in the late 1980's to 16-19% in the late 1990's. Corresponding changes for adolescents are from 63% to 33-35% and for children from 33% to 21-23% (see Figure 3.4). The reduction in the mean exposure for all nonsmokers is predicted to be in the range 60-75% for adults, 40-50% for adolescents, and 20-40% for children (see Table 6.8 and Figure 6.10). The proportionally smaller reduction for adolescents and children is predicted because, relative to adults, a larger portion of their exposure occurs in unregulated indoor environments such as private residences. Although the number exposed has declined, average exposure levels for those who remain exposed have not changed markedly, especially for adolescents and children (compare Tables 5.3-5.5 to Tables 6.2-6.4). Among those exposed, mean daily exposure to benzene from ETS in the late 1990s is estimated to be in the range 12-21 $\mu g\ h\ m^{-3}$ for adults, 9-22 $\mu g\ h\ m^{-3}$ for adolescents, and 12-28 $\mu g\ h\ m^{-3}$ for children (see Tables 6.2-6.4). A large portion of the late 1990s exposure is predicted to occur in personal residences, contributing 58-69% of the total exposure for adults, 58-66% for adolescents, and 72-83% for children (see Figures 6.6-6.8). The average daily exposures to 17 toxic air contaminants were estimated for the Californian nonsmoking population, separately for adults, adolescents, and children, for late 1990's, both for those exposed to ETS on a given day (Tables 6.2-6.4) and for all nonsmokers (Table 6.8). Information on the variability of exposures among individuals is also provided (Figures 6.4 and 6.5).

Conclusion

This study quantifies for the first time the role of environmental tobacco smoke in contributing to the exposure of nonsmoking Californian's to selected toxic air contaminants. Overall, ETS is seen to be a significant, but not dominant source of exposure to benzene, styrene, and xylenes. ETS-caused exposure to other species was quantified, but total inhalation exposures for these species are unknown. Changes in smoking regulations and in smoking behavior during the past decade have reduced by a significant degree the exposure to toxic air contaminants from ETS. However, exposure to ETS in private residences remains a significant means by which Californians encounter toxic air contaminants. The information generated by this research can be used in conjunction with other recently available data to estimate the relative contributions of indoor and outdoor sources of TACs to Californians' current exposures. This information will help ARB identify effective mitigation strategies for reducing the residual public health risks from these TACs.

The specific compounds considered in this study are all of the formally designated toxic air contaminants for which reliable data exist on ETS emission factors. However, tobacco smoke is known to be a source of other hazardous materials such as particulate matter, metals, and polycyclic aromatic hydrocarbons. Exposure to the toxic compounds included in this study is expected to cause only a portion of the total health hazard from ETS.

1. Introduction

Exposure to toxic air contaminants (TACs) is suspected to pose significant health risks. The adverse outcomes are diverse, ranging from odor and eye irritation to cancer, and include respiratory toxicity, central nervous system effects, reproductive toxicity, and systemic effects such as liver and kidney toxicity. Relative to the large number of pollutants of concern, health effects data are limited. For many TACs there may be no safe exposure level. Sudden accidental releases can create immediate and serious health problems, while repeated low-level exposures can cause health effects that become evident long after the exposure began.

Exposure to TACs has gained widespread public attention over the last two decades (Berry, 1990; Möller et al., 1994). As a result, state and federal regulatory agencies have established policies to reduce human exposures (Calabrese and Kenyon, 1989; Robinson and Pease, 1991). In the United States, Title III of the Federal Clean Air Act Amendments of 1990 seeks to reduce the human health risk from exposure to 189 compounds in ambient air (designated as Federal hazardous air pollutants), chosen because they are known to have, or may have, adverse effects on human health or the environment (Buonicore et al., 1992). In California, Assembly Bill (AB) 1807 (enacted in 1983) established a program to identify and control toxic air pollutants. Under this law, the California Air Resources Board (ARB) is required to identify and assess exposures to TACs (Krieger et al., 1993). In 1992, AB 1807 was amended, adopting the full list of Federal hazardous air pollutants as TACs and requiring the ARB to study exposure to these pollutants (AB 2728). Eighteen compounds that are on the federal list had already been identified as TACs under the original AB 1807 (Krieger et al., 1993). As of 1993, the ARB list included 194 compounds.

Nationwide, significant efforts have focused on evaluating outdoor air toxic emissions from sources such as power plants, dry cleaners, and motor vehicles (Sweet and Vermette, 1992; Miller, 1993). Although both point and area sources appear to contribute significantly to the amount of toxic compounds in ambient air, such emissions may not be the predominant contributors to human exposures. Substantial evidence demonstrates that concentrations of many air toxicants are higher indoors than outdoors as a result of indoor sources; yet, limited quantitative information is available concerning the sources of these compounds in indoor air. The evidence that exists suggests that building materials, consumer products, and combustion processes may all contribute significantly to exposures to many compounds. For example, Wallace (1991a) estimated upper-bound lifetime cancer risks for twelve volatile organic compounds (VOCs) that are considered to be air toxicants, based on measurements taken during the 1980 and 1987 U.S. Environmental Protection Agency Total Exposure Assessment Methodology (TEAM) studies. He estimated that indoor sources accounted for 80–100% of the total airborne risk.

A potentially important contributor to the presence of TACs in indoor air is environmental tobacco smoke (ETS). ETS consists of diluted and aged sidestream smoke emitted by the burning tobacco between puffs, plus smaller contributions from exhaled mainstream smoke. Studies have shown that smoking indoors significantly increases indoor concentrations and personal exposure levels of certain air toxicants, including benzene and styrene (Wallace et al., 1988; Heavner et al., 1995). Wallace (1989a, 1989b) estimated that ETS accounts for 5% of the total nationwide exposure to benzene. (Active smoking is the dominant cause of population exposure.) Besides benzene and styrene, ETS is a potentially important source of many other TACs, including acetaldehyde, 1,3-butadiene, cresols, N-nitrosamines, phenols, and xylenes (National Research Council, 1986; Hodgson and Wooley, 1991).

There is a substantial body of scientific evidence demonstrating that ETS exposure increases risks of many diseases (U.S. Environmental Protection Agency, 1992a) and that ETS contains many individual compounds that have been shown to cause cancer in test animals (National Research Council, 1986; U.S. Environmental Protection Agency, 1992a). It is not known, however, which of the many ETS constituents cause adverse health effect outcomes. Nevertheless, there are indications that TACs may be important contributors to the health risks from ETS exposure. It is known that specific gas-phase ETS constituents cause odor and eye irritation to exposed individuals (National Research Council, 1986). Epidemiological studies have shown that exposure to environmental levels of benzene caused by smoking may influence cancer risk: for example, two studies have shown that children of smokers contract leukemia at two or more times the rate of children of nonsmokers (Neutel and Buck, 1971; Sandler et al., 1985).

Despite some decline in recent years, 15-20% of Californian adults still smoke (California Department of Health Services, 1998). Consequently, a large fraction of Californians are exposed to ETS. A survey of the activity patterns of Californians conducted in the late 1980s determined that 62% of the total population over 12 years of age report some exposure to ETS during any given day (Jenkins et al., 1992; Wiley et al., 1991a). A similar study reported that 38% of Californian children are exposed to ETS on a daily basis (Wiley et al., 1991b).

Although ETS may be an important contributor, its quantitative impact on Californians' exposures to TACs is unknown. We have undertaken a three-phase effort to fill this information gap. In Phase I, an assessment for exposure during the mid to late 1980s was conducted primarily based on measurements of personal exposure. In Phase II, an assessment for the same time period was conducted, based on measurements and model predictions of microenvironmental concentrations, combined with activity pattern information, using Monte-Carlo simulation techniques. Phase III used the same approach as Phase II to predict the impact of changing smoking habits and regulations on exposures to ETS-derived air toxicants for the late 1990s. This final report presents the methods and results from these three phases of the work.

The specific objectives of this research are these:

(1) Determine the frequency distribution of exposure to selected toxic air contaminants from ETS for the California population. Individual exposures are to be determined on a 24-h average basis. The distribution of such exposures is to be determined across the population. The exposure distribution is to be determined for each of the toxic air contaminants for which reliable data are available on ETS emission factors. The species included in the study are listed in Table 1.1, along with a brief description of their adverse effects on health. The baseline exposure analysis is to be conducted for the approximate period 1984-1990.

(2) Determine the proportion of Californians' total exposure to air toxics that can be attributed to ETS for selected compounds over the period 1984-1990.

(3) Explore the relative amounts of exposure in the workplace and other public spaces versus exposure in residential settings.

(4) Predict the impact of changes in smoking policy and smoking behavior on exposures to ETS-derived toxic air contaminants for the California population for the second half of the 1990s.

(5) Critique the quality of the resulting distributions, taking into account the methodological limitations and the quality of the input data. Indicate the key sources of uncertainty and provide suggestions on reducing these uncertainties through future research and data collection activities.

The study focuses on the seventeen specific compounds from the list of toxic air contaminants for which reliable data are deemed to exist on emission factors from environmental tobacco smoke. It is known that tobacco smoke is a source of other hazardous materials such as particulate matter, metals, and polycyclic aromatic hydrocarbons. Therefore, exposures to the compounds included in this study are expected to account for only a portion of the total health hazard from ETS.

Table 1.1. Species of toxic air contaminants (TAC) in environmental tobacco smoke (ETS) included in this study and their health effects. [1,2]

compound [3]	health effects
acetaldehyde [4]	A possible human carcinogen[6] as designated by USEPA; nasal and laryngeal tumors observed in experimental animals; acute effects include eye, skin and upper respiratory tract irritation; liquid form causes skin burns and rash.
acetonitrile	No cancer data; vapor may cause irritation of skin, eyes, nose and throat, vomiting, convulsions and death. Overexposure may produce cyanide poisoning following metabolism to cyanide.
acrylonitrile [4]	A probable human carcinogen[7] as designated by USEPA; limited evidence of increased lung cancers in humans; central nervous system (CNS) effects include nausea, headache, dizziness, fatigue and weakness; a respiratory irritant.
benzene [4]	A human carcinogen as designated by USEPA; sufficient evidence of increased leukemia in humans; a CNS depressant; chronic effects include anemia, blood cell and bone marrow damage.
1,3-butadiene [4]	A possible human carcinogen[6] as designated by USEPA; limited evidence of increased leukemia and lymphomas in humans; increased cancer in multiple sites in animals; acute effects include CNS damage; chronic exposure adversely affects lung, heart and blood systems; may have reproductive toxicity.
2-butanone (MEK)	Moderately toxic by ingestion, skin contact, and intraperitoneal routes; acute effects include nose, throat, eye and skin irritation; a CNS depressant; enhances the neurotoxicity of n-hexane.
o-cresol and m,p-cresol	May act as a promotor for forestomach tumors in animals; a CNS depressant; corrosive to the skin and eyes; causes respiratory tract irritation; oral exposure in animals affects the blood, liver, kidney and central nervous system.
ethyl acrylate [4]	May increase incidence of colorectal cancer; irritating to eyes and respiratory tract; adverse effects on the CNS and gastrointestinal system in humans; liquid causes skin sensitization.
ethylbenzene	Limited and inconclusive cancer data; irritating to eyes and respiratory tract; a CNS depressant; chronic exposure in animals affects the blood, liver, and kidney.
formaldehyde [4]	A probable human carcinogen[7] as designated by USEPA; limited evidence of increased lung and nasopharyngeal cancer in humans; highly irritating to eyes and respiratory tract; acute effects include nausea, headaches and difficult breathing; induces or exacerbates asthma.
N-nitroso-dimethylamine [4]	A possible human carcinogen[6] as designated by USEPA; increased incidence of liver, kidney, and lung tumors in animals; causes liver damage including jaundice; nausea, vomiting and malaise.
phenol	No cancer data; highly irritating to eyes and respiratory tract; acute inhalation exposure may cause nausea, vomiting, irregular heart beat, circulatory collapse, convulsions and coma.
styrene	Metabolite from styrene is a direct-acting mutagen which causes cancer in test animals; irritating to eyes, nose, throat, and lungs; a CNS depressant.
toluene [5]	A CNS depressant; may cause irregular heart beat; liver and kidney injury at high exposure; reproductive toxicity; mildly irritating to eye and respiratory tract.
o-xylene and ***m,p-xylene***	A CNS depressant; may cause eye, nose, throat and respiratory tract irritation; may cause digestive system effects; may injure the kidneys.

[1] Reference: Air Resources Board (1997)

[2] Chemicals listed have been identified as Toxic Air Contaminants by the State of California Air Resources Board.

[3] Bold-italics indicate compounds for which monitoring data are available in the Californian Exposures Database.

[4] Chemicals determined to cause cancer by the State of California under Proposition 65.

[5] Chemicals determined to cause reproductive toxicity by the State of California under Proposition 65.

[6] USEPA classifies a chemical in Group B2 as a possible human carcinogen when the chemical has been shown to have limited carcinogenicity evidence in humans in the absence of sufficient evidence in experimental animals. The group may also include chemicals that have sufficient animal evidence of carcinogenicity but have inadequate carcinogenicity evidence in humans or that have no human data.

[7] USEPA classifies a chemical in Group B1 as a probable human carcinogen when the chemical has been shown to have limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals.

2. Overview of Approach

Assessing the exposure of a nonsmoker to environmental tobacco smoke constituents can be achieved by either personal air monitoring or by combining models or measurements of concentrations in microenvironments with the time that an individual comes into contact with ETS in that microenvironment (Leaderer, 1990). In Phase I of this project we conducted an assessment based on personal monitoring; that is, we estimated the distribution of exposures to ETS-derived air toxicants for the Californian population by constructing an exposure distribution based on direct measurements of personal exposure concentrations from the studies conducted in California in the mid to late 1980's. These were the specific goals of Phase I:

- to characterize the distribution of inhalation exposures to 17 TACs for persons who actively smoke (excluding the exposure due to inhaled MS), nonsmokers exposed to ETS, and those unexposed to ETS;
- to derive for nonsmokers the distribution of exposures to TACs that can be attributed to ETS only;
- to estimate the proportion of exposures attributable to ETS; and
- to compare ETS-caused exposures to the concentrations typically present in ambient and indoor air.

To achieve these goals, we conducted descriptive and analytical assessments of previously reported data from (a) human exposure studies conducted in California and (b) laboratory measurements of TAC emission factors for environmental tobacco smoke. Section 3 of this report summarizes and provides usage details for the exposure monitoring data. Section 4 describes the methods applied and results obtained in Phase I of the assessment.

In Phase II, exposure distributions were estimated by combining person activity patterns and concentration predictions of ETS-derived air toxicants in a Monte-Carlo-based model simulation. These were the specific goals of Phase II:

- to derive for nonsmokers the distribution of exposures to 17 toxic air contaminants that can be attributed to ETS only; and
- to derive distributions of exposures attributable to ETS in seven microenvironments.

To achieve these goals, we used (a) activity pattern data for the Californian population, (b) published measurements of ETS tracers — nicotine and particulate matter — in different indoor microenvironments, (c) laboratory measurements of TAC emission factors for environmental tobacco smoke, and (d) material balance models plus pertinent data for key input variables. Most of the key input data were obtained in the mid to late 1980's, so the Phase II effort focused on assessing exposure for this time period. Three population groups were separately assessed: adults

(age 18 and above), adolescents (age 12-17), and children (age 0-11). Section 5 of the report describes the methods and results from Phase II of the investigation.

In Phase III, we used the model and methods developed in Phase II to predict the impact of changing smoking habits and regulations on exposures to ETS-derived air toxics for the late 1990s. This effort is described in Section 6 of the report.

2.1 Phase I: Assessment for late-1980's Using Personal Monitoring Data

In the Phase I assessment, we used data from two types of studies: (1) personal monitoring of inhalation personal exposure to air toxicants; and (2) measurements of ETS emission factors from cigarettes. The personal monitoring data were used to empirically derive exposure distributions for the active (those who smoke cigarettes), passive (nonsmokers exposed to ETS), and unexposed (those not exposed to ETS) populations of California, age$\geq$7 y. The ETS emission factors were used to infer exposure distributions for those compounds which were not directly measured during personal monitoring.

The method employed in Phase I is based on several assumptions: (1) exposures are additive, so that the total exposure can be determined by summing the contributions from all sources; (2) the "unexposed" and "passively exposed" populations have the same distributions of exposure to TACs from all sources other than ETS; (3) the exposure to TACs from ETS for those who report themselves to be "unexposed" is negligible; (4) air toxicant exposures due to ETS only are lognormally distributed; and (5) exposures to different compounds from ETS scale in proportion to their emission factors.

For four monitored compounds (benzene, styrene, o-xylene, and m,p-xylene), we used the empirical exposure distributions for the passive and the unexposed populations to derive an ETS-only distribution of TAC exposure. A three-step procedure was followed: (1) we hypothesized the lognormal parameters for the ETS-only distribution; (2) we generated a *simulated* passive distribution, termed the *ETS-only + unexposed* distribution, by randomly sampling from the ETS-only distribution and the empirical unexposed distribution and summing; (3) and we compared the *ETS-only + unexposed* distribution to the empirical passive distribution using a well-established statistical method for comparing two distributions. The best-estimate ETS-only distribution was obtained by successively and systematically repeating steps (1)–(3) to obtain the best agreement between the simulated and empirical passive distributions.

An ETS-exposure scale factor was defined as the ratio of (a) the mean exposure concentration to a species from ETS ($\mu g\ m^{-3}$) to (b) the emission factor of the species ($\mu g\ cig^{-1}$). This scale factor was separately determined for each of the four monitored compounds, then averaged to obtain a best-estimate overall scale factor. Emission factors for 13 toxic air

contaminants were multiplied by this scale factor to estimate the contribution of ETS to exposure for these other unmeasured species.

A key contributor to uncertainty in these estimates results from the small number of subjects for whom exposures were measured. A numerical experiment was devised and conducted to estimate the uncertainty caused by small sample size.

2.2 Phase II: Assessment for late-1980's Based on Microenvironmental Exposure Modeling

In Phase II of the assessment, we applied a modeling-based approach that features a Monte-Carlo procedure for constructing a probability distribution of indoor concentrations and exposures. Modeling-based methods have gained wide use in predicting population exposures to both gaseous and particulate air pollutants (Lurmann et al., 1989; Ott, 1990; Kleipeis et al., 1992). One of the many advantages to this approach is the ability to predict effects of different control strategies on exposures. Without such capability, one would have to conduct large field monitoring studies before and after implementation of the control strategies under consideration. Another advantage of this approach is that it is relatively inexpensive and can be less complicated logistically to implement than a field monitoring study.

To estimate exposures to ETS-derived air toxicants using the modeling approach, we estimated ETS concentrations in many different groups of microenvironments: residential, occupational, retail/other indoor locations, restaurants, bar/nightclub (adults only), transportation, residential guest, and schools (children and adolescents). Once ETS concentrations were evaluated, we combined them with information on the time a person is exposed to ETS in these microenvironments to estimate exposure for an individual. The distribution of exposures for a population was obtained by repeating this procedure for subjects studied in surveys of the activity patterns of California residents.

We evaluated ETS concentrations by two approaches: measurements of ETS tracers and completely-mixed room (CMR) models. In the case of measurements, we used measured concentrations of nicotine and particulate matter (PM) that were monitored in the microenvironment of interest. To use CMR models, we applied mathematical equations based on the well-established principle of material conservation. These equations specifically account for the dominant processes that control ETS levels indoors: direct emission from smoking and removal by ventilation. The use of material balance equations to predict indoor air pollutant concentrations was first applied by Turk (1963) for odors in test chambers. Since the 1970s, the same approach has been widely used to simulate gaseous and particulate pollutants in indoor air (Shair and Heitner, 1974; Esmen, 1978;

Alzona et al., 1979; Ishizu, 1980; Özkaynak et al., 1982; Nazaroff and Cass, 1986; Ryan et al., 1988; Nazaroff and Cass, 1989; Sparks et al., 1991; Hayes, 1991; Kleipeis et al., 1992).

We derived estimates of exposure time using data from two studies. For adults and adolescents, we used the Activity Patterns of California Residents (APCR) study, funded by ARB (Wiley et al., 1991a). In this study, the time spent in different microenvironments was characterized for a 24-hour period through the use of questionnaires and activity diaries for 1579 adults and 183 adolescents. Time-activity information gathered with diaries detailed the amounts of time individuals spent at specific activities in various locations throughout the course of a day, as well as recording whether or not they reported being exposed to ETS during that activity. For children, we used the study of Children's Activity Patterns (CAP), also funded by the ARB (Wiley et al., 1991b). This study was similar in design to the APCR and collected data on the activities of 1200 children, ages 0-11.

We focused our assessment on nonsmoking subjects in the APCR and the CAP who reported being exposed ETS: 625 adults, 98 adolescents, and 483 children (see Figure 2.1). Excluding a small fraction who were only exposed to ETS outdoors, the exposure to benzene from ETS for each of these subjects was modeled by computing a microenvironmental concentration of benzene in each setting where exposure occurred, multiplying this concentration by the duration of exposure, and summing over all exposures for the 24-hour activity period. Exposure to other toxicants was then determined by multiplying the ETS-only benzene exposure by a ratio of measured ETS emission factors.

Input parameters needed to determine microenvironmental concentrations for our modeling-based assessment are both variable and uncertain. Variability refers to true differences in the value of a parameter when determined across a population. Uncertainty captures factors that contribute to error including, but not limited to, experimental error (reported measurements were inaccurately determined) and sampling error (the population measured doesn't accurately match the population of interest).

We used a probabilistic approach to determine concentrations that captures the effects of both variability and uncertainty. To simulate the effects of *variability*, we developed distributions of input parameters from published data that are intended to represent those variables for the population of California. The data were combined to predict exposures using Monte-Carlo methods. The basic procedure was to repeatedly and randomly draw a number from the parametric distribution for each input variable and, from a given set of such numbers, calculate the resulting exposure. With each iteration, a different estimate of exposure is determined; a properly constructed set of such iterations produces the distribution of exposures for the study population. The iteration process is repeated until the variance in the exposure estimates no longer changes.

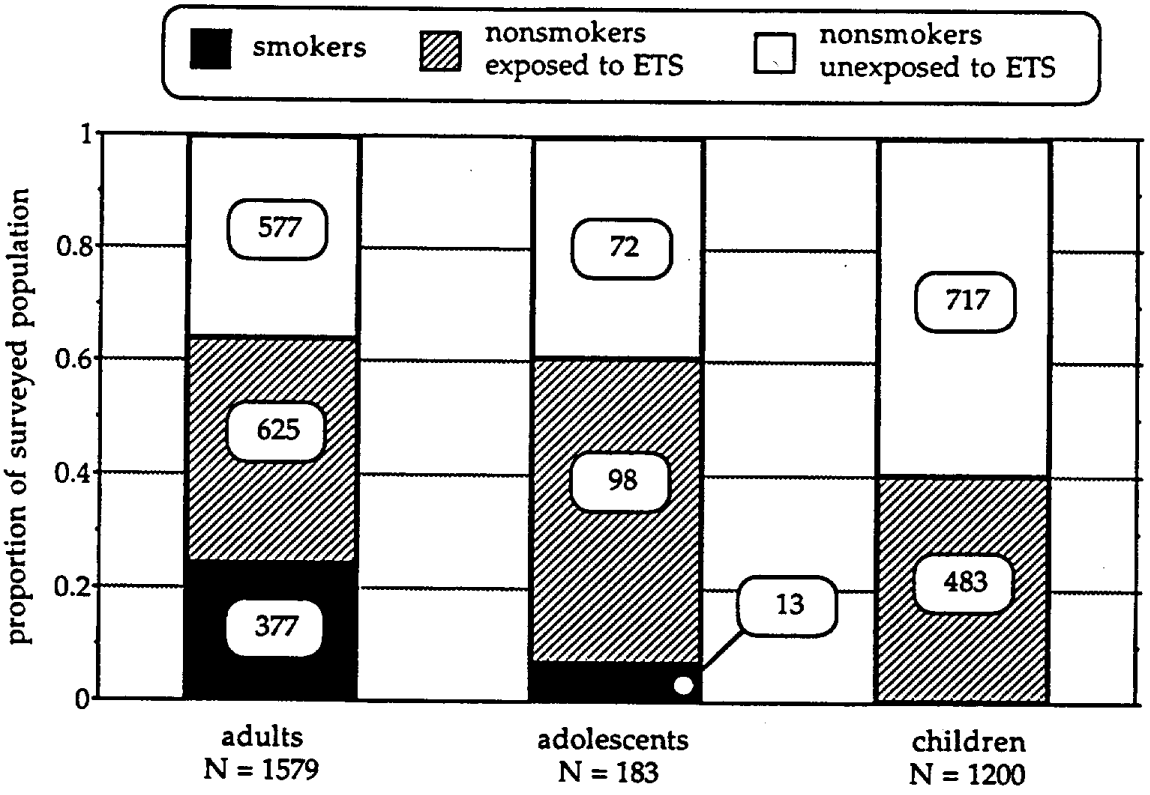

Figure 2.1 Distribution of interview subjects (unweighted) in the Activity Patterns of California Residents (APCR) survey and the Study of Children's Activity Patterns (CAP) survey, sorted according to age group and smoking/ETS exposure status.

To generate information on the *uncertainty* in the exposure predictions, we conducted independent simulations of environmental tobacco smoke exposure according to four *scenarios*, where a scenario represents a set of probability distributions for input parameters needed to compute the distribution of exposures in each microenvironment. The scenarios were initially designed according to a 2×3 matrix: wherever possible, we computed microenvironmental concentrations from both the CMR model (C-) and the tracer method (T-) and generated low, mid-range, and high exposure parameter distributions (-L, -M, and -H, respectively). The six scenarios were ultimately reduced to four. The CL and CH cases were eliminated because a compelling set of mid-range parameter distributions for key input parameters was available. The CM and TM scenarios produce results that agree fairly well and represent the central estimate conditions. The differences between these scenarios and the TL and TH scenarios indicate the scale of uncertainty.

The primary modeling calculations focused on predicting benzene exposure from environmental tobacco smoke. In using the CMR approach to estimating ETS concentrations, we applied the emission factor from Daisey et al. (1994, 1998) of 406 $\mu g\ cig^{-1}$. In using the tracer approach, we used the ratio of emission factors for benzene to the ETS tracer (particulate matter or nicotine). Having estimated benzene exposure from ETS, exposure to other air toxics was obtained by scaling the results by the ratio of emission factors.

2.3 Phase III: Assessment for Late 1990's Based on Microenvironmental Exposure Modeling

Exposure to ETS among Californian's is likely to have diminished significantly between the mid to late 1980's and the late 1990's. The dominant contributors to this change are changes in the incidence of adult smoking — from 23% in 1988 to 17%-19% in 1995-97 (California Department of Health Services, 1998)— and the implementation of AB 13 which, with few exceptions, prohibits smoking in enclosed workplaces. To assess ETS exposures under these altered conditions, the methods employed in Phase II were repeated with input data appropriate to the late 1990's.

In this phase of the research, we assumed that the conditions of AB 13 would be completely met, so that ETS exposures would only occur in private residences (one's own home, or as a guest) and in motor vehicles.[1] Calculations were done for these microenvironments, and other settings were assumed to contribute nothing to exposure. Changes in smoking prevalence were extrapolated from trends between 1988 and 1998. Survey data show that the mean number

[1] AB13's provision for smoke-free bars (and bar-restaurant combinations) became effective only in January 1998. This provision remains politically contentious.

of cigarettes consumed per smoker per day may have changed slightly over this period. These changes were used to adjust the probability of encountering ETS indoors and the concentrations so encountered, such that the exposure could be modeled as in Phase II. Again, four scenarios were simulated to bracket the expected uncertainty in the outcome.

3. Data Sources

To carry out the objectives of this research, data were required for a variety of parameters. In this section of the report, the general criteria applied in selecting the most appropriate data are outlined, and some of the major sources of data employed in the study are described.

To model exposures in Phases II and III of the research, it was necessary to determine ETS concentrations in different microenvironments. As described in §5, two methods were employed. The tracer method used measurements of ETS markers — nicotine or particulate matter — as a means of estimating concentration distributions. The completely mixed room (CMR) model method used a material balance model along with information on smoking frequency, emission factors, and ventilation rates to compute ETS concentrations. The specific data sets employed and how they were interpreted for this study are discussed in §5.

3.1 Criteria for Selecting Data

Data attributes that are desired are listed below, along with a brief discussion of their significance.

- *Compatibility of measured parameter with model formulation.* Excellent information on some parameter related to ETS exposure is of little value if the information does not permit full calculation. We selected modeling approaches based on the goal of generating the best possible estimate of exposure, as constrained by available information. Once a modeling approach was selected, only data in a form that was consistent with the needs of the approach could be used.
- *Complete documentation of method and results.* The measurement method should be clearly and completely documented. Complete information on the results should be available, including not only the central tendency, but also information on variability. Ideally, a full description of the probability distribution function, or alternatively individual measurement results should be available.
- *Archival publication of study.* Studies published in the archival literature are preferred to government reports or conference papers. Information in archival literature is more widely available. It also has a higher level of credibility within the scientific community because of the perceived benefit of peer-review. Therefore, to the extent that our research could rely on archival literature for input data, the results are more likely to be accepted.
- *Appropriateness to California population.* Because the goal is to predict exposures of the California population, studies conducted in California are preferred. Where California-

specific information is unavailable, studies conducted elsewhere in the US are preferred to those from Europe or elsewhere.

- *Statistically representative sampling*. The goal of this research is to generate descriptions of the probability distribution functions of exposure. At the very least, we want information on variability in addition to the central tendency. Bias in either parameter may enter if the environments or populations studied are not statistically representative.

Most data sources do not meet all of these criteria. In some cases, multiple candidate information sources were available, none of which was ideal. Decisions about which specific data sets to include in the analysis and how to interpret and apply the results reflect the scientific judgment of the investigators. Other researchers facing the same problem may have made different choices for any of several reasons: (a) different selection criteria; (b) different relative weights given to the various criteria; or (c) different opinions about how well any given study meets the selection criteria. Different choices about input information would, of course, lead to different results. However, the methods we have employed are designed to bracket the range of likely outcomes. Given the fairly large variability and uncertainty observed, we consider it improbable that different choices made in the selection of input data would alter significantly the overall results.

3.2 Environmental Tobacco Smoke (ETS) Emission Factors

ETS is an aerosol consisting of vapor and particulate phases comprising roughly 4000 compounds. The major source of ETS is sidestream smoke (SS), which is the material released directly into the air from the burning end of a cigarette between puffs. SS may differ substantially from ETS. ETS is diluted and "aged" SS, plus exhaled mainstream smoke (MS), in which aging involves volatilization, chemical oxidation, and other physicochemical changes (National Research Council, 1986). Undiluted SS contains higher proportions of certain toxic chemicals than undiluted MS.

The composition of ETS can be determined from experimentally measured emission factors, defined in this context as the apparent quantity of a contaminant emitted into indoor air per quantity of tobacco or per cigarette burned. Laboratory procedures have been developed to measure emission factors for SS, MS and for ETS. For example, SS emission factors can be measured using small-volume devices designed to allow sampling of freshly generated and minimally diluted SS (Guerin et al., 1992). To properly account for the effects of dilution and aging, ETS emission factors should be measured in a full-sized chamber.

Until recently, quantitative data on ETS emission factors were sparse. Löfroth et al. (1989) and Jermini et al. (1976) measured ETS emissions factors for a few air toxicants. During the 1990s, ETS emission factors for many organic compounds were reported by Daisey et al. (1994, 1998) and Mahanama and Daisey (1996). Experiments were conducted in a full-sized (20 m^3), stainless steel chamber that was designed specifically for measuring emissions of VOCs from

indoor sources. In these experiments, sidestream smoke from several machine-smoked cigarettes was introduced and mixed into the unventilated chamber. Sampling occurred at intervals as the smoke aged. For species in which significant concentration decay was observed, corrections were applied to determine emission factors. The experimental procedure excluded any ETS contributions associated with exhaled mainstream smoke. Daisey and coworkers tested six American cigarette brands representing a combined market share of 63.5% of the cigarettes sold in California in 1990.

More recently, Martin et al. (1997) measured ETS emission factors for the 50 top-selling U.S. cigarette brand styles representing 65.3% of the total U.S. market. In this study, ETS was generated by volunteer smokers in a full-sized (18 m^3), stainless steel chamber. A 12-min background period was followed by an 11-min smoking period and a 60-min concentration monitoring period, during which integrated samples were collected. This study differs from those of Daisey and coworkers on several points: (a) no correction was made by Martin et al. to account for species uptake on chamber surfaces; (b) the measurements of Martin et al. include exhaled mainstream smoke; (c) the cigarettes tested by Martin et al. were smoked by humans, rather than by machines; and (d) a broader range of cigarette products was tested by Martin et al.

Table 3.1 summarizes the emission factors measured by Daisey et al. and Martin et al., along with other published results. For cases in which more than one researcher has made measurements, the emission factors are in general agreement for eight of ten compounds (individual determinations within 20–30% of the mean). The disagreement is larger, with results ranging over a factor of 2–3, for 1,3-butadiene and 2-butanone. In general, the good agreement, especially between the extensive studies of Daisey and coworkers and Martin et al. adds confidence to the validity of using ETS emission factors to predict exposures to air toxicants.

The ETS emission factors of Daisey et al. were used in all phases of this research. Parallel use is made of the emissions data of Martin et al. in Phase I. In Phases II and III the data of Martin et al. are used to estimate exposure to acetonitrile, a compound not measured in the research of Daisey et al.

3.3 Californian Exposures Database (CED)

Data from the California Exposures Database were used in Phase I of the research, as described in §4. By comparing exposure measurements for nonsmoking adults who reported ETS exposure with measurements for those who reported no exposure, the contribution of ETS to exposure was inferred.

Table 3.1. Environmental tobacco smoke (ETS) emission factors for toxic air contaminants.

	emission factors (μg cig^{-1}) average ± standard deviation			
compound[1]	Daisey et al. (1998)[2]	Martin et al. (1997)[3]	Löfroth et al. (1989)[4]	Jermini et al. (1976)[5]
acetaldehyde	2150 ± 477	2496 ± 547	2400	
acetonitrile		1145 ± 457		
acrylonitrile	99 ± 18			
benzene	406 ± 71	280 ± 49	500	415
1,3-butadiene[6]	152 ± 27	373 ± 126	400	
2-butanone (MEK)	291 ± 56			708
o-cresol	35 ± 5			
m,p-cresol	83 ± 26			
ethyl acrylate[7]	<3			
ethylbenzene	130 ± 10	80 ± 14		
formaldehyde	1310 ± 348	1333 ± 337	2000	
N-nitroso-dimethylamine[8]	0.57 ± 0.12			
phenol	281 ± 61			
styrene	147 ± 24	94 ± 18		98
toluene	656 ± 107	498 ± 108		829
o-xylene	67 ± 16	59 ± 16		48
m,p-xylene	299 ± 52	239 ± 48		234

[1] Bold-italics indicate compounds for which monitoring data are available in the Californian Exposures Database.
[2] Average for 6 American brands; 20-m^3 chamber; sidestream smoke only. Acrolein emissions were also measured by Daisey et al.; however, they consider their results unreliable due to measurement problems and recommend against their use in exposure assessment.
[3] 18-m^3 chamber; sidestream plus exhaled mainstream. Standard deviation was estimated from the reported standard error multiplied by $100^{1/2}$ where the factor 100 equals the number of test runs per cigarette brand (2) × the number of brands (50). For some brand styles, 3 test runs were conducted.
[4] 13.6-m^3 chamber; continuous smoking rate of 1 cig/15 min or 1 cig/30 min; sidestream smoke only.
[5] Thirty American cigarettes smoked simultaneously in a 30-m^3 chamber; sidestream smoke only.
[6] Daisey et al. noted that the emission factor for 1,3-butadiene had "greater uncertaint[y] than other emission factors due to evidence of chemical reactions which vary in different environments."
[7] Emission factor measured below the lower limit of detection.
[8] Emission factor reported in Mahanama and Daisey (1996).

3.3.1 Overview

Six monitoring studies conducted in California have measured personal exposure concentrations for selected air toxicants: five of the TEAM studies, sponsored by the U.S. Environmental Protection Agency and the California ARB, and a study sponsored by the ARB and conducted in Woodland, CA. The TEAM studies were designed to develop and demonstrate methods of measuring human exposure to toxic substances in air and drinking water (Wallace et al., 1988). The Woodland study was designed to generate indoor and personal exposure data for the Air Resources Board's TAC identification process (Sheldon et al., 1992). All field operations for these six studies were conducted by the Research Triangle Institute (RTI) as prime contractor. The data were also organized by RTI into the Californian Exposures Database (CED) by recoding, restructuring, and merging the data (Clayton and Perritt, 1993). The following list introduces the six studies, giving their location, monitoring period, and primary literature references:

- *Study 1.* Los Angeles (LA) County, winter (Jan-Feb) 1984
  (Hartwell et al., 1987; Wallace et al., 1988)
- *Study 2.* LA County, summer (May-Jun) 1984
  (Hartwell et al., 1987; Wallace et al., 1988)
- *Study 3.* Pittsburg/Antioch, summer (Jun) 1984
  (Hartwell et al., 1987; Wallace et al., 1988)
- *Study 4.* LA County, winter (Jan-Feb) 1987
  (Hartwell et al., 1992; Wallace, 1991b)
- *Study 5.* LA County, summer (Jun-Jul) 1987
  (Hartwell et al., 1992; Wallace, 1991b)
- *Study 6.* Woodland, summer (May-Jun) 1990
  (Sheldon et al., 1992)

The LA study area comprised several communities in Southern California with a total population of 360,000: El Segundo, Manhattan Beach, Redondo Beach, Torrance, Hermosa Beach, Carson, West Carson, Lomita, and several other census tracts. This area was chosen for its industrial activity, elevated levels of photochemical smog, and its water supply properties (Pellizzari et al., 1987).[1] The Pittsburg/Antioch area, with a total population of 91,000, was chosen because it was in Northern California, it had industrial activity, and the meteorological conditions were unique (Pellizzari et al., 1987). Woodland was selected to represent a medium-size city (population 32,000) in the central valley of California that had a population with different socioeconomic,

[1]El Segundo, Manhattan Beach, Redondo Beach, Torrance, and Hermosa Beach are all coastal cities where the levels of photochemical smog are relatively low compared to the inland areas of Southern California included in the LA study.

employment, and lifestyle patterns than the other California populations studied (Sheldon et al., 1992).

Figure 3.1 summarizes some important characteristics of the studies and illustrates the relationships among them. The five TEAM studies have many common design features such as the data collection methods and the target populations. The four studies conducted in LA county were not independent: the sample population in Studies 2, 4, and 5 were subsamples of the Study 1 population. Although Study 6 had a more varied set of objectives than the previous five, it shared similar characteristics, such as the sampling design and the compounds measured.

Four of the studies (1, 2, 3, and 6) used a statistical sampling design. Participants were selected using a three-stage stratified sample. In the first stage, certain homes were chosen on the basis of socioeconomic status and proximity to major point sources such as petroleum refineries. In the second stage, all participants answered questionnaires on age, sex, smoking status, and occupation. In the final stage, a weighted probability sample was selected from these homes to emphasize higher potential exposures. A statistical sampling design permits direct estimation of exposure distributions of the target population from the sample population by weighting each measurement with the inverse of its selection probability.

Participants in all six studies answered a general questionnaire to obtain information about the household and participant. At the end of the monitoring period, a second questionnaire was used to gather information about the participant's location and activities during the sampling period. During Study 6, participants also filled out a time-activity diary. All of this information was used by RTI to create the participant/household characteristics section of the CED.

Information needed to identify the likely sources and human activities contributing to exposures is included in the participant/household characteristics section. This section contains data that identifies each individual, and provides information on the participant and their home, such as participant age, whether their home is carpeted, occurrence of tobacco smoking during monitoring, episodes of personal exposure to specific items, and time spent in different microenvironments during the study. These data were derived from questions with different temporal characteristics. Some questions, such as "do you smoke?" asked about general time frame behaviors. Other questions, such as "did you smoke during the study?", referred to the study's monitoring time period.

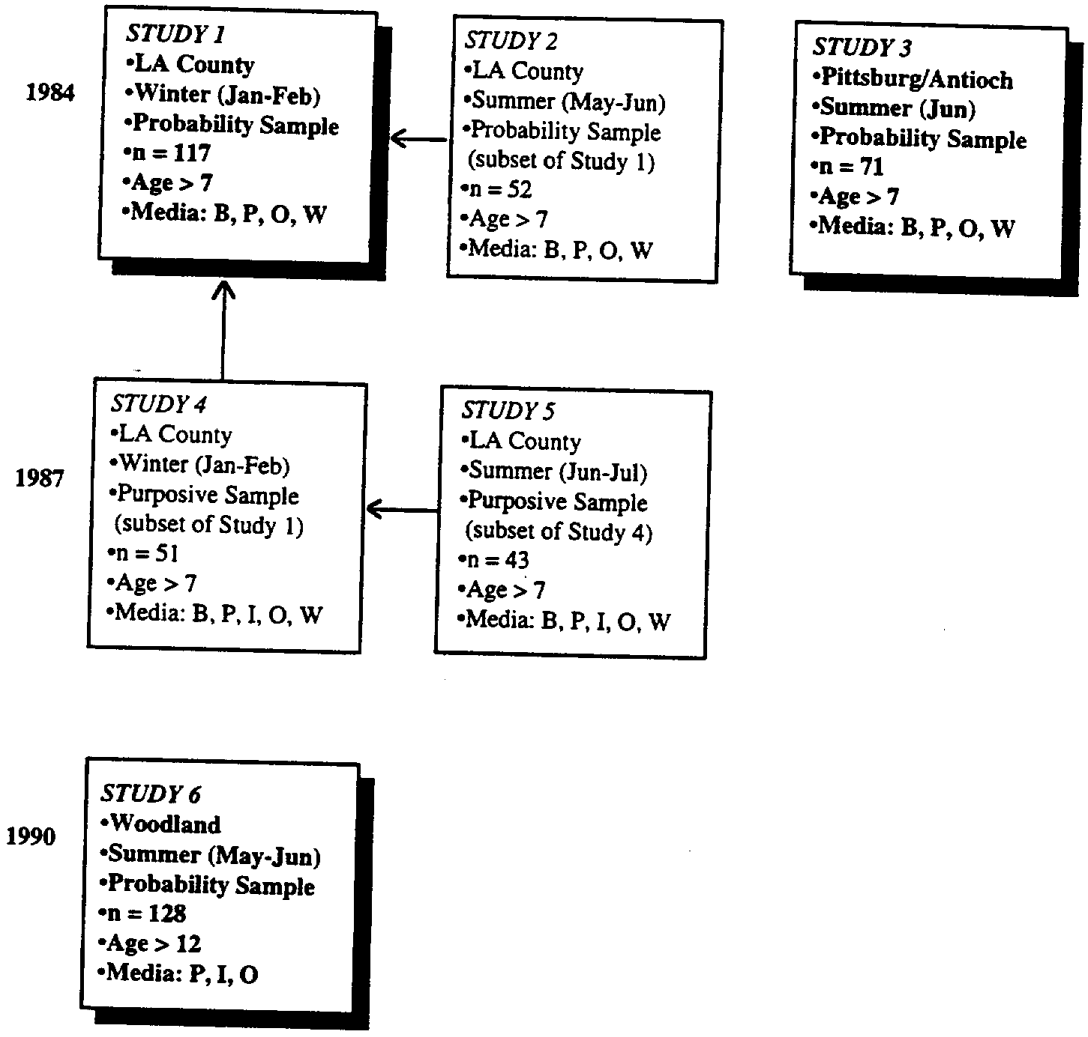

Figure 3.1 Characteristics of the six Californian exposure studies in the Californian Exposures Database (CED).

A total of 33 volatile organic compounds (VOCs) were measured during the six studies (Clayton and Perritt, 1993) and are included in the CED. Seventeen of these compounds are on the Federal list of hazardous air pollutants and, therefore, are also considered TACs by California. Concentration data were collected indoors, outdoors, and in the personal and breath zone of the sample population. In the five TEAM studies, the participants were monitored using personal air samplers over two consecutive periods, each approximately 12 hours long: the daytime sampling period was 6 a.m. to 6 p.m., and the overnight sampling period was 6 p.m. to 6 a.m. Breath samples were collected at the end of the second 12-hour period. Studies 4 and 5 also included three 12-hour indoor air samples (daytime and overnight kitchen, and daytime main-living-area). In Study 6, 24-hour indoor air and personal air samples were collected. Outdoor air concentrations were measured in all six studies during the monitoring periods. All of these measurements were used by RTI to create the breath, daytime, nighttime, and 24-hour air concentration sections of the CED.

3.3.2 Use of the CED in this Study

To estimate exposures for the Californian population, we needed to use the data for the participants included in the CED in a manner that would give the best representation of the target population. In the analysis presented here, we used only data from Studies 1, 3, and 6, because only these studies used probability-based sampling. In addition, each of these studies was conducted in a distinct region of California: (a) Study 1 - Los Angeles county, in Southern California (Hartwell et al., 1987; Wallace et al., 1988); (b) Study 3 - Pittsburg/Antioch, in mid-Northern California (Hartwell et al., 1987; Pellizzari et al., 1987; Wallace et al., 1988); and (c) Study 6 - Woodland, in the Central Valley of California (Sheldon et al., 1992). Studies 2, 4, and 5 were excluded because they were follow-on studies to Study 1: some participants from Study 1 were also monitored in Studies 2, 4, and 5. Studies 4 and 5 were also excluded because they used purposive sample designs and did not report sampling weights.

To determine participants' smoking and ETS exposure status, we used information in the CED that had been extracted from questionnaires filled out by study participants. Two different questionnaires were administered during the study, one obtaining general information about the household and the participant characteristics and one gathering information on the participant's activities and associated microenvironments during the 24-hour monitoring period (administered at the end of each 24 hours of monitoring).

We classified participants into three subpopulations with respect to smoking: active, passive, and unexposed. This classification was accomplished by using two variables, PX41 and PX42, in the CED. These two variables provided information specifically about smoking and exposure during the 24-hour monitoring period. PX41 indicated whether the participant smoked tobacco

products during the monitoring; PX42 indicated whether the participant was in an enclosed area with smokers during the monitoring.

The only means we had to identify participants' ETS exposure status was by their questionnaire answers. Misreporting can introduce errors when relying on proxy reports of smoking status. The incidence rate of misreporting is not precisely known. The proportion of people who say that they are nonsmokers but who in fact smoke, appears to range from 0.5 to 3%, depending on the population studied and the questionnaire used (Wald et al., 1984; Wald et al., 1986; National Research Council, 1986; Lee, 1991).

Since the potential biasing effect of misreporting smoking status may be significant, we attempted to find individuals in the CED who may have been misclassified. This analysis revealed 19 people (roughly 5% of the total study population) whose answers were inconsistent. For example, five people claimed to have smoked during the 24-hour monitoring period despite placing themselves in the category of people who had never been a smoker. However, the effects of this possible misclassification appear minimal. The arithmetic mean, 24-hour benzene exposure concentration increased by only 1% when the five "never-smokers" who indicated that they had smoked during the study were included in the sample population, compared to the mean of the sample in which they were excluded. Similar results were obtained for other compounds. Because the effects appear relatively small, all of our analyses were conducted with the entire exposure subpopulations as originally classified.

The personal exposure measurements in the CED comprise both 12-hour and 24-hour time-weighted average personal exposure concentrations. We focused only on the 24-hour data to avoid the additional complication that would be introduced by analyzing different sampling durations; also, Study 6 only collected 24-hour samples.

The ultimate objective of this study was to make inferences about exposures of the Californian population from the participants' exposures described in the CED. The confidence in the accuracy of any inference about the target population depends on the size of the sample. In general, the optimal sample size for an investigation depends upon the magnitude of the difference that one is trying to detect, the sample variability, and the type of statistical procedure (Dowdy and Wearden, 1983). As the sample size increases, confidence in the sample statistics also increases. Small sample size constitutes one of the major limitations of our investigation, especially because the fractional difference that we are trying to detect between the ETS-exposed and unexposed populations is small. For individual studies, with population stratified according to smoking and ETS exposure status, the smallest sample size was 11, for the active and passive subpopulations in Studies 4 and 5, and the largest sample size was 53, for the unexposed subpopulation in Study 1. Because of the small sample sizes, we expect that there will be large uncertainties associated with our results and that such uncertainties could be reduced by increasing the sample size—that is, by

making more exposure measurements. As part of our research, we quantified the uncertainty in our estimate of the exposure from ETS caused by the small sample sizes; these results are presented §4.3.1.

Our assessment of exposures to air toxicants associated with ETS only addressed those compounds listed in Table 1.1. Analyses of the CED focused on five target compounds, listed in bold-italic face in Table 1.1—benzene, ethylbenzene, styrene, o-xylene, and m,p-xylene. Previous studies have shown that tobacco smoke is one of the main indoor sources of these air toxicants. Exposures for the other compounds are determined based on their relative emission factors in comparison with those of the five target compounds.

Most measurements in the CED were above the quantifiable limit (QL) for the compounds of interest in our study. Measurements below the QL were assigned a concentration value by RTI based on the QL or limit of detection (Clayton and Perritt, 1993). Specifically, for the data that we used in our analysis (Studies 1, 3, and 6), only one measurement was below the QL. Thus, we used all of the values as reported in the CED in our analysis.

3.3.3 Computer Implementation

Prior to analyzing data, the CED had to be installed on our computers. This database consists of 17 ASCII data files: five files contain the basic data, and 12 auxiliary files contain supplementary information. We used only three of the basic data files in our analyses: the participant/household characteristics, the daytime and nighttime concentrations in air, and the 24-hour concentrations in air. The files we did not use contained water and breath concentrations. The size of the entire database is approximately 2.1 megabytes; the files we used totaled 1.8 megabytes. We installed the database on a DEC workstation, using the UNIX[2] operating system.

For most analyses we used the SAS[3] System. SAS is an integrated applications system that includes a powerful programming language used to store, retrieve, and modify data, conduct statistical analyses, and produce reports. The CED was structured to allow many types of analyses using SAS, and, in fact, the report accompanying the CED contained several SAS programs suggested for use in analysis of the data, some of which we used. In addition to using SAS programs, the derivation of the exposure distributions was accomplished by using Fortran computer programs and UNIX shell programs.

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[2]UNIX is a registered trademark of AT&T.

[3]SAS is a registered trademark of SAS Institute, Inc.

3.4. Activity Patterns of California Residents (APCR)

The APCR data were used to model exposures of adults and adolescent nonsmokers in Phases II and III of the project. Information from the APCR was also used in Phase I to estimate the proportion of nonsmoking Californians who were exposed to ETS on a daily basis.

3.4.1 Overview

In conjunction with its program to identify and combat sources of air pollutant exposure, the California Air Resources Board sponsored the Activity Patterns of Californian Residents study. The primary objective of this study was to determine the proportion of time spent in various locations by Californians in general, and by demographic and socioeconomic subgroups of Californians (Wiley et al., 1991a; Jenkins et al., 1992). The target populations for this study were adult residents of California aged 18 and older and adolescents, aged 12–17.

The APCR data were generated through a randomly dialed telephone sampling of Californian residences. Telephone interviews were conducted over the course of a year (October 1987 through September 1988) to include the seasonal variation of responses. A total of 1762 interviews were conducted during this time: 1579 with adults and 183 with adolescents. Each respondent was interviewed only once. Weighting factors in the data files are available and were used to account for deliberate oversampling of the San Francisco Bay Area and the rest of California, relative to the South Coast region (Los Angeles and San Diego). The weighting factors also correct for the uneven distribution of sampling by season.

The APCR files contain two types of data. First, each respondent was asked a series of interview questions. These included general questions about their family, home, and occupation, as well as those concerning specific activities during the previous day. These questions ranged from very general, such as the number of people living in their household or the number of hours per week they usually work, to very specific, such as the number of minutes the heat was on in their home during the previous day. Second, each respondent was asked to recount a "24-hour diary" of their activities from the previous day, from midnight to midnight. For each diary activity, respondents were asked to identify their location, the exact time (to the nearest minute) when the activity began and ended, and whether they were in the presence of a smoker.

3.4.2 Use of the APCR in this Study

We focused our study on assessing the exposure of nonsmokers who were exposed to environmental tobacco smoke, separately evaluating exposure for adults and adolescents. As shown in Figure 2.1, 625 nonsmoking adults and 98 nonsmoking adolescents reported exposure to ETS in the study. We excluded adults and adolescents who smoked because their exposure to

air toxics in mainstream cigarette smoke greatly exceeds that associated with secondhand smoke generated by others.

To calculate individuals' total exposure, seven distinct indoor microenvironments were considered:

1. Residential
2. Office/Occupational
3. Retail/Other indoor locations
4. Restaurant
5. Bar/nightclub (for adults, only)
6. Transportation
7. Residential guest
8. School (for adolescents, only)

These categories were chosen in an attempt to consolidate the locations where significant ETS exposure occurs into a manageable number of distinct microenvironmental classes. These categories include all of the indoor environments in the APCR where ETS exposure was reported to occur. Tables 3.2 and 3.3 lists the individual locations that we combined into microenvironmental classes. We excluded 12 locations from our model (most of which are outdoors), out of a total of 46 separate activities.

To meet our study goals, we needed to determine each APCR participant's smoking status. The participants were not asked if they were smokers or if they were former smokers. Instead, all of the smoking-related questions were about smoking *activities*. We used the response to the following question to distinguish nonsmokers from smokers: "Did you smoke any cigarettes yesterday – even one?" If a participant answered "yes" to this question, we labeled him or her as a smoker; if the response was "no," the participant was labeled as a nonsmoker. The weighted percentage of APCR adults and adolescents who smoked the previous day was 22% and 6%, respectively.

Once we identified all nonsmoking participants, we needed to determine whether they were exposed to ETS. The data in the APCR study that addressed when and where a nonsmoker was exposed to ETS was found in the 24-hour diary section. Along with their location and activity at each moment in the day, participants were also asked whether a smoker was present during that activity: "Were you around anyone (else) who was smoking a cigarette, cigar, or pipe while you were (doing that activity)?" If a study participant answered yes to this question, then we assumed an ETS exposure occurred. We refer to this question and its response as *self-reported proximity (SRP)*.

Table 3.2. Microenvironmental locations and codes from the Activity Patterns of California Residents (APCR) study, as grouped for assessing adult exposures to ETS.

location code	location	location code	location
RESIDENTIAL		RESIDENTIAL GUEST	
1	in kitchen at home	32	at other's home
2	in living room, family room, den at home	TRANSPORTATION	
3	in dining room at home	51	in car
4	in bathroom at home	52	in pickup truck/van
5	in bedroom at home	55	on bus
6	in study/office at home	56	on rapid transit
7	in garage at home	57	in other truck
8	in basement at home	OTHER	
9	in utility/laundry room at home	(not included in our analysis)	
12	room to room at home	10	in pool, spa at home
13	in other household room	11	in yard, patio at home
RETAIL/OTHER		34	at park, playground
23	at grocery store	38	at amusement park
24	at shopping mall	40	other outdoor location
27	at hospital	53	walking
30	at church	54	at bus stop, train, ride stop
31	at indoor gym	58	on airplane
33	at auto repair, parking garage, gas station	59	on bicycle
35	at hotel, motel	60	on motorcycle, scooter
36	at dry cleaner	61	other transportation
37	at beauty parlor, barbor shop	99	unknown location
39	other indoor location		
OFFICE			
21	at office building		
22	at industry plant, factory		
25	at school		
26	at other public place		
38	at work, varying places		
RESTAURANT			
28	at restaurant		
BAR/NIGHTCLUB			
29	at bar, nightclub		

Table 3.3. Microenvironmental locations and codes from the Activity Patterns of California Residents (APCR) study, as grouped for assessing adolescent exposures to ETS.

location code	location
RESIDENTIAL	
1	in kitchen at home
2	in living room, family room, den at home
3	in dining room at home
4	in bathroom at home
5	in bedroom at home
6	in study/office at home
7	in garage at home
8	in basement at home
9	in utility/laundry room at home
12	room to room at home
13	in other household room
RETAIL/OTHER	
23	at grocery store
24	at shopping mall
27	at hospital
30	at church
31	at indoor gym
33	at auto repair, parking garage, gas station
35	at hotel, motel
36	at dry cleaner
37	at beauty parlor, barbor shop
39	other indoor location
OFFICE	
21	at office building
22	at industry plant, factory
26	at other public place
38	at work, varying places
SCHOOL	
25	at school

location code	location
RESTAURANT	
28	at restaurant
BAR/NIGHTCLUB	
29	at bar, nightclub
RESIDENTIAL GUEST	
32	at other's home
TRANSPORTATION	
51	in car
52	in pickup truck/van
55	on bus
56	on rapid transit
57	in other truck
OTHER	
(not included in our analysis)	
10	in pool, spa at home
11	in yard, patio at home
34	at park, playground
38	at amusement park
40	other outdoor location
53	walking
54	at bus stop, train, ride stop
58	on airplane
59	on bicycle
60	on motorcycle, scooter
61	other transportation
99	unknown location

Figure 3.2 shows the distribution of self-reported proximity time among nonsmokers exposed to ETS in the APCR. Ninety percent of the total time that nonsmoking adults reported in exposed to ETS is accounted for by the time spent in seven microenvironment groups we modeled (Figure 3.2); for adolescents, 92% of the self-reported proximity time is included in the seven modeled microenvironmental groups. Because self-reported proximity probably underestimates time of exposure in one's own residence and in occupational settings, the percentage of exposure that is not modeled in this study is expected to be significantly less than the 8-10% of self-reported proximity time in the "other" microenvironment class.

We considered but rejected the possibility of trying to estimate exposure in the "other" group of microenvironments. The difficulty, of course, is these exposures mainly occurred outdoors. Exposure concentrations could vary over orders of magnitude, from relatively high if the nonsmoker is very close and directly downwind of the smoker to essentially zero if the nonsmoker is several meters away and upwind. There is no way to make even a reasonable estimate, given only self-reported proximity and the type of activity being undertaken, the ETS exposure concentration outdoors. Because air movement and pollutant dispersion is much stronger outdoors than indoors, we expect that the average exposure concentrations in the group of microenvironments "other" is significantly smaller than it is indoors. Therefore, we expect that the size of the bias associated with not modeling these sources to be very much smaller than the percentage of the self-reported proximity in these settings.

We note, however, that the data in Figure 3.2 refer to conditions in the late 1980's when smoking was still widely permitted in public buildings in California. Since the implementation of AB13, one expects that the proportion of cigarettes smoked outdoors to have increased substantially, and, therefore, that the significance of exposure to ETS in the category "other" is greater for the late 1990's than for the late 1980's.

In addition to excluding the microenvironments "other" from the assessment, we also excluded all individual nonsmokers who reported being exposed to ETS only in these microenvironments. The weighted percentage of nonsmoking adults in the APCR survey who reported being exposed to ETS the previous day was 56%. Eliminating those only exposed in the "other" group of microenvironments reduced this percentage to 52%. For adolescent nonsmokers, the weighted percentage who reported being exposed in any microenvironment was 68%; excluding those exposed only in "other" reduced the fraction to 63%.

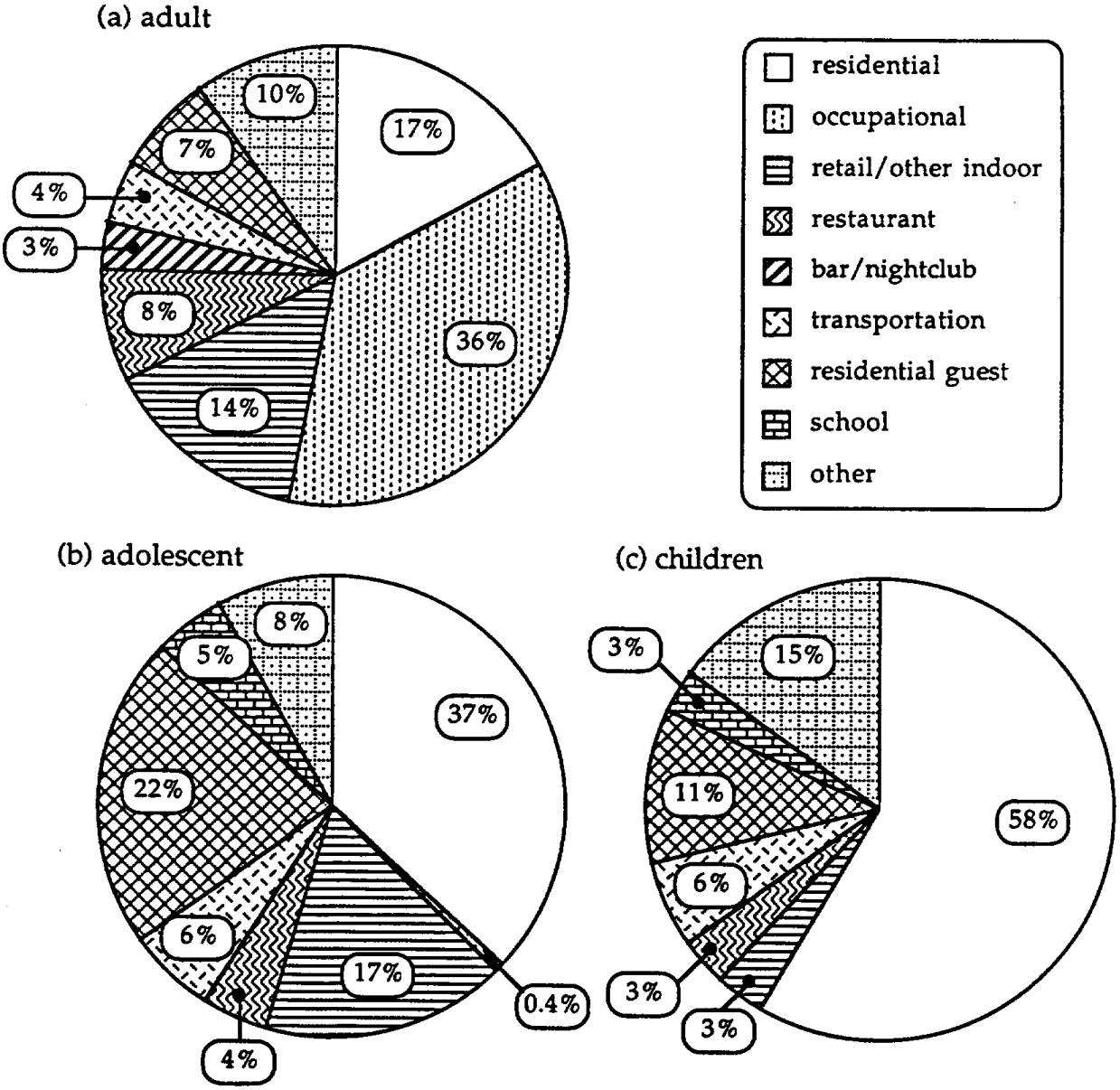

Figure 3.2 Distribution of minutes of self-reported exposure (weighted) to ETS for nonsmokers: (a) adults in the APCR study; (b) adolescents in the APCR study; and (c) children in the CAP study.

Figure 3.3 shows the weighted fraction of the nonsmoking population exposed in different microenvironments, based on the APCR data (for adults and adolescents) and the Children's Activity Pattern data (for children; see the following section). The figure shows, for example, that 11.8% of all nonsmoking adults reported being in the presence of someone smoking in their home during the previous day.

Once we determined that a nonsmoking participant was exposed to ETS during an activity, we estimated the duration of his or her exposure. This evaluation was not straightforward in all cases. In the APCR data, each activity of a participant is associated with the time spent doing that activity. This question arises: if a participant reports being in the presence of a smoker during an activity, should the ETS exposure time be the entire duration of the activity or the SRP time?

The primary difficulty arises because of the vagueness of the SRP question. An affirmative answer definitely indicates exposure, but a negative response does not conclusively indicate the absence of exposure. In residences and in large buildings, one may be exposed to environmental tobacco smoke even if not in the immediate proximity of a smoker. Of course, SRP also provides no information on the intensity of the exposure.

Despite these difficulties, we used SRP time as the measure of ETS exposure time in all but two of the microenvironmental classes. We used this approach because of the specific method used to gather APCR diary data. People reported on their day in small increments of time, broken down by location. The participants were asked, "What did you do next?... How long were you there?... Were there any smokers present during this activity?" So, for example, if someone was in a restaurant, and smoking only occurred for part of their stay, we have assumed that they would label that entire activity as being "in the presence of a smoker."

The two exceptions to this treatment are for the residential and occupational microenvironments. At these sites, we treated the SRP response as a binary switch. If a participant reported proximity at some time during the day in either microenvironment, we assumed that ETS exposure occurred in that setting. Instead of SRP time, however, we separately determined an exposure duration for that day. (In most cases, this was the nonsleeping hours spent indoors at home; see Section 5.1.3.1 for details.) Conversely, if no proximity was reported, we assumed that ETS exposure was zero in that setting. This approach is consistent with how we estimated ETS concentrations. Our procedure for estimating concentrations generates estimates of awake-hour average concentrations for residences and working-hour average concentrations for occupational microenvironments.

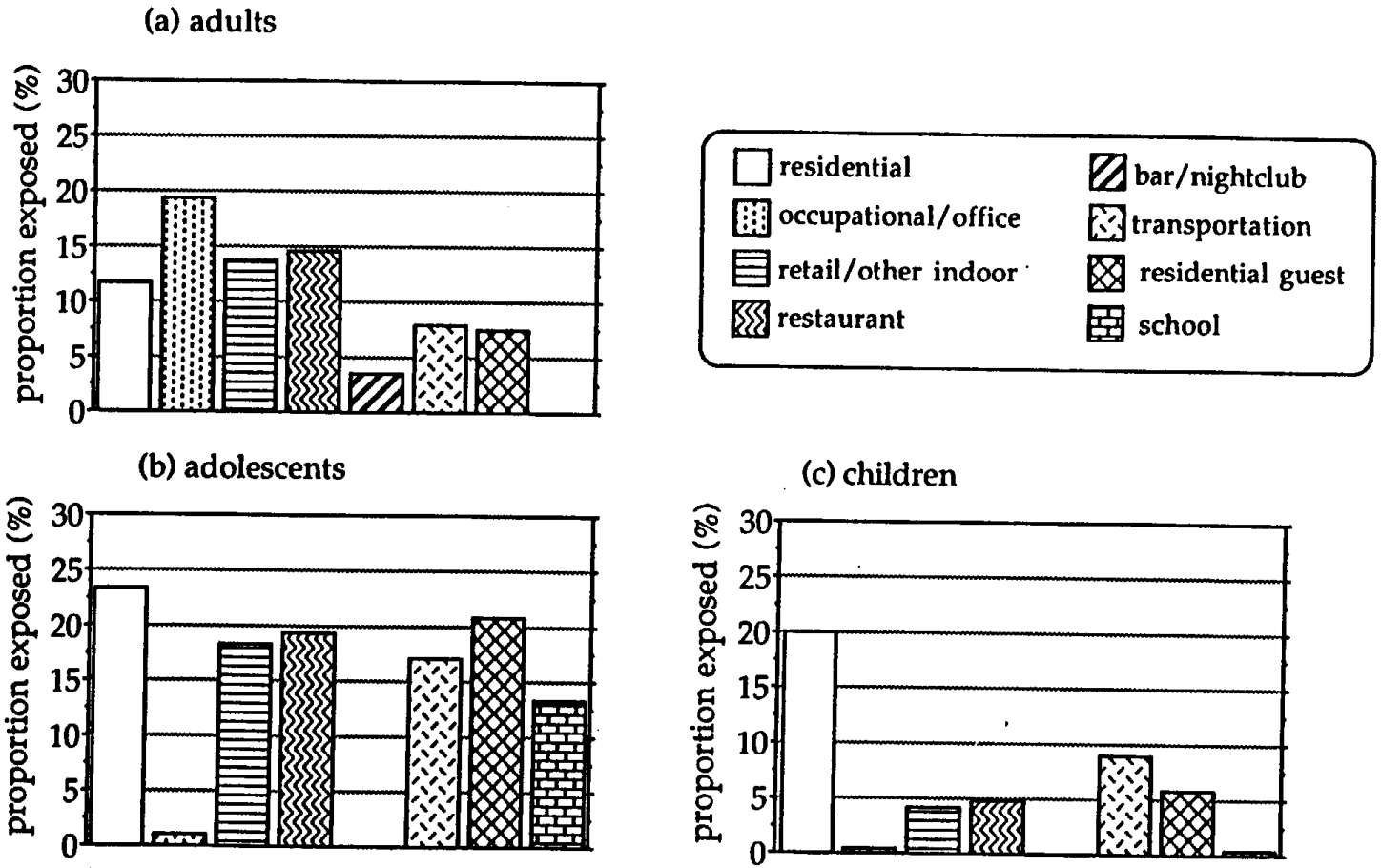

Figure 3.3 Percentage (weighted) of nonsmokers reporting exposure to ETS in different microenvironments: (a) adults in the APCR, (b) adolescents in the APCR, and (c) children in the CAP study.

We conducted the following analysis of the adult data to scrutinize whether self-reported proximity time reasonably reflects ETS exposure duration. An activity in a particular microenvironment that was reported as being in the proximity of a smoker was termed an SRP activity. Similarly, an activity not reported as being in the proximity of a smoker was termed a nonSRP activity. We determined the percentage of occurrences that an SRP activity was followed by a nonSRP activity in the *same* microenvironment. For five of the seven microenvironments (residences and workplaces excluded), this percentage was 3–15%. This result suggests that in these microenvironments respondents did not consider the end of active smoking in their presence as terminating their activity. Consequently, SRP time should be a good surrogate for exposure duration in these five microenvironments.

For residences and workplaces, however, adult nonsmokers reported that an SRP activity was followed by a nonSRP activity 78% and 39% of the time, respectively. Thus, in these two microenvironments, SRP time is not a suitable proxy for ETS exposure time. Activities in residences and workplaces are actually designated in the APCR study by the room of the house or the work area in which the person is located. When people leave a room or area where smoking occurs, they may no longer be in the proximity of a smoker. This does not, however, indicate that ETS exposure has been terminated or that the exposure is insignificant. Because of internal air exchange between different zones in a building, exposure can occur in rooms where smoking does not. We judged that the most accurate assessment of exposure period for those exposed in residences and workplaces is based on the total time in these settings, rather than self-reported proximity time.

Since misreporting of smoking and ETS exposure status may bias our results, especially when questionnaire answers are the only means to identify these characteristics, we examined the APCR for participants who may have been misclassified. We compared variables representing answers to a series of questions, all of which related to smoking: "Did you smoke any cigarettes yesterday – even one?" and "Roughly, how many cigarettes did you smoke yesterday?" We found no inconsistencies in the answers to these questions. Misclassification appears not to be an issue in this study—all participants who answered they had smoked yesterday, also answered that the number of cigarettes they smoked was greater than zero; those saying they hadn't smoked, answered that the number of cigarettes smoked was zero.

3.4.3 Computer Implementation

Prior to our analyses, the APCR data were installed on our computers. The seven files containing the raw data occupy approximately 13 megabytes of disk storage space. Three of these files contained questionnaire responses from the telephone interviews, and the last four contained 24-hour diary information. In our study, we used only the four files which contained the interview

and diary data for adult and adolescent respondents. We installed these data on a DEC workstation, using the UNIX operating system.

For most analyses, we used the Statistical Analysis Software (SAS) System. SAS is an integrated applications system that includes a powerful programming language used to store, retrieve, and modify data, conduct statistical analyses, and produce reports. Sample SAS programs and results are included in a separate appendix.

3.5 Study of Children's Activity Patterns (CAP)

As a follow-on study to the APCR, the California Air Resources Board sponsored the study of Children's Activity Patterns (CAP). This study, conducted by the same investigators as the APCR, ascertained activities, locations and potential exposure to selected indoor air pollution sources for 24-hour periods (Wiley et al., 1991b). The target population was California children aged 0-11. We used the CAP data to model exposures of children to air toxicants from ETS in Phases II and III of the project.

The CAP data were generated through a randomly dialed telephone sampling of Californian residences. Telephone interviews were conducted over the course of a year (April 1989 through March 1990). A total of 1200 interviews were conducted, either directly with the child (if age 9-11), or with an adult respondent who lived in the household. Weighting factors in the data files are available and were used to account for deliberate oversampling of the San Francisco Bay Area and the rest of California, relative to the South Coast region (Los Angeles and San Diego). The weighting factors also correct for the uneven distribution of sampling by season. The study method excluded households without telephones and in which there was no English-speaking adult. The overall response rate was 78%.

As much as possible, the CAP data were managed like the APCR data. The activity and location codes were somewhat distinct. Our clustering of these codes into microenvironmental groups is presented in Table 3.4. Seven groups of microenvironments were included in the assessment:

1. Residential
2. Retail/other indoor locations
3. Office
4. School or childcare
5. Restaurant
6. Residential guest
7. Transportation

Table 3.4. Microenvironmental locations and codes from the Study of California Children's Activity Patterns (CAP), as grouped for assessing children's exposures to ETS.

location code	location	location code	location
RESIDENTIAL		RESIDENTIAL GUEST	
1	in kitchen at home	3201	in kitchen at other's home
2	in living room, family room, den at home	3202	in living room, family room, den at other's home
3	in dining room at home	3203	in dining room at other's home
4	in bathroom at home	3204	in bathroom at other's home
5	in bedroom at home	3205	in bedroom at other's home
6	in study/office at home	3206	in study/office at other's home
7	in garage at home	3207	in garage at other's home
8	in basement at home	3208	in basement at other's home
9	in utility/laundry room at home	3209	in utility/laundry room at other's home
12	room to room at home	3212	room to room at other's home
13	in other household room	3213	in other household room at other's home
RETAIL/OTHER		TRANSPORTATION	
23	at grocery store	51	in car
24	at shopping mall	52	in pickup truck/van
27	at hospital	55	on bus
30	at church	56	on rapid transit
31	at indoor gym	57	in other truck
33	at auto repair	69	other closed transit
35	at hotel/motel		
36	at dry cleaner		
37	at beauty parlor		
39	other indoor location		

Table 3.4. (continued)

location code	location
OFFICE	
21	at office building
22	at industry plant, factory
26	at other public place
SCHOOL/ CHILDCARE	
251	at school
253	at childcare, house
255	at childcare, commercial
259	at other school/childcare
RESTAURANT	
28	at restaurant

location code	location
OTHER	
(not used in our model)	
11	in yard, patio, other outside house at home
3211	in yard, patio, other outside house at other's home
22	at plant, factory
26	at public building (e.g., museum, library, theater)
29	at bar, nightclub
34	at park/playground
38	at amusement park
40	other outdoor location
53	walking
54	at bus stop
58	on airplane
59	on bicycle/skateboard/roller skate
60	on motorcycle
63	in stroller/carried by adult
70	other outdoor transit
998	don't know, can't say

Among the 1200 subjects, ETS exposure was reported to occur some time during the day for 38% (weighted). Excluding those exposed only in "other" settings, this proportion was reduced to 33%. The proportion of reported exposure time distributed among microenvironmental groups and the proportion of the study population exposed in these microenvironments are shown in Figures 3.2(c) and 3.3(c).

Figure 3.4 summarizes the percentages of the nonsmoking population groups exposed to ETS in this study. For each age group, the left-hand bar indicates the total percentage of the population reporting exposure to ETS during a day. The second bar represents the percentage that remains after excluding those who are only exposed in "other" microenvironments. The third bar represents predictions for the late 1990's and will be discussed in §6.

We note that the left-hand bars for adults (56%) and adolescents (68%) in Figure 3.4 seem to differ from the results reported by Jenkins et al. (1992). The abstract of that paper reports that "46% of nonsmokers are near others' tobacco smoke at some time during the day." However, Table 4 of that paper suggests that this number represents the fraction of the *total* population (including smokers) that is nonsmokers who were exposed to ETS. The data in Figure 3.4 excludes smokers from the denominator. The results are therefore in approximate agreement, as shown below.

Based on 1990 census data for California (Bureau of the Census, 1992), the fractions of the total population that are adults and adolescents are 74% and 8.7% respectively. Since 22% of adults smoke, the proportion of nonsmokers to smokers is 0.78:0.22 = 1.0:0.28. Therefore, 56% of nonsmoking adults exposed to ETS corresponds to a condition in which 44% (= 56%/1.28) of all adults are nonsmokers exposed to ETS. For adolescents, since 6% smoke, the proportion of nonsmokers to smokers is 0.94:0.06 = 1.0:0.064. So the statement that 68% of nonsmoking adolescents are exposed to ETS is equivalent to the statement that 64% (= 68%/1.064) of all adolescents are nonsmokers exposed to ETS. Weighting by their respective proportions in the population, we find that 46% ($44\% \times 0.74 + 64\% \times 0.087 = 46\% \times [0.74 + 0.087]$) of the population age 12 and over are nonsmokers exposed to ETS, in agreement with Table 4 of Jenkins et al. (1992).

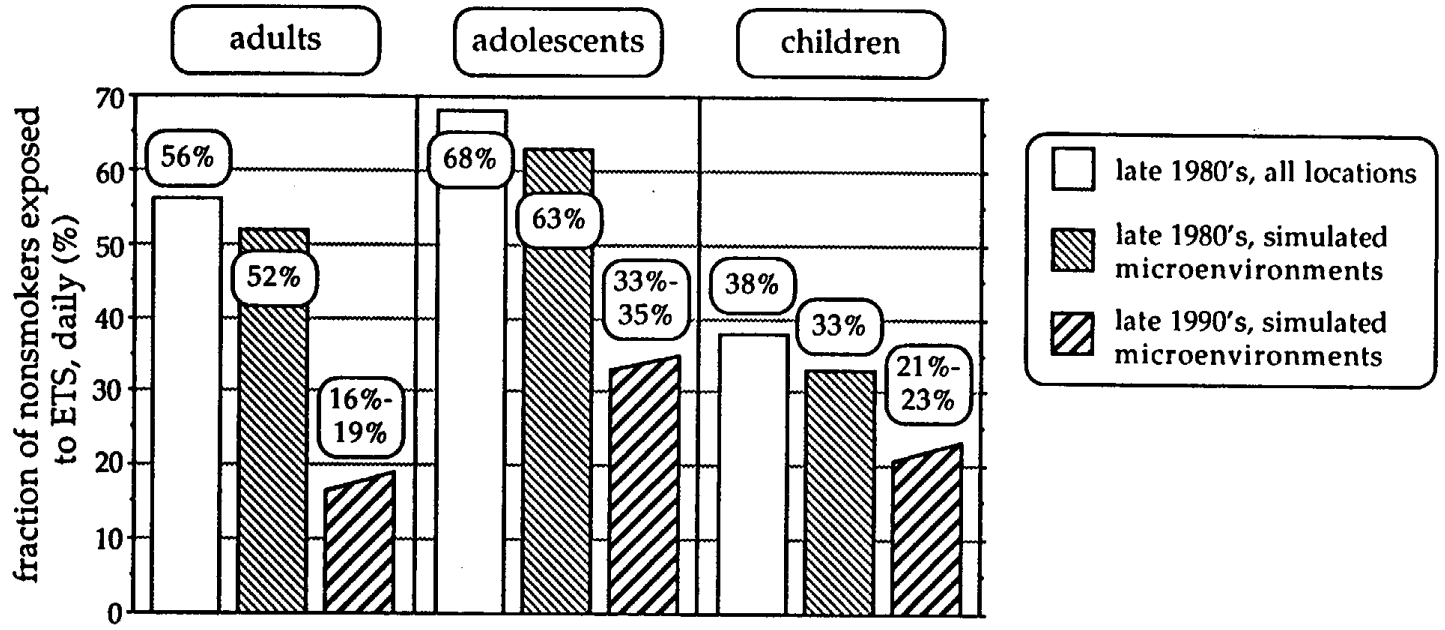

Figure 3.4 Percentage (weighted) of California nonsmokers exposed to ETS, sorted according to age group. The unshaded bar represents the direct results of our analysis of the APCR data (Wiley et al., 1991a) and the CAP data (Wiley et al., 1991b), including all locations. The bars with thin stripes represent the part of the population exposed in microenvironments included in the Phase II assessment. The bars with thick stripes represent predictions of the proportion of the nonsmoking population that remains exposed to ETS in the simulated microenvironments in the late 1990's (Phase III). The range of values for Phase III bars reflects variations in predictions among four simulation scenarios (see §6).

4. Phase I: An Assessment for late-1980's Using Personal Monitoring Data

4.1. Methods

The objective of this phase of the research was to use personal exposure measurements to estimate the contribution of ETS to the air toxicant exposure distribution for California nonsmokers aged 7 y. In essence, the method involves comparing the measured exposures of nonsmokers who report exposure to those who report no exposure. Our approach is based on the assumption that exposures are additive: the total exposure is the sum of contributions from all sources, including outdoor air, ETS, and other indoor sources. We made two additional important assumptions to estimate the ETS-only portion of exposure: (1) that the air toxicant exposure from ETS is lognormally distributed; and (2) that exposures to different compounds from ETS scale in proportion to their emission factors. These assumptions are reasonable. In support of assumption (1), we note that many environmental parameters, including species concentrations, are found to conform to lognormal distributions. Regarding assumption (2), our analysis results show that the relative amounts of excess exposure for nonsmokers exposed to ETS scale approximately in proportion to relative emission factors for four measured compounds — benzene, styrene, o-xylene, and m,p-xylene.

Exposure measurements of the type needed for this assessment are only available for a pooled study group that includes participants aged $\geq$7 y. [1] To estimate exposures for the statewide population of nonsmoking Californians, we needed to use the data for the participants included in the CED in a manner that would give the best representation of the target population. For this purpose, we constructed a pooled data set from the CED. Although, in principle, we could have used data from all six studies in the CED for our analysis (that is, used the combined data set), we chose to focus on Studies 1, 3, and 6, because each of these studies used probability-based sampling. In addition, each of the studies was conducted in a distinct region of California: (a) Study 1 — Los Angeles county, in Southern California; (b) Study 3 — Pittsburg/Antioch, in mid-Northern California; and (c) Study 6 — Woodland, in the Central Valley of California. Studies 2, 4, and 5 were excluded from our pooled data because they were follow-on studies to Study 1: some participants from Study 1 were also monitored in Studies 2, 4, and 5. Studies 4 and 5 were

[1] Study 6 only included participants aged $\geq$12 y. But since this study is used to represent only about 10% of the state's population, we can approximate that the overall pooled data set applies for the population age distribution included in Studies 1 and 3, i.e. aged $\geq$7 y.

also excluded from the pooled data because they used purposive sample designs and did not report sampling weights.

Figure 4.1 illustrates the method we devised to sample the pooled data, to match, as best possible, the data from the three studies to the Californian population. For regulating ambient air quality, California is divided into fourteen air basins, as designated by the Air Resources Board, whose boundaries are based on geographical and meteorological factors. The boundaries also follow political boundaries so far as possible (Air Resources Board, 1995). Each of Studies 1, 3, and 6 was located in a separate air basin: Study 1 in the South Coast, Study 3 in the San Francisco (SF) Bay area, and Study 6 in the Sacramento Valley air basin. To extrapolate the pooled data from these three studies to the Californian population, we assumed that the air basin population was effectively represented by the study population. We also assumed that the Californian population could be represented by the population that lives in these three air basins. A detailed comparison of the demographics of California, the three air basins, and the pooled data from the CED is presented in §4.3.2.

In Figure 4.1, the numbers of samples from each of the three studies used in our analysis are similar to but smaller than the numbers shown in Figure 3.1 for the same three studies. This is due to missing data in the CED database (either exposure measurements or information about smoking or ETS exposure status). For Studies 1 and 3, the missing data are a relatively small proportion of the total (7 of 117 and 4 of 71, respectively), but for Study 6 the difference is larger (35 of 128). The reason for this discrepancy is that only 98 personal exposure measurements of the compounds of interest were collected during Study 6 (Sheldon et al., 1992).

4.1.1 Measured Compounds: Benzene, Styrene, o-Xylene, and m,p-Xylene

Figure 4.2a summarizes our method for determining the distribution of ETS-only exposures for measured compounds. We used a Monte Carlo-based simulation to extract probability distributions for exposure from the pooled data. We derived the ETS-only distribution using two sets of personal exposure measurements from the pooled data: the subset of measurements from persons who did not smoke but were exposed to ETS during monitoring (passive), and the subset of measurements from persons who were not exposed (unexposed).

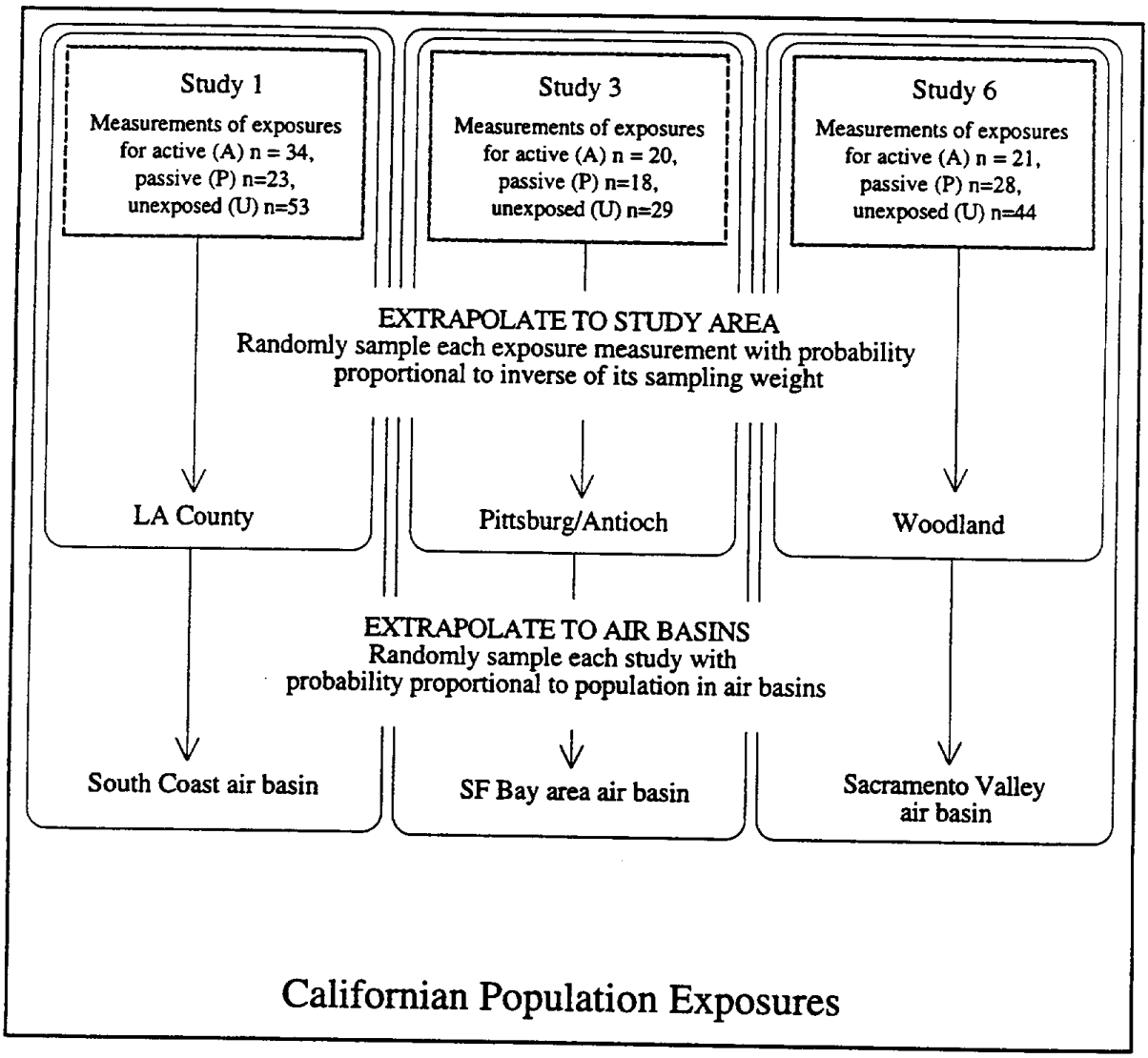

Figure 4.1 Schematic of the method used to sample from Studies 1, 3, and 6 in the CED to best represent the population of California nonsmokers.

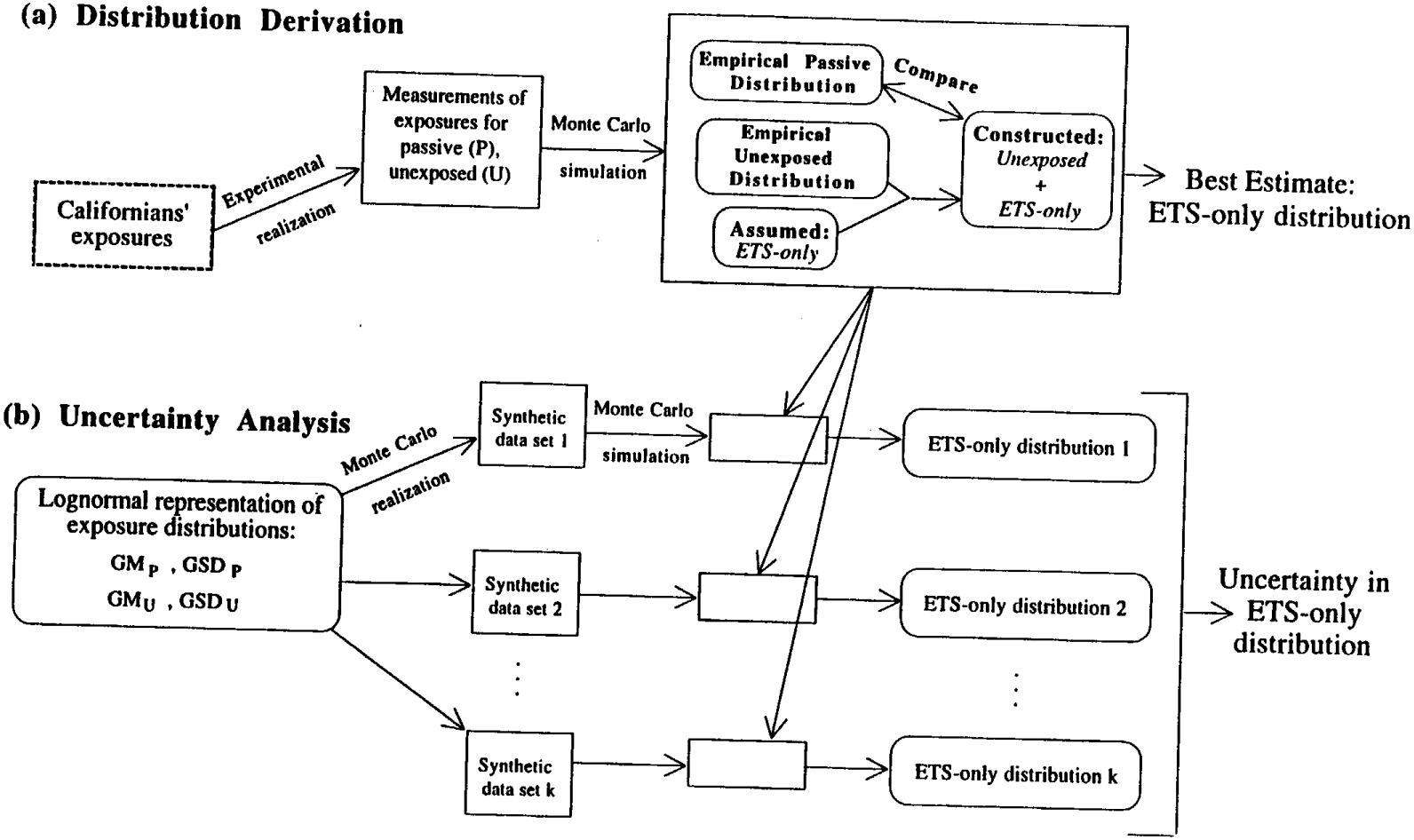

Figure 4.2 Schematic of the method used to (a) estimate the distribution of 24-hour personal exposures to air toxics due to ETS only, and (b) estimate the uncertainty in the ETS-only distribution.

The method that we used to derive the ETS-only distribution is described in detail in Appendix C. In summary, for each of the four measured species, we first derived empirical distributions from the pooled data for the passive and unexposed subpopulations. Then, we followed a three-step procedure: (1) we hypothesized the lognormal parameters for the ETS-only contributions to exposure; (2) we generated a *simulated* passive distribution, termed the *ETS-only + unexposed* distribution, by randomly sampling from the hypothesized ETS-only distribution and the empirical unexposed distribution and summing; and (3) we compared the simulated *ETS-only + unexposed* distribution to the empirical *passive* distribution using a well-established statistical method for comparing two distributions. We determined the optimal ETS-only distribution by iteratively repeating steps (1)–(3) to obtain the best agreement between the simulated and empirical passive distributions.

4.1.2 Other Toxic Air Contaminants

ETS contains many more air toxicants than those four compounds for which we had suitable exposure data to make a direct estimate of the ETS-only contribution to exposure. To estimate exposure to these other compounds, we scaled the derived ETS-only distributional parameters for benzene, styrene, o-xylene, and m,p-xylene, with a ratio of ETS emission factors. Similar approaches have been used by other researchers: Stolwijk (1990) used a measured benzene concentration distribution to scale other compound distributions; Heavner and coworkers (1995) used the ratio of measured 3-ethenylpyridine (a compound derived primarily from tobacco smoke) to VOCs to determine the proportion of personal exposure attributable to ETS. We scaled the average exposure according to the following equation:

$$X_i = \left(\frac{e_i}{e_r}\right) X_r = \left(\frac{X_r}{e_r}\right) e_i = Z\, e_i \qquad (4.1)$$

where

X_i = mean ETS-only exposure concentration for compound i ($\mu g\ m^{-3}$)

X_r = mean ETS-only exposure concentration for reference compound r ($\mu g\ m^{-3}$)

e_i = emission factor for compound i ($\mu g\ cig^{-1}$)

e_r = emission factor for reference compound r ($\mu g\ cig^{-1}$)

$Z \equiv$ ETS exposure scale factor ($\mu g\ m^{-3}/(\mu g\ cig^{-1})$, or $cig\ m^{-3}$)

Physically, the ETS exposure scale factor, Z, approximately represents the number of cigarettes smoked indoors per volume of ventilation air provided. This factor was first determined separately for each of the four compounds (benzene, styrene, o-xylene, and m,p-xylene) by dividing the population arithmetic mean ETS-only exposure by the mean emission factor. The best estimate

value was obtained by averaging these numbers. The variation in scale factors among the four species is an indicator of uncertainty associated with this method.

4.2. Results

4.2.1 Measured and Inferred Exposure Distributions

The distributions of active, passive, unexposed, and ETS-only 24-hour personal exposures that we estimated for California nonsmokers are plotted in Figure 4.3. Distributions were determined for benzene (Figure 4.3a), styrene (Figure 4.3b), o-xylene (Figure 4.3c), and m,p-xylene (Figure 4.3d). The relatively straight lines in the plots indicate that the empirical distributions are approximately lognormal. Generally, the unexposed distribution lies below the passive distribution, except at the tails, where it tends to rise above the passive distribution. The distribution for active smokers mostly lies above both of the other empirical distributions, as would be expected assuming that cigarette smoking is a significant source of these species.

Table 4.1 summarizes the univariate statistics for the 24-hour personal exposures estimated for Californian nonsmokers aged $\geq$7 y. The estimated arithmetic mean contribution for nonsmokers exposed to ETS is shown to be 1.02 $\mu g\ m^{-3}$ for benzene, 0.36 $\mu g\ m^{-3}$ for styrene, 0.77 $\mu g\ m^{-3}$ for o-xylene, and 0.99 $\mu g\ m^{-3}$ for m,p-xylene.

In addition to the postulated ETS-only distributions, we present in Table 4.1 parameters of the empirical active, unexposed, and passive distributions, and the constructed *ETS-only + unexposed* distributions. To determine whether these distributions are statistically different, a two-tailed, two-sample, unequal variance *t* test was applied to the logarithms of the data under the assumption that the data are lognormally distributed and thus the logarithms are normally distributed (Guttman et al., 1982). The difference between the *ETS-only + unexposed* and passive distributions was not statistically significant at the 0.05 level for all four compounds indicating that good agreement was achieved between these distributions. The difference between the active and passive distributions was statistically significant at the 0.05 level for all four compounds using the same *t* test. The difference, however, between the passive and unexposed distributions was not statistically significant at the 0.05 level (benzene was significant at the 0.09 level and styrene was significant at the 0.15 level). These results suggest that there is a discernible difference in exposures between the active and passive populations; the difference is smaller, however, and thus more difficult to detect, between the passive and unexposed populations.

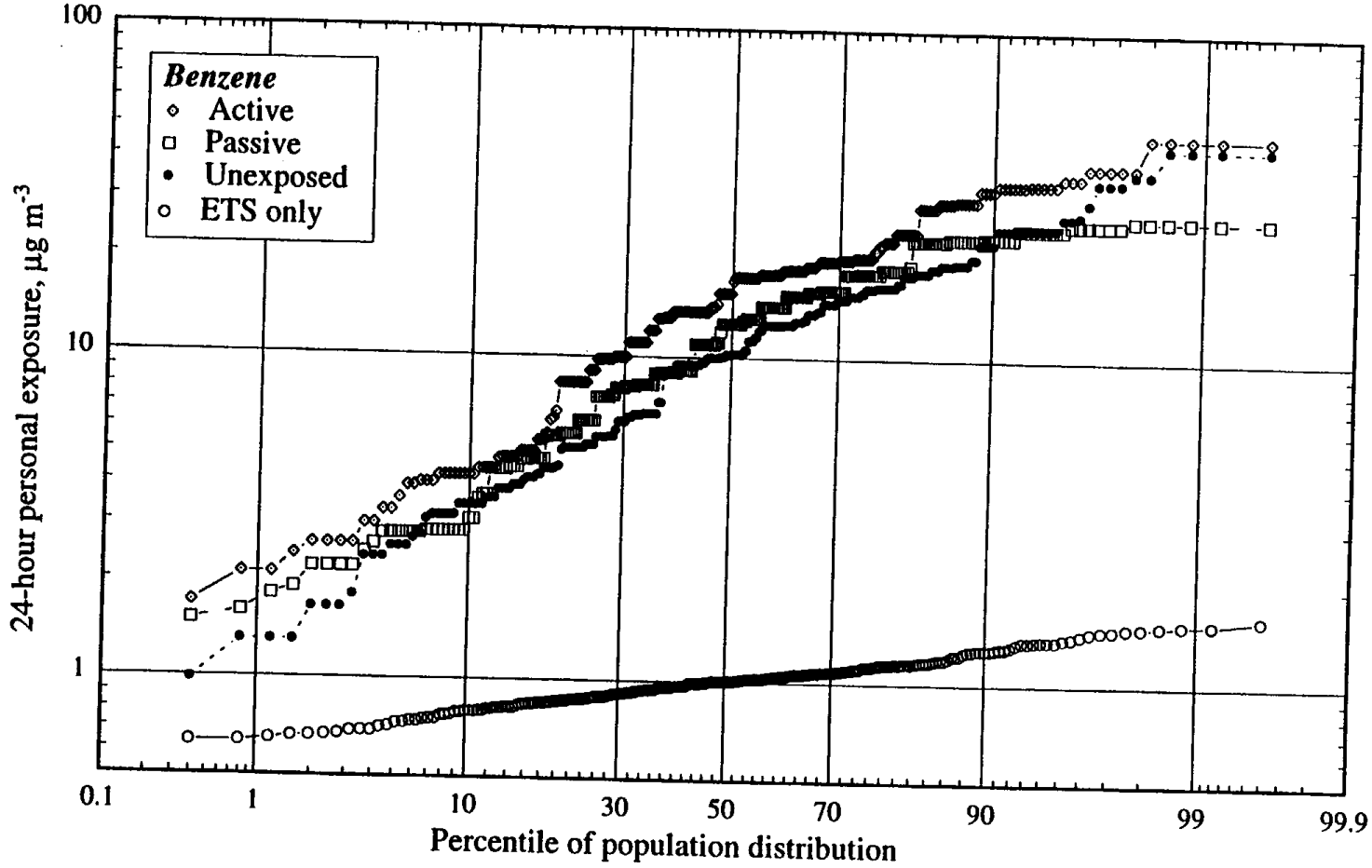

Figure 4.3a Lognormal-probability plot of the distributions of 24-hour personal exposure concentration for Californians in the mid to late 1980's, segregated by smoking/ETS exposure category, for benzene.

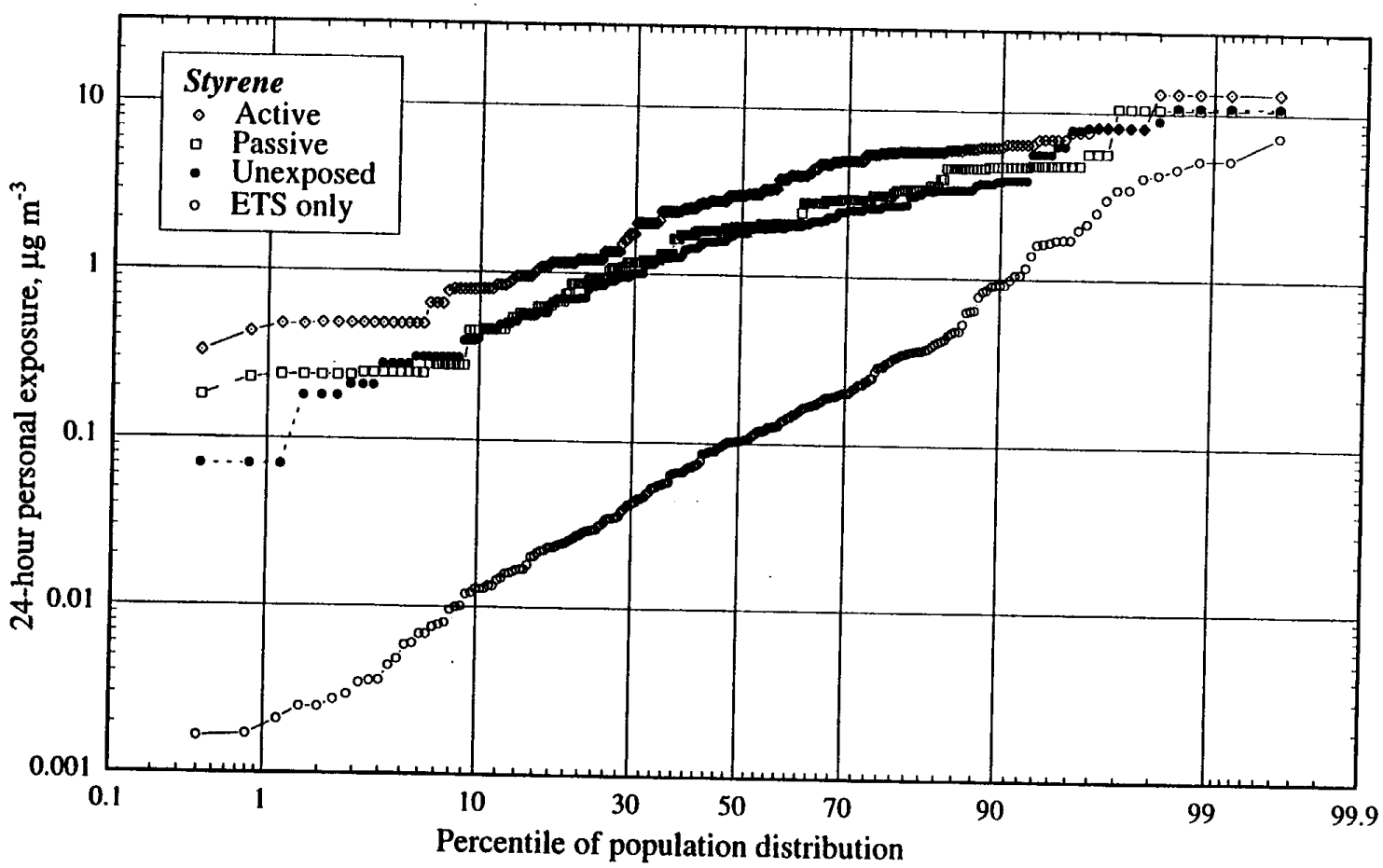

Figure 4.3b Lognormal-probability plot of the distributions of 24-hour personal exposure concentration for Californians in the mid to late 1980's, segregated by smoking/ETS exposure category, for styrene.

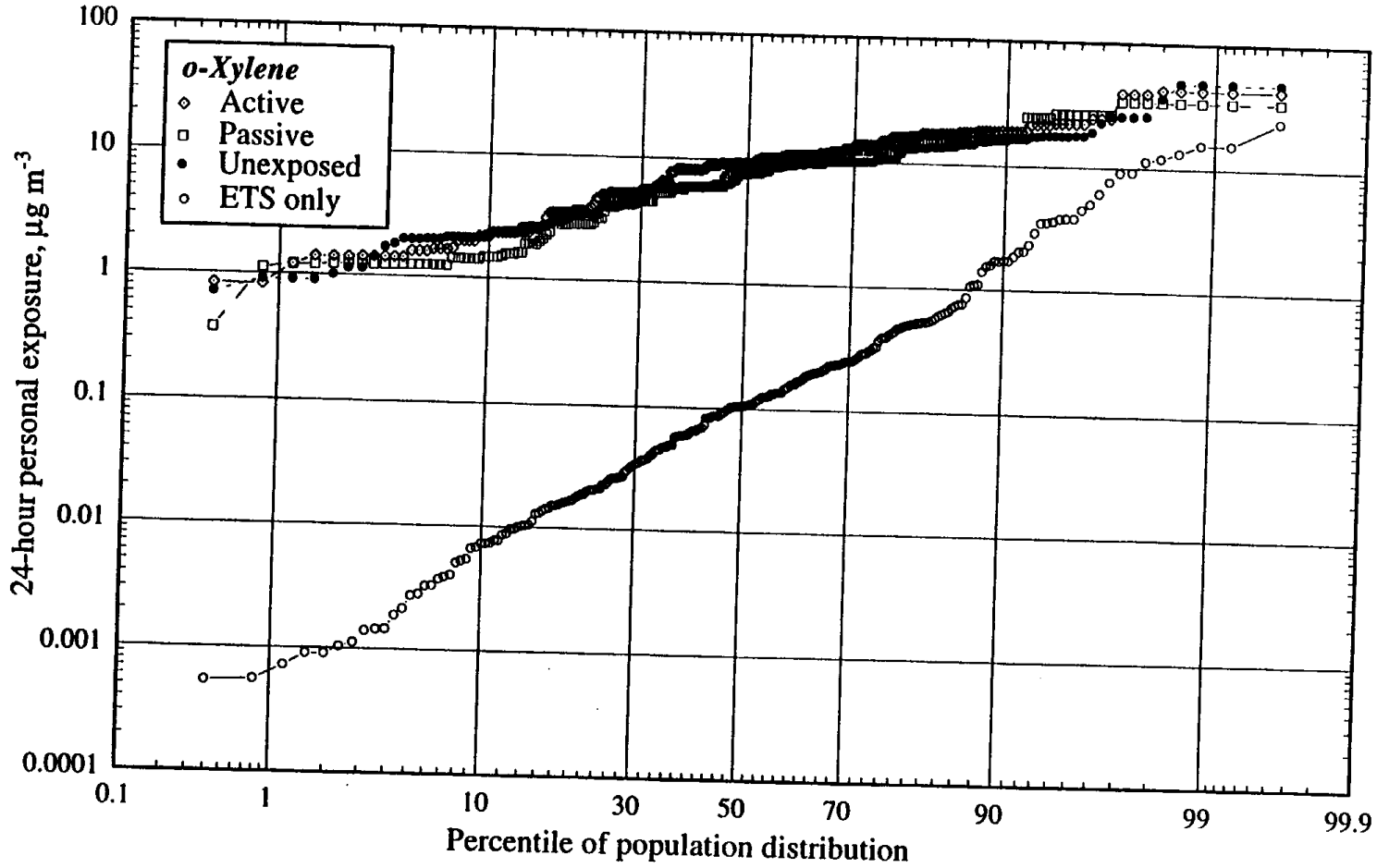

Figure 4.3c Lognormal-probability plot of the distributions of 24-hour personal exposure concentration for Californians in the mid to late 1980's, segregated by smoking/ETS exposure category, for o-xylene.

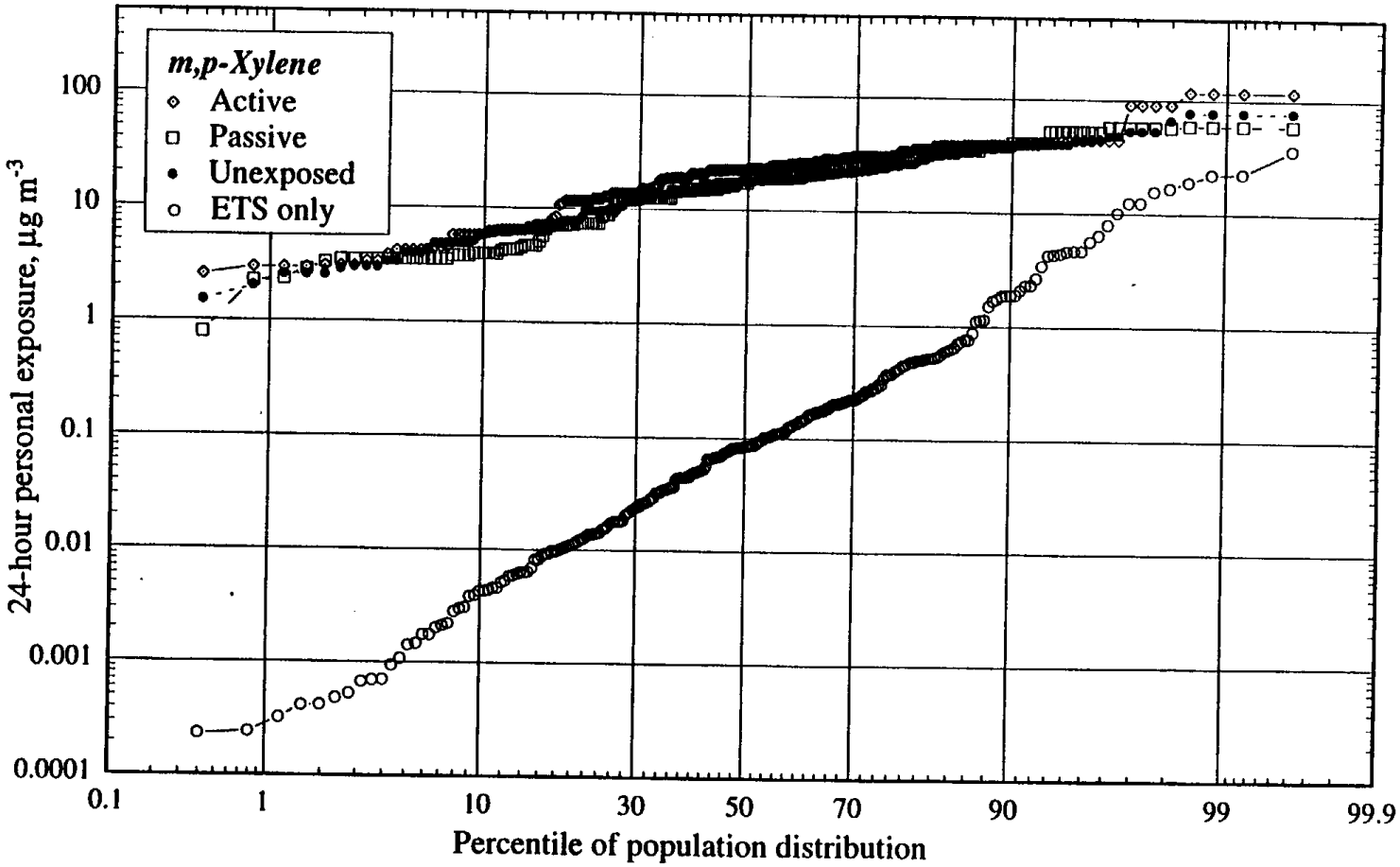

Figure 4.3d Lognormal-probability plot of the distributions of 24-hour personal exposure concentration for Californians in the mid to late 1980's, segregated by smoking/ETS exposure category, for m,p-xylene.

Table 4.1. Univariate statistics for 24-hour personal exposures, pooled data, representing the Californian population, age > 7 y, during the mid to late 1980s.[1]

compound	exposure category	# obs[2]	range (μg m^{-3})	AM (μg m^{-3})	SD (μg m^{-3})	GM (μg m^{-3})	GSD	25th %ile (μg m^{-3})	50th %ile (μg m^{-3})	75th %ile (μg m^{-3})	SE (μg m^{-3})	COV
benzene	active	75	1.50-48.4	16.9	11.2	13.0	2.2	8.27	14.8	20.4	0.11	66%
	passive	69	1.20-27.0	12.6	7.49	9.99	2.1	5.63	12.0	18.2	0.07	60%
	unexposed	126	0.35-44.9	12.0	9.08	8.95	2.3	4.55	10.0	15.9	0.09	75%
	ETS-only + unexposed		*1.06-46.6*	*13.1*	*9.08*	*10.3*	*2.0*	*5.68*	*11.0*	*17.0*	*0.09*	*70%*
	ETS only		*0.50-1.89*	*1.02*	*0.19*	*1.00*	*1.2*	*0.88*	*1.00*	*1.13*	*0.002*	*18%*
styrene	active	75	0.31-13.2	3.53	2.62	2.59	2.3	1.20	2.98	5.40	0.03	74%
	passive	68	0.18-10.6	2.39	2.14	1.62	2.6	0.87	1.95	3.08	0.02	89%
	unexposed	125	0.07-48.0	2.37	3.42	1.45	2.8	0.84	1.73	2.56	0.03	144%
	ETS-only + unexposed		*0.07-52.0*	*2.73*	*3.54*	*1.80*	*2.5*	*1.03*	*1.98*	*3.10*	*0.04*	*130%*
	ETS only		*0.0-29.4*	*0.36*	*0.96*	*0.10*	*5.1*	*0.033*	*0.10*	*0.30*	*0.01*	*268%*
o-xylene	active	75	0.83-42.2	10.8	8.59	7.69	2.5	4.60	9.28	15.0	0.09	80%
	passive	68	0.37-33.6	9.55	7.89	6.49	2.6	3.15	7.77	12.6	0.08	83%
	unexposed	126	0.50-48.1	8.83	7.85	6.36	2.3	3.31	6.65	10.6	0.08	89%
	ETS-only + unexposed		*0.50-144*	*9.60*	*8.52*	*7.03*	*2.3*	*4.04*	*7.67*	*11.7*	*0.09*	*87%*
	ETS only		*0.0-141*	*0.77*	*3.35*	*0.10*	*7.9*	*0.025*	*0.10*	*0.41*	*0.03*	*437%*
m,p-xylene	active	75	1.80-118	24.6	19.2	18.4	2.3	11.6	22.8	30.3	0.19	78%
	passive	68	0.79-59.5	20.5	14.7	15.0	2.4	7.42	18.2	27.5	0.15	72%
	unexposed	126	1.30-84.0	19.5	14.6	14.7	2.2	9.00	17.7	23.6	0.15	75%
	ETS-only + unexposed		*1.30-272*	*20.4*	*15.6*	*15.6*	*2.2*	*9.43*	*17.9*	*25.2*	*0.16*	*76%*
	ETS only		*0.0-264*	*0.99*	*5.48*	*0.080*	*10.1*	*0.017*	*0.081*	*0.38*	*0.05*	*556%*

[1]Italicized exposure categories indicate distributions constructed by the authors; the other distributions are derived from the Californian Exposures Database (see Figure 4.2a for diagram of methodology).

[2]Number of observations that resulted from pooling studies 1, 3, and 6 together. These observations were then sampled 10,000 times using a weighting scheme designed to approximate Californian population exposures (see Figure 4.1 for diagram of weighting scheme).

For three of the four compounds plotted in Figure 4.3 (styrene and the xylenes), the estimated ETS-only distributions have very steep slopes, reflecting large GSDs and suggesting that exposures caused by ETS vary greatly for these compounds. The inferred GSD for benzene is small, 1.2 (Table 4.1); a GSD of this size implies only small variability across the population. It is unlikely that the true variability in ETS-only exposures to these four compounds differs as much as suggested by these results. The relatively small variability observed in emission factors among different cigarette products supports this view (cf. small ratio of standard deviations to average for entries in Table 3.1). One would expect that the main factors that control exposure to ETS constituents, such as proximity to smokers and the amount of time exposed to ETS, would vary similarly for all compounds in ETS. The large differences in GSD between benzene and the other compounds probably reflect the small sample populations and the sensitivity of the GSD to the extremes of the empirical distributions. Estimates of the mean (arithmetic or geometric) appear more robust. These points are explored further below.

4.2.2 ETS Exposure Scale Factor and Estimates of Mean Exposure

The scale factor, Z, was determined for each of four compounds by dividing the population arithmetic mean ETS-only exposure (Table 4.1) by the mean emission factor (Table 3.1). Using the emissions data of Daisey et al. (1994, 1998) the following results are obtained: with benzene as the reference compound, $Z = 2.51 \times 10^{-3}$ cig m^{-3}; with styrene as the reference compound, $Z = 2.45 \times 10^{-3}$ cig m^{-3}; with m,p-xylene as the reference compound, $Z = 3.31 \times 10^{-3}$ cig m^{-3}; and with o-xylene, $Z = 11.5 \times 10^{-3}$ cig m^{-3}. We take the best estimate of Z to be the mean of these four results, $Z = 4.94 \times 10^{-3}$ cig m^{-3}. The range of results for the four compounds, about a factor of two from the mean, indicates the uncertainty associated with the use of different reference species in this method. The result is consistent with an estimate based on the physical interpretation of Z: smoking 15.8 cigarettes per day[2] in a 297 m^3 residence[3] with an air-exchange rate of 0.5 h^{-1} (Murray and Burmaster, 1995) would produce a scale factor of 4.4×10^{-3} cig m^{-3}.

From the best-estimate value of Z, we estimated the population mean exposure concentration for all toxic air contaminants for which Daisey et al. measured emission factors. Table 4.2 presents our estimates of the arithmetic mean 24-hour personal exposure concentration to air toxicants from ETS for that part of the Californian population exposed to ETS.

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[2]Arithmetic mean number of cigarettes smoked per smoker per day derived from the Activity Pattern of California Residents database (Wiley et al., 1991a).

[3]Geometric mean volume for a house with 3 occupants based on data from a study of US housing (Residential Energy Consumption Survey, 1982).

Table 4.2. Estimates of 24-hour personal exposure to toxic air contaminants from ETS for California passive smokers, late 1980's.[1]

	arithmetic mean ($\mu g\ m^{-3}$)		
compound[2]	CED[3]	Daisey et al. (1998)[4]	Martin et al. (1997)[5]
acetaldehyde		11	15
acetonitrile			7.0
acrylonitrile		0.49	
benzene	1.02		
1,3-butadiene		0.75	2.3
2-butanone		1.4	
o-cresol		0.17	
m,p-cresol		0.41	
ethyl acrylate[6]		<0.015	
ethylbenzene		0.64	0.49
formaldehyde		6.5	8.2
N-nitroso-dimethylamine[7]		0.0028	
phenol		1.4	
styrene	0.36		
toluene		3.2	3.1
o-xylene	0.77		
m,p-xylene	0.99		

[1] Results apply to the 56% of the nonsmoking population in California (age ≥ 7 y) that report some exposure to ETS during a day (percentage exposed based on our analysis of APCR and CAP data).

[2] Bold-italics indicate compounds for which monitoring data are in the Californian Exposures Database.

[3] Based on analysis of personal exposure measurements as reported in the California Exposures Database (Table 4.1).

[4] Arithmetic means were estimated using the ETS scale factor of $Z = 4.94 \times 10^{-3}\ \mu g\ m^{-3} / \mu g\ cig^{-1}$, the emission factors from Daisey et al. (1998) (Table 3.1), and equation 4.1.

[5] Arithmetic means were estimated using the ETS scale factor of $Z = 6.17 \times 10^{-3}\ \mu g\ m^{-3} / \mu g\ cig^{-1}$, the emission factors from Martin et al. (1997) (Table 3.1), and equation 4.1.

[6] Emission factor measured below lower limit of detection; thus, only an upper-bound estimate of the ETS-caused exposure was determined.

[7] Emission factor reported in Mahanama and Daisey (1996).

We also determined Z using the mean emission factors from Martin et al. (1997) (Table 3.1). In this case, we find the best estimate value to be 6.17×10^{-3} cig m^{-3}, the arithmetic mean value of Z for the four compounds. The range of results for the four compounds is 3.64×10^{-3} cig m^{-3} for benzene to 0.013 cig m^{-3} for o-xylene. Table 4.2 also presents estimates of mean nonsmoker exposures derived using Equation 4.1, the emission factors of Martin et al. (Table 3.1), and the value $Z = 6.17 \times 10^{-3}$ cig m^{-3}. The results are seen to be largely consistent whether the emission factors of Daisey et al. or Martin et al. are employed. For four of the five compounds for which evaluations are made using both emissions studies, the relative differences vary from 3% (toluene) to 31% (acetaldehyde). Only for 1,3-butadiene is the difference large — a factor of 3 higher using Martin et al. data than using emission factors from Daisey et al.

4.2.3 Fraction of Nonsmoker Exposure Attributable to ETS

Insight into the overall significance of ETS as a source of air toxicants can be gained by comparing the estimates of exposure caused by ETS to estimates of total exposure from all sources. Table 4.3 presents the fraction of passive smokers' exposure that can be attributed to ETS (2nd column); this fraction was obtained by dividing the mean exposure from ETS (Table 4.2) by the arithmetic mean total passive exposure (Table 4.1). The fraction of total nonsmoker (passive and unexposed) exposure that can be attributed to ETS is presented in the last column of Table 4.3. These results were obtained by dividing the mean exposure caused by ETS by the arithmetic mean, 24-hour total exposure for all nonsmokers. The mean exposure caused by ETS for all nonsmokers was estimated by scaling the values from Table 4.2 by 56% to convert from the exposed to total nonsmoking population.[4] In the case of benzene, for example, the ETS-derived exposure concentration is 1.02 μg m^{-3} × 0.56 = 0.57 μg m^{-3}. The total exposure for all nonsmokers was estimated as a weighted average of the arithmetic mean exposure concentration (Table 4.1) for the passively exposed (56%) and unexposed populations (44%). For example, for benzene, the total exposure concentration is 12.6 μg m^{-3} × 0.56 + 12.0 μg m^{-3} × 0.44 = 12.3 μg m^{-3}. For those Californians exposed, the average contribution of environmental tobacco smoke to their total

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[4] The factor 0.56 is derived as the population weighted average of the fraction of nonsmokers, aged ≥7 y, exposed to ETS in any environment on a daily basis. The estimate is based on the fraction of adult nonsmokers (56%) and adolescent nonsmokers (68%) exposed to ETS daily (Wiley et al., 1991a) and on the fraction of children (38%) exposed to ETS daily. The population weighting factors are obtained from the 1990 census data for California (Bureau of the Census, 1992) which shows that 74% of Californians are aged 18 y and over. We approximated the age distribution of children and adolescents as uniform with age, so that 26% of the total population aged 0-17 y corresponds to 1.44% per year of age. We further corrected for smoking by applying the APCR data which indicates that 22% of California adults and 6% of adolescents smoke daily (Wiley et al., 1991a). Applying these data, we estimate that the percentage of Californians who are adult nonsmokers is (1-0.22) × 74% = 57.7%. Likewise, the percentage of Californians who are adolescent (age 12-17 y) nonsmokers is (1-0.06) × 1.44%/y × 6 y = 8.1%. The percentage of Californians who are children (age 7-11) is 1.44%/y × 5 y = 7.2%. Therefore, the percentage of all Californian nonsmokers who are exposed daily to ETS is estimated to be (0.577 × 56% + 0.081 × 68% + 0.072 × 38%) ÷ (0.577 + 0.081 + 0.072) = 56%.

Table 4.3. Proportion of Californians' 24-hour personal exposure to selected toxic air contaminants attributable to ETS, late 1980's.

compound	fraction of exposure from ETS for exposed nonsmokers [1]	fraction of exposure from ETS for all nonsmokers[2]
benzene	8%	5%
styrene	15%	8%
o-xylene	8%	5%
m,p-xylene	5%	3%

[1] Results apply to the nonsmoking population in California (age ≥ 7 y) who are exposed to ETS. These results are obtained by dividing the arithmetic mean, 24-hour exposure due to ETS for nonsmokers exposed to ETS (AM from Table 4.2) by the arithmetic mean, 24-hour exposure of passive smokers (Table 4.1).

[2] Results apply to the entire nonsmoking population in California, age ≥ 7 y. These results are obtained by dividing the arithmetic mean, 24-hour exposure caused by ETS for all nonsmokers (AMs from Table 4.2 weighted by 0.56) by the arithmetic mean, 24-hour total exposure (weighted average of AMs for passive (0.56) and unexposed (0.44), Table 4.1).

inhalation exposure is estimated to be 5% for m,p-xylene, 8% for o-xylene and benzene, and 15% for styrene. The estimated proportion from ETS toward the total inhalation exposure of nonsmoking Californians to these compounds are 3% for m,p-xylene, 5% for o-xylene and benzene, and 8% for styrene.

4.3. Discussion

4.3.1. Uncertainty Analysis

One of the aims of exposure assessment is to minimize the uncertainty in the knowledge of the distribution of population exposures, while accurately describing the true exposure variation. In this context, uncertainty can be considered as the lack of knowledge about possible exposure outcomes which may be diminished through further measurements (Burmaster and Anderson, 1994). There are different types of uncertainty that can be associated with exposure assessment, including the uncertainty that arises because of fluctuations in experimental measurements and those associated with the inadequate formulation of the theory behind the problem (Bevington and Robinson, 1992). Uncertainties associated with the fluctuations of measurements from experiment to experiment can be reduced by conducting more experiments. Reducing uncertainty due to model formulation is more difficult. For example, when scientists realized that the actual levels with which people come into contact differed significantly from ambient measurements, due in part to measurements taken indoors and in the personal zone, the conceptual model of human exposures was updated and refined (Ott, 1990).

Uncertainty analysis is directed at obtaining the most useful information from the data on hand without being able to conduct more experiments. Our specific application of uncertainty analysis involves determining an interval of plausible values for the target population parameters of the ETS-only distribution so that we determine what confidence we can place in our results. We believe that a dominant source of uncertainty in our exposure estimates results from the small sample sizes in the CED. We hypothesize that with an increase in sample sizes, the uncertainty associated with the exposure distribution would decrease.

As illustrated in Figure 4.2b, we employed numerical experiments to estimate uncertainty due to limited sample sizes. In this method, we numerically replicate the exposure monitoring experiments by randomly sampling from lognormal distributions having the same parameters as the actual measurements; that is, the GM and GSD derived for the passive and unexposed exposure distributions in the pooled data are used in a Monte Carlo procedure to generate many synthetic data sets. We then analyze each of these data sets to estimate the ETS-only distribution of exposure. A key point in this method is that the synthetic data sets each have only as many data points as the original pooled data. For example, only 126 points were sampled from the lognormal

formulation of the unexposed distribution for benzene since there are 126 measurement of benzene exposure for participants with unexposed status. Ten such numerical experiments were conducted for each of the measured compound — benzene, styrene, o-xylene, and m,p-xylene.

The resulting parameters distribution for exposures due to ETS only are summarized in Figure 4.4 (Figure 4.4a shows uncertainty in the geometric mean and Figure 4.4b shows uncertainty on the geometric standard deviation) and Figure 4.5 (arithmetic mean). Figure 4.5 shows, for example, that for benzene, nine of the ten numerical experiments generated arithmetic means of the ETS contribution to exposure in the range 0.20 to 7 $\mu g\ m^{-3}$. The analogous results for the other compounds are as follows: styrene — 0.06 to 0.9 $\mu g\ m^{-3}$; o-xylene — 0.14 to 2 $\mu g\ m^{-3}$; and m,p-xylene — 0.4 to 27 $\mu g\ m^{-3}$. One of ten simulations for each compound generated a best estimate of zero exposure due to ETS, which occurred when the mean of the passive exposure distribution was higher than that of the unexposed distribution. The 90% confidence bounds on the mean exposures for each compound are estimated by taking the square root of the ratio of the highest to lowest of the nonzero values as a multiplicative error factor. So, for example, for benzene, the estimated uncertainty due to limited sample size is $\times/\div$ 6, obtained as $(7/0.20)^{1/2}$. For all four compounds the multiplicative error estimates in the arithmetic mean so determined is 4 for styrene and o-xylene, 6 for benzene, and 8 for m,p-xylene. We note that the ETS scale factors determined by the four different compounds all varied within about a factor of four, indicating that the errors associated with using different reference compounds are no larger than the errors resulting from limited study size.

4.3.2. Geographical Extrapolation

Inferences about the entire California population from the CED involve some degree of uncertainty because the measurements are based on a limited sample population, rather than the whole population. Due to the methodological features of the six studies' designs, the combined sample population is not directly representative of the target population. For example, in our pooled data set, part of LA county is represented, the SF Bay area is represented with a sample population in Pittsburg/Antioch, the Sacramento Valley is represented with a study of the city of Woodland. The Eastern, South Eastern, and most Northern Californian counties are not represented at all. Also, smoking habits may be different in the sampled portions of the state as compared to the rest of California.

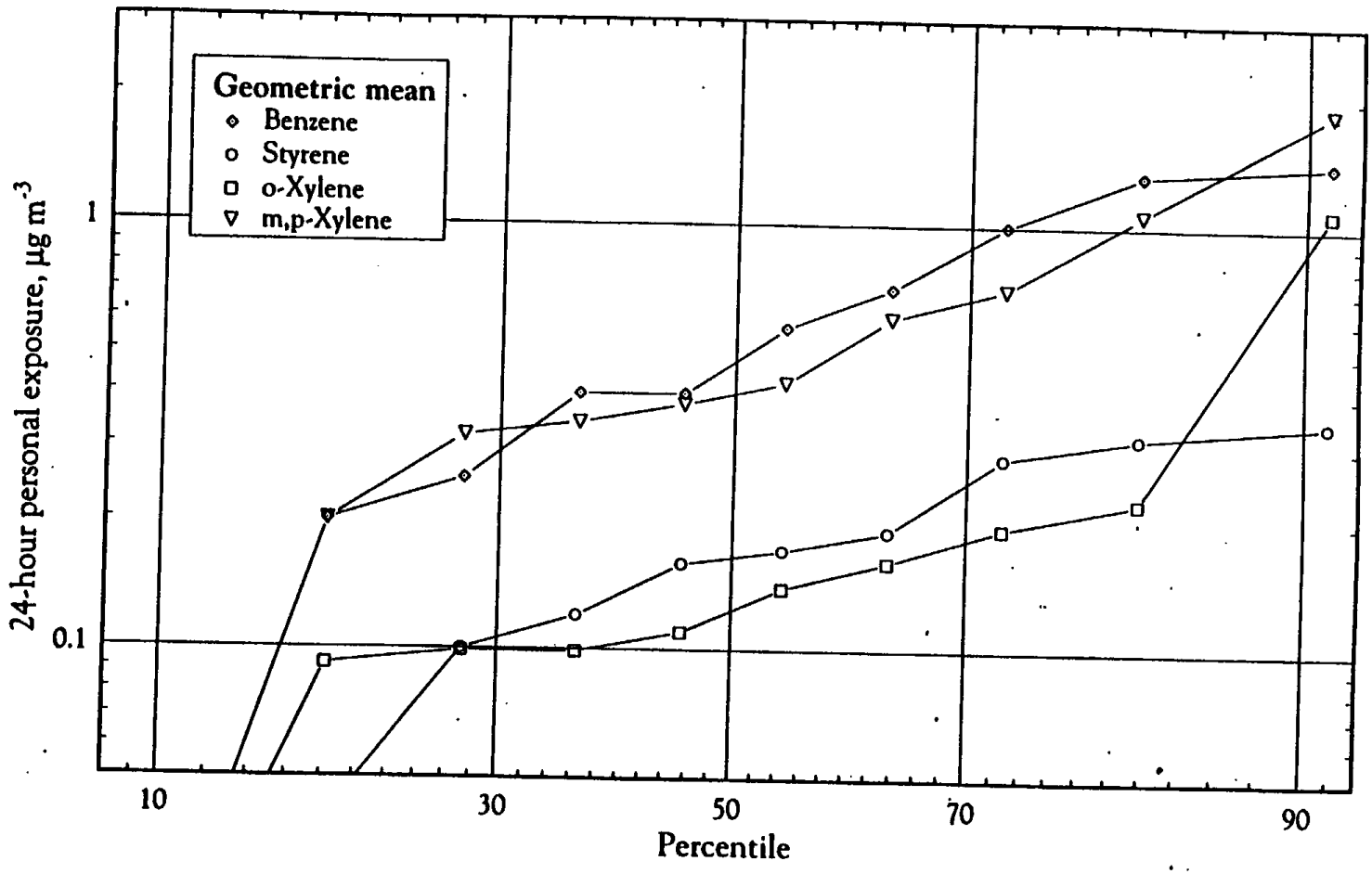

Figure 4.4a Lognormal-probability plot indicating the uncertainty in the predicted geometric mean of 24-h average exposure concentration for Californians (mid to late 1980's) from ETS for benzene, styrene, o-xylene, and m,p-xylene.

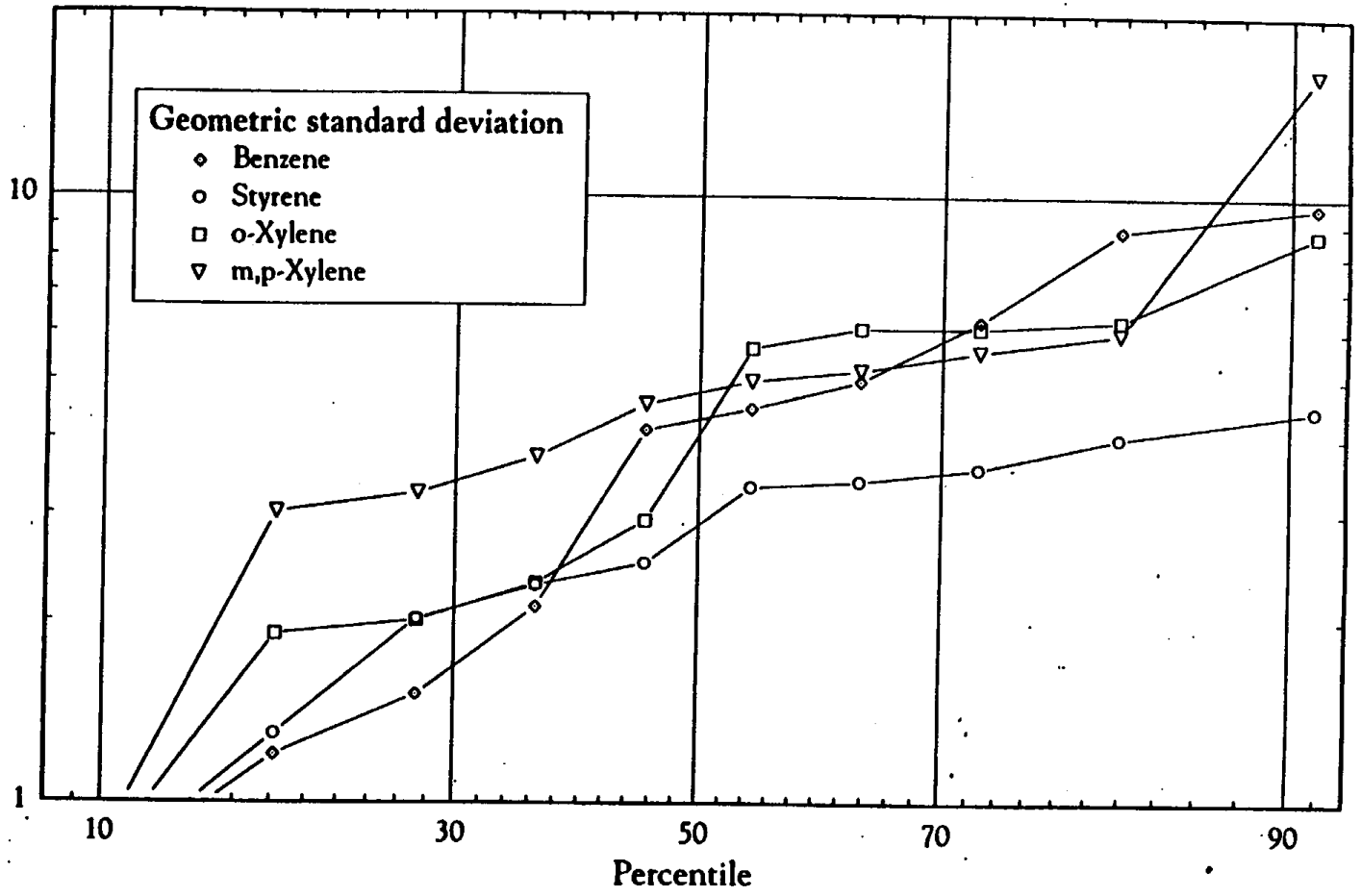

Figure 4.4b Lognormal-probability plot indicating the uncertainty in the predicted geometric standard deviation of 24-h average exposure concentration for Californians (mid to late 1980's) from ETS for benzene, styrene, o-xylene, and m,p-xylene.

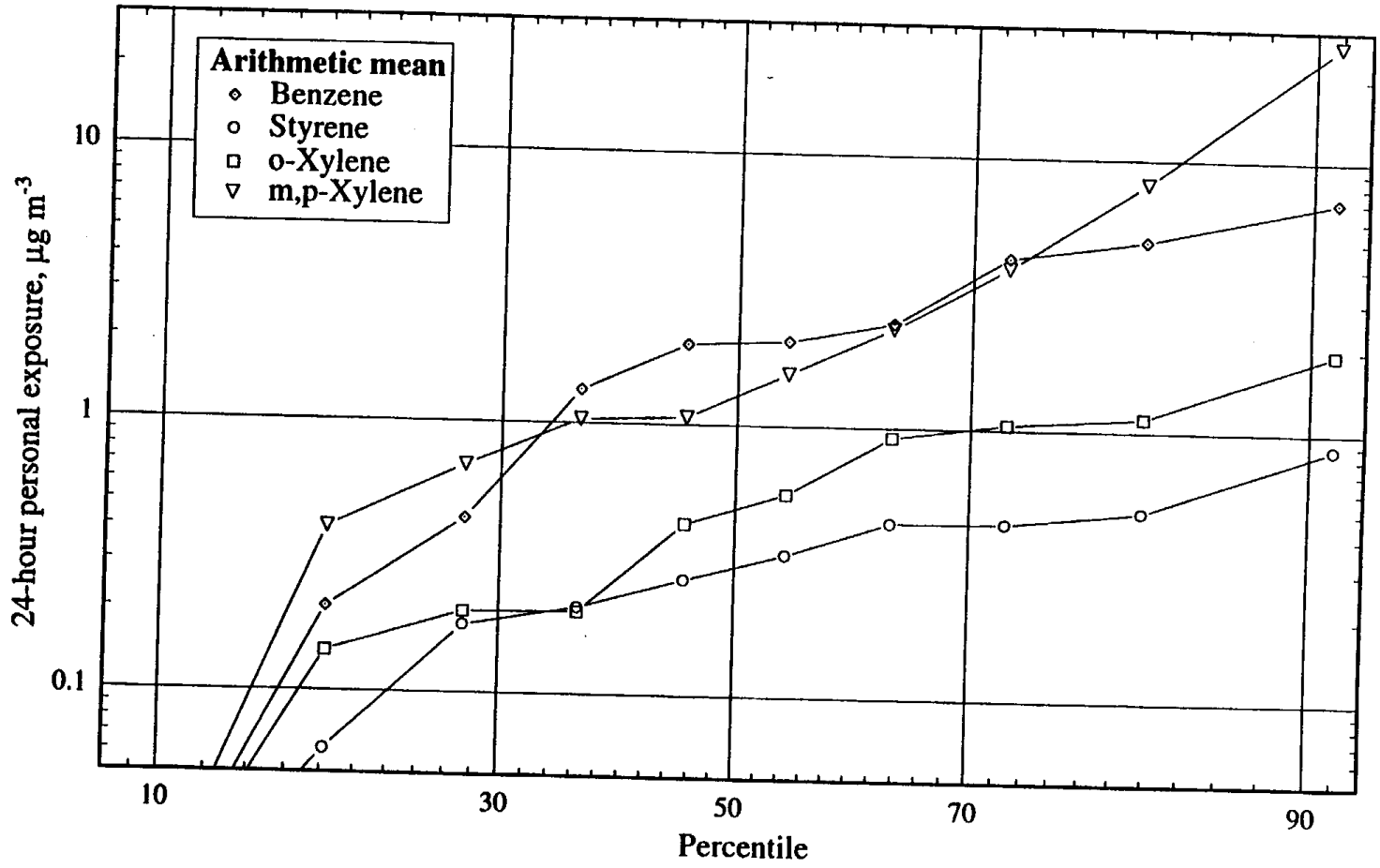

Figure 4.5 Lognormal-probability plot indicating the uncertainty in the predicted arithmetic mean of 24-h average exposure concentration for Californians (mid to late 1980's) from ETS for benzene, styrene, o-xylene, and m,p-xylene.

Tables 4.4 and 4.5 enumerate the similarities and differences between the pooled data and the target population by exploring in detail the characteristics of the pooled data set and comparing them to target population characteristics. Pooled data characteristics were determined from the participant/household characteristics section of the CED. The target population characteristics were determined from the Californian census data (Bureau of the Census, 1992) and from the 1993 Air Resources Board emission inventory (Air Resources Board, 1995).

4.3.2.1 Demographics for California and for each Air Basin

The three areas of concern in this project represent the three largest air basins in California; the demographics for these regions are presented in Table 4.4. Together, these areas contain almost 70% of the total Californian population (although covering less than 20% of the total land area). South Coast has the largest population of the three, with over 12 million people.

The gender and age composition of the three areas is similar to that of the entire state. The split between male and female in each area is almost exactly 50-50, as is also true for California as a whole. The age distribution for each area is also very similar to that of the state. The largest group in each case is the 25 to 44 age group, which accounts for about a third of each population.

The racial compositions of the air basins are less similar. None of the basins matches the composition of California as a whole. The white populations are the largest in each area, as is the case for California as a whole. South Coast has the largest Hispanic population, which comprises a third of the total population. This figure, while similar to that of the state, is twice the percentage of Hispanics in the San Francisco basin, and three times that of Sacramento. The black populations are more constant, reflecting the state-wide average. The largest Asian population is in San Francisco; this population is 60% larger than those of the other two regions, and is twice the state-wide average.

4.3.2.2. Demographics for Data in CED

Studies 1, 3, and 6 each contain sampling weights which allow the data to be extrapolated to the metropolitan area where each study was conducted. While the extrapolated areas are not as large as the surrounding air basin, the demographics of these areas do roughly parallel those of the air basins, with a few exceptions, as presented in Table 4.5. The data from Study 1, then, was assumed to be representative of the South Coast region; Study 3, of the San Francisco Bay Area; and Study 6, of the Sacramento Valley area.

Table 4.4. Demographic data for the State of California and for the three air basins used to represent the Californian population. [1]

category	CA total		South Coast air basin		San Francisco Bay Area air basin		Sacramento Valley air basin		total of represented air basins
general									
total population	29,760,021		12,801,260		5,885,077		1,972,238		20,658,575
% of CA population	100.0%		43.0%		19.8%		6.6%		
% of represented studies			62.0%		28.5%		9.5%		
gender									
males	14,897,627	50.1%	6,401,283	50.0%	2,929,583	49.8%	971,004	49.2%	49.9%
females	14,862,394	49.9%	6,399,977	50.0%	2,955,494	50.2%	1,001,234	50.8%	50.1%
race/ethnicity									
Hispanic	7,687,938	25.8%	4,260,797	33.3%	906,369	15.4%	226,583	11.5%	26.1%
White	17,029,126	57.2%	6,241,681	48.8%	3,563,000	60.5%	1,465,039	74.3%	54.6%
Black	2,092,446	7.0%	1,052,551	8.2%	503,947	8.6%	116,744	5.9%	8.1%
American Indian [2]	184,065	0.6%	50,637	0.4%	29,255	0.5%	23,877	1.2%	0.5%
Asian	2,710,353	9.1%	1,168,704	9.1%	872,386	14.8%	137,154	7.0%	10.5%
other	56,093	0.2%	26,890	0.2%	10,120	0.2%	2,841	0.1%	0.2%
geography									
area (km^2)	403,970		16,964		14,217		38,711		69,893
% of total CA area	100%		4.2%		3.5%		9.6%		17.3%
people km^{-2}	74		755		414		51		296
age (y)									
0-17	7,750,725	26.0%	3,390,500	26.5%	1,393,298	23.7%	522,332	26.5%	25.7%
18-24	3,412,257	11.5%	1,538,615	12.0%	601,645	10.2%	211,377	10.7%	11.4%
25-44	10,325,692	34.7%	4,456,905	34.8%	2,147,795	36.5%	658,224	33.4%	35.2%
45-59	4,036,476	13.6%	1,707,336	13.3%	873,584	14.8%	272,412	13.8%	13.8%
60+	4,234,871	14.2%	1,707,904	13.3%	868,755	14.8%	307,893	15.6%	14.0%

[1] sources: Air Resources Board (1995); Bureau of the Census (1992).
[2] Also includes Eskimo and Aleut.

Table 4.5. Demographic data for the State of California and for the exposure studies used to represent the Californian population. [1]

category	CA total		Study 1 Los Angeles Co.		Study 3 Pittsburgh/Antioch		Study 6 Woodland		total population the 3 studies
general									
total population	29,760,021		359,493		90,696		31,470		481,659
% of CA population	100.0%		1.2%		0.3%		0.1%		
% of represented studies			74.6%		18.8%		6.5%		
gender									
males	14,897,627	50.1%	168,508	49.6%	38,994	43.0%	16,269	51.7%	46.5%
females	14,862,394	49.9%	190,985	53.1%	51,702	57.0%	15,201	48.3%	53.5%
race/ethnicity									
Hispanic	7,687,938	25.8%	54,236	15.1%	4,918	5.4%	N/A		
White	17,029,126	57.2%	240,794	67.0%	70,525	77.8%	N/A		
Black	2,092,446	7.0%	23,797	6.6%	6,187	6.8%	N/A		
American Indian [2]	184,065	0.6%					N/A		
Asian	2,710,353	9.1%	35,499	9.9%	6,338	7.0%	N/A		
other	56,093	0.2%	5,167	1.4%	2,728	3.0%	N/A		
age (y)									
0-17	7,750,725	26.0%	63,721	17.7%	23,705	26.1%	4,740	15.1%	19.1%
18-24	3,412,257	11.5%	44,562	12.4%	6,037	6.7%	3,822	12.1%	11.3%
25-44	10,325,692	34.7%	143,199	39.8%	45,438	50.1%	17,149	54.5%	42.7%
45-59	4,036,476	13.6%	62,503	17.4%	11,905	13.1%	2,488	7.9%	16.0%
60+	4,234,871	14.2%	45,508	12.7%	3,611	4.0%	3,271	10.4%	10.9%

[1] sources: Air Resources Board (1995); Bureau of the Census (1992).

[2] Also includes Eskimo and Aleut.

For each of the three studies, the population density is much larger than that of the corresponding air basin. While the studies are extrapolated to the local metropolitan area, the air basins contain a much larger area, much of which is far less densely populated than the study areas. A comparison of the demographics of the study areas and the air basins is, therefore, a much more telling indicator of their similarities and differences than population density.

The gender make-up of each study was relatively close to 50-50, as was that of the air basins. Study 3 showed the largest deviation from the air basin data, although the difference was still small (7%). The age distribution was also similar, although each study had a larger percentage of 25–44 year-olds than their corresponding air basins. This discrepancy may be partially explained by the fact that this group was most likely to participate in each study. This factor might also explain the under-representation of people younger than 18 and older than 60.

The one area where the studies do differ markedly from the air basins they represent is in racial composition. Study 6 did not ask the race of its participants, so this information was only available for Studies 1 and 3. In both of these studies, the white population was greatly over-represented. Since whites are the largest racial/ethnic group, this had a large effect on the percentages of all other races. For example, the Hispanic population of Study 1 was less than half of the corresponding percentage for the South Coast air basin. Likewise, the Asian population for Study 3 was also less than half of the percentage for the San Francisco air basin. The black populations for both Studies 1 and 3 also slightly under-represent the corresponding air basins.

It is possible that the differences in racial composition between the studies and the air basins they are intended to represent may have affected our results. Research has shown that there are differences in smoking prevalence among different racial groups. For example, many recent studies have noted an increased prevalence of smoking among black Americans, compared to the population average. Similar studies have also noted that the intensity of smoking (i.e. the number of cigarettes smoked per day) among blacks is less than average. (Kabat et al., 1991; Satariano and Swanson, 1988) The U.S. Department of Health and Human Services (1990) estimated that both prevalence and intensity of smoking varied greatly among Asian Americans; smoking is low in some Asian-American groups and extremely high in others. Hispanics in the U.S. have been estimated to have a reduced smoking prevalence and intensity than the average (Palinkas et al., 1993). The variety of trends reported above makes it difficult to quantify how the number of active smokers and the intensity of smoking in the CED studies is affected by the under-representation of minorities.

4.3.3. Assessment Limitations

In addition to limited sample size and geographical extrapolation, other factors can contribute to uncertainty in exposure estimates by the method we employed. Statistical bias may be introduced

in the exposure estimate due to misclassification of ETS exposure status among the exposed and the unexposed groups. There is also some uncertainty in the exposure measurements themselves: readings can vary according to personal sampling device placement and low concentrations may be difficult to quantify near the detection limit.

Implicit in our method is the assumption that the passive and unexposed populations are similar in all characteristics other than cigarette smoke exposure. In our comparison of demographics for California, the air basins, and the CED studies, we determined that most population characteristics, save race, were similar. Comparison of demographics in the CED showed that many participant characteristics were similar for the passive and unexposed populations: respectively 52% and 49% males; 68% and 67% whites; and 56% and 42% in the 25–44 year-old age group.

A concern may arise about the contribution of mainstream smoke to ETS exposure because the emission factors from Daisey et al. were measured by emitting only sidestream smoke into an experimental chamber. The exposure measurements included in the CED do implicitly account for exhaled MS exposure. Furthermore, since the Daisey et al. emission factors do not include exhaled MS for any ETS species, to a first-order approximation, the errors from excluding exhaled MS from ETS cancel. Limited data on MS contributions to ETS suggest that the proportion is small: in ETS, approximately 15% of particulate matter, 13% of CO, and 9% of nicotine is exhaled MS (Baker and Proctor, 1990). Furthermore, Martin et al. (1997) measured emission factors using human smokers; their data include exhaled MS plus sidestream and do not differ markedly from Daisey et al.'s results (Table 3.1). Exposure estimates using Daisey et al. emission factors are in good agreement with those using data from Martin et al. (Table 4.2).

Pollutant-surface interactions, which are not explicitly incorporated into our estimates, can also influence the results. The test chamber surfaces used to measure emission factors were stainless steel, unlike real indoor surfaces such as walls and carpets. Recent research indicates that nicotine, an ETS component that interacts strongly with indoor surfaces, may still be a suitable marker for ETS particles in indoor environments in which smoking is habitual (Van Loy et al., 1998). Those results add confidence to the assumption that exposures to individual compounds in ETS scale in proportion to emission factors.

4.3.4. Comparison with Prior Studies

Heavner et al. (1995) present apportionment results from personal exposures measured for nonsmoking women living in smoking homes. They estimate that the median percentage attributable to ETS for benzene and styrene is 13.2% and 12.6%, respectively. Our results for nonsmokers exposed to ETS — an average contribution of 8% for benzene and 15% for styrene — are consistent with those of Heavner et al.

Wallace (1989a) estimated that mainstream smoke inhaled by smokers constitutes 50% of the total US population burden of benzene exposure and that ETS contributes 5% of the total, or 10% of that portion other than mainstream smoke inhalation. Our estimate for all nonsmokers for benzene, 5%, is of the same magnitude, but somewhat lower than the 10% estimate from Wallace. Note that our estimate does not include the ETS exposure of smokers as do those of Wallace; we expect our fraction would increase if smokers were included. Also, the Wallace estimate is based on a mixture of California and New Jersey TEAM data.

4.3.5. Comparison of ETS-Only Exposures to Ambient and Indoor Air Concentrations

Regulatory agencies rely heavily on quantitative assessments of environmental health risks as the scientific basis for decisions about how best to protect public health. While significant advances have been made in providing the information needed to accurately assess risk, namely in the identification of some potentially toxic compounds and their levels in ambient air, research is still needed in the quantification of indoor source emissions and personal exposure levels (Möller et al., 1994). Much information has been gathered by measuring ambient levels of TACs for the purpose of understanding the concentrations to which the public is exposed when breathing outdoor air. Similar information (albeit more sparse) is available for indoor air concentrations. Our research was specifically aimed at providing more information on personal exposure resulting from a specific indoor emission source: ETS.

Measurements of pollutant levels in indoor and ambient air do not give a direct picture of personal exposure; rather, indoor and ambient concentrations can be used to indirectly characterize exposure when combined with information on the time of contact with the pollutants (U.S. Environmental Protection Agency, 1992b). Assuming that the concentration in the bulk medium is the same as the exposure concentration is a source of potential error. Generally, the closer the concentration can be measured to the point of contact between a human and the pollutant, the less uncertainty there is in the exposure assessment (U.S. Environmental Protection Agency, 1992b). One of the major conclusions of the TEAM studies was that personal exposure concentrations are higher than outdoor air concentrations (Wallace, 1987). A study by Michael et al. (1990) showed that, in fact, concentrations measured at residential-indoor, residential-outdoor, and centralized locations can disagree substantially. These particular investigations illustrate that, in many situations, a centrally-located monitoring site cannot be used to predict outdoor residential concentrations, which in turn cannot be used to predict the concentrations in the residence, which ultimately may not be representative of the personal exposure concentration.

Although indoor and ambient levels are not always good representations of personal exposure, we compare exposure concentrations to bulk media concentrations in Table 4.6 to try

and put our results into perspective, and to bridge the gap between what is known about ambient and indoor air concentrations and what is known about personal exposure concentrations. In Table 4.6, we compare our ETS-only 24-hour exposure results to reported measurements of ambient and indoor air toxicant levels. To be specific, the personal exposure concentrations presented in Table 4.6 are our estimates of the average TAC exposure for the entire Californian nonsmoking population (age $\geq$7 y) that can be attributed to ETS. Comparing these exposures to reported California ambient air measurements,[5] we find that for three of the compounds (acetaldehyde, 1,3-butadiene, and formaldehyde) the exposure concentration associated solely with ETS is approximately the same as the reported concentrations in outdoor air. Several other compounds (benzene, 2-butanone, ethylbenzene, styrene, toluene, and o-xylene) have ETS contributions to exposure that exceed 5% of the reported ambient levels in California. The results of these comparisons indicate that to the extent that outdoor air concentrations contribute to exposure, a significant fractional reduction in the ambient level would be needed to gain the same reduction in exposure as could be achieved by substantially reducing ETS exposure.

For compounds measured both indoors and outdoors, indoor air concentrations as reported in Table 4.6 are typically higher. Many of the indoor air values in Table 4.6 are based on measurements from buildings that were suspected to have elevated indoor concentrations of pollutants.[6] The average ETS-only contribution to exposure is typically small when compared with the average reported indoor air concentrations, ranging from several percent for toluene to as much as 20% for styrene.

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[5]Many of the ambient measurements were made in areas of industrial activity or urban high-traffic regions; also, some of these measurements were made 10-20 years ago, when fewer controls existed on outdoor sources.

[6]Some of these indoor air values may be biased because the buildings in which measurements were made were selected for some specific purpose related to finding high indoor concentrations; for example, to determine levels in buildings where there had been complaints or knowledge of the materials used (Daisey, 1996; Shah and Singh, 1988).

Table 4.6. Comparison between Californians' average exposure to toxic air contaminants from ETS, and concentrations measured in indoor and ambient air ($\mu g\ m^{-3}$).

	ETS only exposure concentration	*indoor air concentration*		*outdoor air concentration*	
compound	this study[1]	U.S.[2]	worldwide[3]	U.S.[4, 5]	California[5,6]
acetaldehyde	6.1-8.4			2.7	3.3 (ND-25)
acetonitrile	3.9				
acrylonitrile	0.27			0.2	
benzene	0.57	17	8	5.1	8.3 (ND-38)
1,3-butadiene	0.42-1.3			0.4	0.8 (ND-5.3)
2-butanone (MEK)	0.78	27	4	ND	2.5 (ND-13)
o-cresol	0.10			1.5	
m,p-cresol	0.23			ND[7] (0.5 – 20)[8]	
ethyl acrylate[9]	<0.008			ND	
ethylbenzene	0.27-0.36	13	5	1.1	4.3 (ND-17)
formaldehyde	3.6-4.6	61		3.3	2.6 (ND-25)
N-nitroso-dimethylamine	0.0016		<1	0.04	
phenol	0.78		9[10]	17	
styrene	0.20		1 – <5	0.6	1.5 (ND-12)
toluene	1.7-1.8	28	37	8.6	16 (1.1-180)
o-xylene	0.43		6	2.2	3.3 (ND-27)
m,p-xylene	0.55		18	4.2 – 4.3	12 (ND-100)

[1] Arithmetic mean (AM) for nonsmoking Californian population aged ≥ 7 y, calculated by multiplying the AM from Table 4.2 by 0.56, the fraction reporting exposure to ETS at some time during a day, based on our analysis of APCR and CAP data.
[2] Shah and Singh (1988); arithmetic mean.
[3] Brown et al. (1994); studies from Netherlands, Germany, Italy, U.S.; weighted-average geometric mean.
[4] Kelly et al. (1994); median.
[5] ( ) indicates range.
[6] Redgrave (1996); data from California's Ambient Toxics Monitoring Network, for the years 1990-91; arithmetic mean.
[7] Median for m-cresol.
[8] Range for p-cresol.
[9] Emission factor measured below lower limit of detection; thus, only an upper-bound estimate of the ETS-caused exposure was determined.
[10] Based on measurements in only one building.
ND = not detected.

In Appendix A, we investigate the indoor air levels of TACs that result from smoking. Table A.1 summarizes measurements of TACs in smoking environments; the levels reported there are comparable to the indoor air concentrations in Table 4.6. This supports the observation that the indoor air measurements in Table 4.6 may not truly reflect average indoor air levels, but rather reflect levels in those environments that have significant sources of TACs. In fact, we can estimate the proportion of indoor air levels due to ETS for smoking environments using the data in Table A.1 and Figure A.1 (see Appendix A). The right-hand bar of Figure A.1 shows, for residences in which smoking occurs, estimated mean concentrations of air toxics attributable to ETS. Table A.1 reports the total mean concentration of air toxics in these same residences. Comparing the results suggests that, on average, ETS accounts for 4-30% of indoor air levels of benzene, ethylbenzene, styrene, o-xylene, and m,p-xylene in California residences in which smoking occurs. [7]

[7] The estimates are derived by dividing the "measurement" result plotted in Figure A.1 by the average AM for smoking environments from Studies 1-6 as reported in Table A.1. For benzene, the result is 3.6/11.5 = 31%; for ethylbenzene, the result is 0.9/5.9 = 15%; for styrene, the result is 0.1/2.4 = 4%; for o-xylene, the result is 0.8/6.7 = 12%; and for m,p-xylene, the result is 3.1/18 = 17%.

5. Phase II: An Assessment for late-1980's Based on Microenvironmental Exposure Modeling

The assessment conducted in this phase of the research is based on constructing a probability distribution of exposures from survey data of activity patterns combined with estimated concentrations of ETS constituents in microenvironments. A simulation typically consists of thousands of iterations. In each iteration, the exposure of a nonsmoker is computed by summing over all exposure activities the product of two terms: (a) time exposed to ETS in a given microenvironment and (b) the concentration of an ETS constituent in that environment. The result is a probability distribution of total daily exposures for the exposed nonsmoking population. Information is also preserved during the simulations about the level of exposure in different microenvironments. The primary simulations were conducted for exposure to benzene from ETS. Exposure to other toxic air contaminants in ETS were determined by scaling with the ratio of emission factors. Simulations were conducted separately for adults, adolescents (12-17 y), and children (0-11 y). Four distinct scenarios were executed to establish bounds on the range of probable outcomes. The work described in this section focused on conditions in California in the late 1980's. In §6, the same methods are applied to predict the distribution of daily exposures of nonsmoking Californians in the late 1990's.

5.1. Methods

5.1.1 Computing Microenvironmental Concentrations

We applied two independent methods for determining microenvironmental concentrations of ETS constituents. One method utilizes completely-mixed room (CMR) models, which are based on the principle of mass conservation. We applied this method to five microenvironments: residential, occupational, schools, retail/other indoor, and transportation. The second method relies on published measurements of ETS tracer concentrations, specifically nicotine and respirable suspended particles (RSP). This tracer method was applied in all microenvironments except transportation for which suitable data do not exist. For some microenvironments such as restaurants, bars and nightclubs, we judged that adequate data on parameters such as ventilation rates or smoking intensity do not exist to support a CMR model calculation and so only applied the tracer method. Table 5.1 summarizes the methods used for evaluating microenvironmental concentrations for each of the four scenarios.

Table 5.1. Four scenarios used to estimate the toxic air contaminant exposures for the nonsmoking California population.

scenario	input parameter level	CMR model [1]	tracer method
TL	low exposure	transportation	residential occupational school retail/other restaurant bar/nightclub residential guest
TM	mid-range	transportation	residential occupational school retail/other restaurant bar/nightclub residential guest
CM	mid-range	residential occupational school retail/other transportation	restaurant (TM) bar/nightclub (TM) residential guest (TM)
TH	high exposure	transportation	residential occupational school retail/other restaurant bar/nightclub residential guest

[1] CMR model = completely mixed room model

5.1.1.1. Completely-Mixed Room (CMR) Model

The basic version of this model describes a building or other microenvironment as a single, well-mixed zone of volume, V. Pollutant concentrations are assumed to be uniform throughout the space. Pollutants may be introduced into the space by direct indoor emissions at a rate E ($\mu g\ h^{-1}$). Air flow passes through the building at a rate Q ($m^3\ h^{-1}$). Air flow may be due to infiltration, natural ventilation, or mechanical ventilation (ASHRAE, 1993). We use Q to represent the sum of outdoor air supply from any or all of these modes and we refer to Q as the ventilation rate. Normalizing the ventilation rate by the building volume yields the air-exchange rate, $\lambda = Q/V$ (air changes per hour (ACH), h^{-1}). We assume that the presence of environmental tobacco smoke in outdoor air is negligible. We also assume that removal of ETS constituents from indoor air occurs only because of ventilation. Pollutants are removed by ventilation at a rate $Q \times C$ ($\mu g\ h^{-1}$), where C denotes the species concentration indoors contributed by smoking. The time-dependent equation expressing the conservation of species mass in indoor air is then

$$\frac{d(CV)}{dt} = E - Q\,C \qquad (5.1)$$

To apply the model in any given microenvironment, approximations must be made to obtain a form that provides the most accurate estimate of concentrations predictable from available data. Thus, for example, in addition to assuming that indoor air is well mixed, the form of equation (5.1) does not account for pollutant-surface interactions (such as sorption-desorption). These approximations introduce some uncertainty into the assessment that cannot be avoided since data are lacking that would permit either assumption to be meaningfully relaxed. In each microenvironment we use an equation based on equation (5.1) to estimate the time-averaged concentration during exposure, rather than the time-dependent concentration. This step requires that we make approximations about the temporal pattern of smoking and ventilation rate.

5.1.1.1.1 Application to Residences. For estimating ETS constituent concentrations in residences using the CMR model, we derived the following equation, based on equation (5.1):

$$C_{avg} = \frac{n\,N_r\,e}{\lambda_{avg}\,V} \qquad (5.2)$$

where

n = number of smokers in the building (-)

N_r = rate at which cigarettes are smoked inside residence (cig h^{-1} per smoker)

e = emission factor for the air toxicant in ETS ($\mu g\ cig^{-1}$), and

λ_{avg} = time-averaged air-exchange rate (ACH, h^{-1})

Appendix D presents a derivation of equation (5.2). The use of equation (5.2), including a discussion of the data sources, is described in §5.1.3.

Model equation (5.2) is based on the approximation that the entire house can be represented as a single well-mixed zone and that steady-state conditions prevail. This approach requires only one air-exchange rate for each residence instead of detailed information about flows between rooms (information which is lacking in any case). When estimating average exposure over the course of several hours, there is empirical support for making this approximation. Experimental data show that long-term average RSP concentrations measured in different rooms of the house are relatively consistent (Ju and Spengler, 1981). Also, a behavioral study has shown that the distribution of cigarette smoking throughout the day tends to be regular (Shiffman, 1996), suggesting that ETS is introduced into indoor air at regular intervals during the time a smoker is at home and awake.

5.1.1.1.2 Application to Occupational, School, and Retail/Other Indoor Settings. The form of equation (5.2) is not convenient for predicting ETS concentrations in occupational settings. Data on the probability distribution of smokers in a given building and on the distribution of volumes of public buildings are not readily available. Also, for workplaces, airflow rates in terms of ventilation rate per occupant are more practical to use than building volumes and air-exchange rates. So, as an alternative, we based our calculations on a modified form of equation (5.2). Dividing both the numerator and denominator by Γ, the total number of occupants in the building, produces this result:

$$C_{avg} = \frac{n\, N_o\, e\, (1/\Gamma)}{Q_{avg}\, (1/\Gamma)} = \frac{f\, N_o\, e}{q_{avg}} \qquad (5.3)$$

where $f = n/\Gamma$ is the fraction of the occupants that smoke, N_o is the rate at which cigarettes are smoked inside occupational settings (cig smoker^{-1} h^{-1}), and $q_{avg} = Q_{avg}/\Gamma$ is the time-averaged outdoor-air ventilation rate per occupant. Application of equation (5.3) and a discussion of data sources, is presented in §5.1.3.

Although children and adolescents spend a significant portion of their time at schools, this microenvironment is not expected to be a large source of exposure to ETS. Smoking in class rooms has been unacceptable for several decades. The activity pattern data reveal (see Figure 3.2) that of the self-reported exposure time only 5% is in school for adolescents and only 3% for children (this microenvironment includes child care). The modeling approach for predicting concentrations in schools is the same as for occupational settings, i.e. equation (5.3) is used.

For the retail/other group of microenvironments, equation (5.3) was judged to provide the best basis for estimating ETS concentrations.

5.1.1.1.3 Application to Vehicles For motor vehicles, we used a modified form of equation (5.2), replacing the air-exchange rate and volume with the ventilation rate:

$$C_{avg} = \frac{n\, N_t\, e}{Q_{avg}} \qquad (5.4)$$

where N_t is the rate at which cigarettes are smoked in the vehicle. Application of equation (5.4) is discussed, along with the input data sources, in §5.1.3.

5.1.1.2 Tracer Method

Because it is neither practical, nor possible, to measure the full range of air pollutants associated with ETS, tracer pollutants are measured as ETS indicators. While a variety of tracers have been used for ETS, no single compound is ideal. An ideal tracer, as described by the National Research Council (NRC, 1986), should (1) be sufficiently unique to tobacco smoke that other sources are negligible in comparison; (2) have similar emission rates for a variety of cigarette types; (3) exist in sufficient quantities that it can be measured at detectable concentrations, especially at low smoking rates; and (4) be present in a consistent ratio to the pollutants of interest (in this case, TACs).

We considered using several different ETS tracers in our modeling-based assessment: respirable suspended particles (RSP), nicotine, carbon monoxide (CO), 3-ethenylpyridine, pyridine, and pyrrole. Many researchers have used RSP as a tracer for ETS because the combustion of tobacco smoke emits large quantities of particulate matter (Collett et al., 1992; Drake and Johnson, 1990; Spengler et al., 1981). However, there are many sources of particles in indoor environments, and so the RSP measurement is not a specific to ETS. Nevertheless, RSP can serve as a tracer if measurements in nonsmoking environments are used as a control.

Nicotine is the major alkaloid in tobacco. A large number of published studies measuring ETS constituents have used nicotine as a tracer. Because nicotine is derived solely from tobacco smoke, its presence is a very strong indicator of the presence of environmental tobacco smoke in a microenvironment. Nicotine's use as an ETS marker has been criticized because it is a semivolatile compound which interacts strongly with indoor surfaces and exhibits different dynamic behavior than other ETS constituents (Nelson et al., 1992). However, field studies have shown a good correlation between RSP and nicotine (Leaderer and Hammond, 1991). Furthermore, recent laboratory and modeling studies by our research group at the University of California suggest that in environments where smoking is habitual, the average nicotine levels in air may reflect accurately the ETS concentrations (Van Loy et al., 1997 and 1998).

Smoking also emits carbon monoxide (NRC, 1986) and CO has been used in some studies as a tracer of ETS (Leaderer et al., 1984; Muramatsu et al., 1984). However, because of small total fuel consumption, tobacco smoking is a relatively weak source of CO in comparison with

other sources such as unvented space heating and motor vehicles. Consequently, it is difficult under field conditions to use CO as a tracer of ETS and we excluded it in this work.

Other gas-phase ETS markers include 3-ethenylpyridine, pyridine, and pyrrole. Data on ETS emission factors exist for these compounds (Hodgson et al., 1996). These species are considered superior to nicotine by some investigators because they appear to interact less strongly with indoor surfaces (Eatough et al., 1989; Nelson et al., 1992). However, these markers have been used in very few field studies of ETS levels and so did not provide useful data for our assessment.

Consequently, in this study, we used nicotine measurements, when available, as the primary ETS marker compound. Measurements of particulate matter (RSP or $PM_{2.5}$) were used as a secondary marker when nicotine data were unavailable. Corrections for nonsmoking sources of particulate matter were made in each case to avoid bias.

Our primary ETS exposure simulations were conducted for benzene, and so we converted the measured particulate matter or nicotine levels to an ETS contribution to benzene according to these expressions

$$C_{benzene} = 0.050\ C_{PM} \tag{5.5}$$

$$C_{benzene} = 0.44\ C_{nicotine} \tag{5.6}$$

In these equations, C_i represents the contribution of environmental tobacco smoke to the indoor concentration of species i. The constant in equation (5.5) derives from the ratio of ETS emission factors for benzene (406 $\mu g\ cig^{-1}$) to $PM_{2.5}$ (8100 $\mu g\ cig^{-1}$) measured by Daisey et al. (1994 and 1998). It is justified for ETS to use a measurement of particulate matter smaller than 2.5 μm as a surrogate for RSP because almost no particle mass in tobacco smoke is larger than 2.5 μm (Nazaroff et al., 1993). The constant 0.44 in equation (5.6) is obtained as the ratio of ETS emission factors for benzene to nicotine (919 $\mu g\ cig^{-1}$) reported by Daisey et al. (1994 and 1998). Note that the ratio of emission factors for $PM_{2.5}$ and nicotine (8100/919 = 8.8) from laboratory emission tests by Daisey et al. (1998) agrees well with reported slope of 9.8 for RSP vs. nicotine measured in 47 homes with smokers (Leaderer and Hammond, 1991).

Application of the emission factors reported by Martin et al. (1997) would yield smaller constants in each case. Reported emission factors of 280 $\mu g\ cig^{-1}$ for benzene, 1585 $\mu g\ cig^{-1}$ for nicotine, and 13.7 $mg\ cig^{-1}$ for RSP would produce a coefficient of 0.021 in equation (5.5) and 0.18 in equation (5.6). These coefficients are only 40-42% of those determined from the Daisey et al. emission factors. If the emission factors of Martin et al. were to be consistently used in the tracer method in this study, the predictions of TAC exposures would be smaller, but not, on average, by more than a factor of 2.5. The lesser difference is a consequence of the fact that the

ratio of emission factors (Daisey et al. to Martin et al.) is relatively high for benzene as compared to most species (see Table 3.1).

Our review of the literature revealed suitable data on the concentrations of ETS tracers in six microenvironments. For four microenvironments — residential, occupational, restaurant, and residential guest — many data are available of generally good quality. For the bars and nightclubs and retail/other settings, some tracer data are available, but not of high quality. For five of these six settings (all except retail/other), adequate data have been published to justify the selection of low-, medium-, and high-range concentration distributions. The parameters that we selected are summarized in Table 5.2 and described in detail in §5.1.3.

5.1.2 Monte-Carlo Simulation Method

The central analysis in Phase II consists of predicting the probability distribution function for 24-hour exposure of Californian nonsmokers to benzene generated by tobacco smoking. For each of four scenarios and each of three population subgroups (adults, adolescents, and children), many iterations were executed where a single iteration yields the 24-h ETS-only benzene exposure for one subject. For each scenario, each of the nonsmoking subjects exposed to ETS in the APCR or CAP study was systematically sampled 40 times. Each time a subject is sampled, an independent realization of exposure is created. The total exposure is obtained as the sum of the exposures in each microenvironment which, in turn, is determined as the product of exposure period times the average microenvironmental concentration. The exposure periods for each microenvironment are constant for a given subject from one iteration to the next. The microenvironmental concentrations are determined stochastically, by either the tracer or the CMR method, depending on the scenario and the microenvironment. For the tracer method, the concentration is selected randomly from the constructed parent probability distribution function. For the CMR method, input parameters are selected at random from appropriate probability distribution functions and combined using the model equations (5.2), (5.3), or (5.4).

Each participant was sampled multiple times to generate a sufficient number of iterations to minimize fluctuations in the results caused by small sample size. Weighting factors from the APCR or CAP study were subsequently applied to the results to construct, from the iterations executed in a given scenario, probability distribution functions of exposure that represent the population of Californian nonsmokers who report being exposed to environmental tobacco smoke. The mean from these model calculations can be scaled by the percentage of Californian nonsmokers who report some exposure to ETS to determine the mean exposure for the statewide population of nonsmokers.

Table 5.2. Distributions of input parameters used in the four scenarios.

parameter	GM[1]	GSD[1]	reference
scenario TL (tracer low)			
residential — tracer			
ETS-only benzene ($\mu g\ m^{-3}$)			Quackenboss et al., 1991
summer	0.87	1.6	
spring/fall	0.71	4.2	
winter	1.67	2.5	
occupational — tracer			
ETS-only benzene ($\mu g\ m^{-3}$)	0.077	8.2	Jenkins et al., 1996
school — tracer			
ETS-only benzene ($\mu g\ m^{-3}$)	0.077	8.2	Jenkins et al., 1996
retail/other indoor — tracer			
ETS-only benzene ($\mu g\ m^{-3}$)	0.077	8.2	Jenkins et al., 1996
restaurant — tracer			
ETS-only benzene ($\mu g\ m^{-3}$)	1.2	1.5	Lambert et al., 1993
bar/nightclub — tracer			
ETS-only benzene ($\mu g\ m^{-3}$)	3.7	1.7	Miesner et al., 1989
transportation — CMR model [2]			
ventilation rate ($m^3\ h^{-1}$) [3]	104	3.1	see text
smoking rate (h^{-1})	0.74	2.35	APCR
residential guest — tracer			
ETS-only benzene ($\mu g\ m^{-3}$)			Quackenboss et al., 1991
summer	0.87	1.6	
spring/fall	0.71	4.2	
winter	1.67	2.5	
scenario TM (tracer medium)			
residential — tracer			
ETS-only benzene ($\mu g\ m^{-3}$)	1.15	3.4	Coultas et al., 1990
occupational — tracer			
ETS-only benzene ($\mu g\ m^{-3}$)	1.06	4.1	Turner et al., 1992
school — tracer			
ETS-only benzene ($\mu g\ m^{-3}$)	1.06	4.1	Turner et al., 1992
retail/other indoor — tracer			
ETS-only benzene ($\mu g\ m^{-3}$)	1.06	4.1	Turner et al., 1992
restaurant — tracer			
ETS-only benzene ($\mu g\ m^{-3}$)	4.7	2.2	Repace and Lowrey, 1980
bar/nightclub — tracer			
ETS-only benzene ($\mu g\ m^{-3}$)	8.3	2.3	Repace and Lowrey, 1980
transportation — CMR model [2]			
ventilation rate ($m^3\ h^{-1}$) [3]	104	3.1	see text
smoking rate (h^{-1})	0.74	2.35	APCR
residential guest — tracer			
ETS-only benzene ($\mu g\ m^{-3}$)	1.15	3.4	Coultas et al., 1990

Table 5.2. (continued)

parameter	GM[1]	GSD[1]	reference
scenario CM (CMR model medium)			
residential — CMR model [2]			
volume (m^3) [4]	310	1.78	RECS, 1982
air-exchange rate (h^{-1}) [5]			Murray and Burmaster, 1995
N. California, winter	0.38	1.80	
N. California, spring	0.45	2.19	
N. California, summer	0.56	1.84	
N. California, fall	0.46	1.57	
S. California, winter	0.51	1.91	
S. California, spring	0.62	1.95	
S. California, summer	1.05	2.49	
S. California, fall	0.42	2.03	
smoking rate (cig $smoker^{-1}$ d^{-1})	see text		APCR
occupational — CMR model [2]			
ventilation rate (m^3 $pers^{-1}$ h^{-1}) [6]	36.9	1.81	Persily, 1989
smokers (%)	22	1.0	APCR
smoking rate (cig $smoker^{-1}$ h^{-1})	0.99	1.0	APCR
school — CMR model [2]			
ventilation rate (m^3 $pers^{-1}$ h^{-1}) [6]	36.9	1.81	Persily, 1989
smokers (%)	22	1.0	APCR
smoking rate (cig $smoker^{-1}$ h^{-1})	0.99	1.0	APCR
retail/other indoor — CMR model [2]			
ventilation rate (m^3 $pers^{-1}$ h^{-1}) [6]	36.9	1.81	Persily, 1989
smokers (%)	22	1.0	APCR
smoking rate (cig $smoker^{-1}$ d^{-1})	11.76	2.35	APCR
restaurant — tracer			
ETS-only benzene (μg m^{-3})	4.7	2.2	Repace and Lowrey, 1980
bar/nightclub — tracer			
ETS-only benzene (μg m^{-3})	8.3	2.3	Repace and Lowrey, 1980
transportation — CMR model [2]			
ventilation rate (m^3 h^{-1}) [3]	104	3.1	see text
smoking rate (h^{-1})	0.74	2.35	APCR
residential guest — tracer			
ETS-only benzene (μg m^{-3})	1.15	3.4	Coultas et al., 1990

Table 5.2. (continued)

parameter	GM[1]	GSD[1]	reference
scenario TH (tracer high)			
residential — tracer			
ETS-only benzene (μg m^{-3})	2.1	2.8	Spengler et al., 1985
occupational — tracer			
ETS-only benzene (μg m^{-3})			Hammond et al., 1995
White collar worksites	3.7	2.6	
Blue collar worksites	0.92	3.2	
school — tracer			
ETS-only benzene (μg m^{-3})	3.7	2.6	Hammond et al., 1995
retail/other indoor — tracer			
ETS-only benzene (μg m^{-3})	4.5	1.4	Löfroth, 1993
restaurant — tracer			
ETS-only benzene (μg m^{-3})	6.2	2.7	Löfroth, 1993
bar/nightclub — tracer			
ETS-only benzene (μg m^{-3})	19.2	1.6	Collett et al., 1992
transportation — CMR model [2]			
ventilation rate (m^3 h^{-1}) [3]	104	3.1	see text
smoking rate (h^{-1})	0.74	2.35	APCR
residential guest — tracer			
ETS-only benzene (μg m^{-3})	2.1	2.8	Spengler et al., 1985

[1] GM - geometric mean; GSD - geometric standard deviation

[2] CMR model - completely mixed room model

[3] Minimum vehicle ventilation rate permitted in simulations is 3.0 m^3 h^{-1}.

[4] Minimum residence volume permitted in simulations is 40 m^3.

[5] Minimum residential air-exchange rate permitted in simulations is 0.05 h^{-1}.

[6] Minimum occupational ventilation rate permitted in simulations is 5 m^3 person h^{-1}.

When using the CMR approach, we have attempted to retain correlations that may exist among parameters. For example, air-exchange rates in indoor environments are expected to vary systematically with season, since it is likely that people leave windows and doors open more frequently during the months when the outdoor air temperature is in a comfortable range. We have incorporated this phenomenon into our model by using the month of participation in the APCR to determine the distribution from which to draw the residential air-exchange rate.

5.1.3 ETS Concentrations and Exposures in Microenvironments

In this section, we provide a detailed description of the input parameters that were used to determine microenvironmental concentrations of environmental tobacco smoke. A summary of the parameters for all four scenarios is presented in Table 5.2.

5.1.3.1 Residences

For nonsmokers living with smokers who regularly smoke in the home, the residence can be a significant site for exposure to ETS (Emmons et al., 1992). While ETS concentrations in houses are often comparable to those found in other settings where smoking occurs, the amount of time that people spend at home can often lead to high levels of exposure. In the APCR study, for example, the average person spent 7.4 nonsleeping hours in their own home. Based on self-reported proximity, this location makes up about 17% of the total amount of time nonsmoking adults in the APCR spent in the presence of ETS. For adolescents and children this proportion is much higher, 37% and 58%, respectively. (See Figure 3.2.)

ETS concentrations in residences were determined using both the CMR model and ETS tracer methods. Exposure was then calculated as the product of this concentration times an exposure period spent in the residence.

For exposure period, we used the total waking time spent by the nonsmoker at home plus, for adolescents and children, any self-reported proximity time while asleep, rather than just the self-reported proximity time. (For adults, the self-reported proximity time while asleep contributes a small fraction of total exposure time, ~ 3%, and was not included.) In this setting, self-reported proximity was used as a binary indicator to determine which nonsmokers received ETS exposure in their own residence. The use of the self-reported proximity as the total ETS exposure time introduces several potential sources of error or ambiguity, such as the ability of the nonsmoker to detect ETS and the definition of "proximity." Because of the structure of the activity pattern survey, these limitations are magnified in residential settings, where a nonsmoker could be in a different room from a smoker, and still be exposed to ETS, but not report that they are in the proximity of a smoker.

In constructing a method for estimating concentrations, we assumed that ETS concentrations were zero during sleeping hours (8 h per day). Although the reality is more complex, this is a reasonable approximation assuming that there is a period of time of ~ 8 h duration at night when no smoking occurs within the residence. In applying the tracer method, where the reported measurements were based on integrated samples over periods of 24 h or 1 week, daytime average concentrations were estimated by multiplying the reported daily average concentration by a factor of 1.5 (24 h total per day divided by 16 waking h per day). Exposure was then computed as the product of a randomly sampled concentration for the awake hours times the at-home exposure period for the nonsmoker. Similarly, for the CMR method, the daily consumption of cigarettes by a smoker is assumed to occur evenly over a waking period of 16 h. The number of cigarettes smoked at home is then the daily smoking rate times the fraction of the waking day spent at home. The exposure of a nonsmoker is computed, as in the tracer method, as the product of a computed awake-period concentration times the residential exposure period of the nonsmoker.

5.1.3.1.1. CMR Method. For the CMR model calculation, we used equation (5.2) to calculate the residential ETS concentration. For adult nonsmokers, we assumed that there was no more than one smoker in any given residence (that is, n = 1). An annual study organized by the California Department of Health Services gathers information concerning the number of nonsmokers living with smokers across the state, as well as on the existence of smoking rules in California homes (California Department of Health Services, 1995). These results show that it is unlikely for an adult nonsmoker to live with more than one smoker.

For adolescents and children, the probability of living with more than one smoker is higher than for an adult. (This is true because the probability of living with 2 or more other adults is higher for children and adolescents than for adults.) For both of these population groups, we used survey data that shows the family structure of California adolescents in 1990 by family smoking status (Pierce et al., 1993). These data show that when an adolescent lives with a smoker, 72% of the time there is only one smoker in the household, 26% of the time there are two smokers, and 2% of the time there are three smokers (two parents/guardians plus an older sibling). In the CM scenario, we used these proportions to probabilistically select the number of smokers in the household whenever an adolescent or child was exposed to ETS in their residence.

We used data from the APCR study to simulate the number of cigarettes smoked inside the home. The survey reports pertinent information on participants who are smokers, such as the number of cigarettes smoked per day and the fraction of time spent at home. Considering all smokers surveyed in the APCR study, the number of cigarettes consumed daily is represented by a lognormal distribution with a GM of 11.8 (AM = 15.8) cigarettes per smoker per day, and a GSD

of 2.35. The cigarette smoking rate, N_r, is obtained by multiplying the daily number of cigarettes consumed by a smoker by the nonsleeping time that the smoker was in his/her residence and dividing the result by 16 waking hours in a day.

For adults and adolescents, we linked the responses from a randomly selected smoker (or smokers) from the APCR study with each exposed nonsmoker. This "matchmaking" scheme produced both the number of cigarettes and the time spent at home by the smoker. Using smoker responses allowed us to avoid some of the pitfalls of self-reported proximity, and to make use of real data, rather than relying on representative distributions of smoking rates.

Because of the different structure of the APCR and CAP data files, it was impractical to use this approach for children. Instead, we used the APCR data on smokers to determine lognormal parameters for cigarettes smoked at home per day. Specifically, for each smoker in the APCR, we estimated the at home cigarette consumption by multiplying the total number of cigarettes smoked by the fraction of the waking day spent at home. The resulting (weighted) GM and GSD were 0.31 cigarettes smoker^{-1} h^{-1} and GSD = 3.0. (The daily arithmetic mean was 8.0 cigarettes per smoker, 51% of the total smoking rate, indicating that on average the smokers in the APCR spent about half of their waking day at home.) For children, a lognormal distribution with these parameters was applied to randomly select the cigarette consumption rate N_r for each smoker in an exposed child's residence.

The emission factor for benzene is drawn from a normal distribution based on the results of Daisey et al. (1998), who report a mean ± standard deviation of 406 ± 71 μg cig^{-1} for measurements of the brands most commonly smoked in California.

House volumes were based on data published from a study of US housing conducted for the US Department of Energy (Residential Energy Consumption Survey, 1982). The floor area was measured for the heated portion of 6051 randomly selected residences (from across the country). The report tabulates the number of residences in each of seven size classes for six categories corresponding to the number of household members. After assuming a fixed ceiling height of 2.4 m, we constructed a population-weighted lognormal distribution for volume per household. The resulting estimate is a geometric mean of 310 m^3 and a geometric standard deviation of 1.77. For each iteration in our simulations, we selected the house volume at random from this lognormal distribution. To avoid selecting an unrealistically low home volume, we set a minimum value for volume of 40 m^3. Any randomly selected volume that was below this minimum was discarded.

Air-exchange rate data were derived from the report of Murray and Burmaster (1995) who summarize measurements made with perfluorocarbon tracers in 2,844 US households. We used data segregated by season for Regions 3 and 4 as defined by those authors. Region 3 was used to represent Northern California; 71 of 332 total measurements in the region were made in Northern

California households. Region 4 was used to represent Southern California; here 95% of the 1549 total measurements were made in California. The parameters of the lognormal distributions are reported directly in the reference (Table III) and reproduced in our Table 5.2. These distributions are generally consistent with the air-exchange rates reported for a smaller, although still substantial, set of measurements in California houses by Wilson et al. (1996).

In addition to time spent in one's own residence, the APCR and CAP studies contain data on the time spent as a guest in another's home. We decided to represent time spent as a "residential guest" as a separate microenvironment (§5.1.3.8) since the activity pattern data that we have on household characteristics does not pertain to homes visited by study participants.

5.1.3.1.2. Tracer Method We identified three studies that contained suitable data from which to derive a distribution of ETS concentrations in residences.

The low-range (TL) scenario is based on the study of Quackenboss et al. (1991). In this study, concentrations of particulate matter ($PM_{2.5}$ and PM_{10}) were measured over week-long periods in residences in the vicinity of Tucson, Arizona. Median, 25th percentile, and 75th percentile concentrations are reported for 112 cases with "smokers at home" and 113 cases with "no smokers at home," segregated according to season (summer, spring/fall, and winter) (Table 2 of the reference). We used the $PM_{2.5}$ data. The geometric mean (GM) of the ETS-only contribution to particulate matter is estimated as the median concentration for sites with smokers at home minus the median for sites without smokers. The geometric standard deviation (GSD) is estimated from the 25th and 75th percentile measurements. The 75th percentile concentration of the ETS-only contribution (C_{75}) is assumed to be given by the difference between the 75th percentile concentrations with and without smokers at home, and the 25th percentile concentration (C_{25}) is similarly determined. Then, the GSD is obtained from the relationship

$$\frac{C_{75}}{C_{25}} = GSD^{1.35} \tag{5.7}$$

where the power 1.35 derives from the properties of a normal distribution (Selby, 1974). The GM of the ETS contribution to PM is converted to an estimate for benzene using equation (5.5). The GSD for benzene is assumed to be the same as for PM. Since measurements were made over a 24-h period, the daytime distribution is estimated by multiplying the GM by a conversion factor of 1.5 (24 h total divided by 16 h awake). The GSD is not altered by this correction.

The mid-range (TM) scenario utilizes the results of Coultas et al. (1990). This study reports the concentration of nicotine and RSP in 10 homes in and around Albuquerque in which at least one cigarette smoker resided. Measurements were made over 24-h periods and repeated for 10 separate days at each site. Our assessment is based on the nicotine data reported in Figure 2 of that paper and in the text. We read from the figure the highest 50 individual points from among 99

total measurements. The GSD was computed by fitting a linear regression to the natural logarithm of the nicotine concentration plotted against z, where z represents the number of standard deviations away from the mean for the given percentile in a standard normal distribution. The exponential of the slope of this line is the GSD. The arithmetic mean (AM) was then computed as the mean of the average values for the ten houses (also directly reported in Figure 2). Finally, the GM was computed from this relationship, which holds for lognormal distributions:

$$AM = GM \exp\left[\frac{\ln(GSD)^2}{2}\right] \tag{5.8}$$

The GM for nicotine is converted to a GM for benzene by equation (5.6). Again, since the measurements were made over 24-h periods, the daytime average concentration was determined by multiplying the daily average by a factor of 1.5. The resulting lognormal distribution for benzene from ETS is GM = 1.15 $\mu g\ m^{-3}$ and GSD = 3.4.

The high-range (TH) scenario is based on the findings of Spengler et al. (1985). The investigators in this study measured RSP levels over 24-h periods in 80 homes with smokers and 186 homes without smokers in two towns in Tennessee: Kingston and Harriman. Table III of that paper reports arithmetic mean (AM) and standard errors (SE) for the two groups: 74 $\mu g\ m^{-3}$ (SE = 6.6) for households with smokers and 28 $\mu g\ m^{-3}$ (SE = 1.1) for households without smokers. The arithmetic standard deviation is determined as the product of the standard error times the square root of the number of measurements: 59 $\mu g\ m^{-3}$ for smoking households and 15 $\mu g\ m^{-3}$ for nonsmoking households. Next, the arithmetic statistics of the ETS-only contribution to RSP are estimated. The AM is obtained as 74-28 = 46 $\mu g\ m^{-3}$. The standard deviation is estimated to be 61 $\mu g\ m^{-3}$ using a formula from the theory of propagation of errors (Bevington and Robinson, 1992):

$$SD_{ETS\text{-}only} = \left[(SD_{smoking})^2 + (SD_{nonsmoking})^2\right]^{1/2} \tag{5.9}$$

These parameters are then converted from RSP to benzene using equation (5.5) (AM = 2.3 $\mu g\ m^{-3}$ and SD = 3.0 $\mu g\ m^{-3}$). Finally, the parameters, GM and GSD, are estimated assuming that the true distribution is lognormal, and that the arithmetic mean and standard deviation are as derived. The method for this conversion is based on a custom spreadsheet program. In this program, the user provides the AM and SD of a distribution and a guess for the GM. The program computes the GSD from equation (5.8) assuming a lognormal distribution, then computes 1000 evenly distributed percentiles (0.1-99.9%) and computes from these the AM and SD of the lognormal. These computed values are compared against the input AM and SD. By iteratively adjusting the GM, the user obtains a best-fit lognormal in which the AM and SD agree with input values to within 1%. As with the other residential tracer measurements, the daily-average result is converted

to a daytime average by multiplying the GM by 1.5. The final estimate for the lognormal distribution of benzene from ETS is GM = 2.1 μg m^{-3}, GSD = 2.8.

The Particle Total Exposure Assessment Methodology (PTEAM) study measured indoor and outdoor air concentrations and personal exposures to particulate matter and nicotine in a random sample of 178 participants who represent the 139,000 nonsmoking residents of Riverside, CA (Özkaynak et al., 1996a and 1996b). Statistical parameters are reported for daytime and overnight measurements (12-h averages) of $PM_{2.5}$, PM_{10}, and nicotine in home with and without smokers in Tables 5.6 and 5.7 of Özkaynak et al. (1996b). The arithmetic mean nicotine concentration in homes with smokers is 1.2 μg m^{-3}. The average increase in $PM_{2.5}$ in homes with smokers relative to those without smokers is 29 μg m^{-3}. Figure 5.19 shows that the nicotine measurements in homes with smokers conform reasonably to a lognormal (up to the 95th percentile) with a GSD of 2.8. Applying equations (5.5) and (5.6) to the PTEAM data indicates a mean indoor benzene level from ETS in homes with smokers of 0.53 μg m^{-3} (based on nicotine measurements) or 1.45 μg m^{-3} (based on the $PM_{2.5}$ measurements). Correcting for a null contribution during sleeping hours, the awake-hour means would be 0.8 μg m^{-3} and 2.2 μg m^{-3}, respectively. The corresponding arithmetic means for benzene from ETS used in this study are 1.0 μg m^{-3} (summer), 2.0 μg m^{-3} (spring/fall), 2.5 μg m^{-3} (winter) for TL, 2.4 μg m^{-3} for TM, and 3.6 μg m^{-3} for TH, with GSDs in the range 1.6-4.2. Thus, the ETS contribution to benzene inferred from the $PM_{2.5}$ data in the PTEAM study are consistent with the data employed here. The nicotine measurements in PTEAM would indicate a lower contribution of ETS to indoor air concentrations of TACs. Özkaynak et al. (1996a) noted this discrepancy and suggested the possibility that the analytical method employed may have undersampled gas-phase nicotine.

It is noteworthy that the air-exchange rates in the Riverside homes were found to be relatively high. The GM and GSD were 0.97 h^{-1} and 2.18 (Özkaynak et al., 1996a). PTEAM monitoring was conducted during the autumn, a period when the weather tends to be pleasant in Riverside. The high average air-exchange rate values suggest that windows may have been open often, which would tend to reduce the significance of indoor emission sources on personal exposure.

5.1.3.2 Office/Occupational

Many Californians work in large, commercial buildings with mechanical ventilation systems. These systems usually combine fresh air, drawn from outside the building, with recirculated air, which is recycled through a large zone, even, in some cases, the entire building. This design, along with the lack of high-quality particle filters or other pollutant removal systems in most buildings, leads to air contaminants from one area being spread throughout a larger region. Studies have shown that in buildings with designated smoking areas, nonnegligible ETS concentrations can be detected in

nonsmoking areas of the same building, even when they are some distance away (Hayward et al., 1995). Clearly, in buildings with lenient smoking policies, nonsmokers throughout the building may receive significant ETS exposures.

For commercial buildings, data on building parameters necessary to calculate concentrations using the CMR model were available. Exposure in workplaces was also determined using the tracer method.

5.1.3.2.1. CMR Method The right-hand side of equation (5.3) contains four parameters that must be determined to predict ETS concentrations in occupational settings. For f, the fraction of the building occupants that smoke, we assume that a fixed value of 22% applies, which corresponds to the average smoking rate among adults in California, based on the APCR survey. For N_o, the cigarette consumption rate, we use the arithmetic mean value, 0.99 cigarettes smoker^{-1} h^{-1}, derived from the APCR data (15.8 cigarettes per smoker per day divided by 16 waking hours per day). This value is treated as fixed, rather than stochastic. In buildings with a large number of occupants, the variability in average cigarette consumption rate per smoker will be much smaller than the individual variability and, therefore, is not expected to contribute significantly to the variability in exposure. For the emission factor, e, we use the same input parameter as for residences, 406 ± 71 μg cig^{-1} for benzene.

Data on the air-exchange rates of office buildings were derived from a study that reports on 3000 ventilation rate measurements from 14 large office buildings distributed across the United States (Persily, 1989). Figure 19 of that paper presents, in histogram form, an unbiased distribution of individual, whole-building air-exchange measurements (averaged over a few hours) using tracer gas decay. A linear fit to the cumulative distribution plotted on log-probability coordinates shows that the data conform well to a lognormal distribution with a GM of 0.74 h^{-1} and a GSD of 1.81. A minimum air-exchange rate of 0.1 h^{-1} was imposed in the Monte-Carlo simulation procedure.

In exploratory simulations, we included low and high-range scenarios based on the CMR method. Ultimately, these were discarded for two reasons: (a) multiple high-quality data sets did not exist for most parameters for the CMR model method as compared with the tracer method; and (b) the use of six scenarios instead of four seemed to obfuscate the important findings, rather than illuminate them. In these early runs, we used an additional source of ventilation rate data for commercial buildings (Lagus, 1995). This study was conducted in a convenience sample of 49 nonresidential buildings in California, including offices, schools, and some retail stores. Air-exchange rate measurements were made using a tracer-gas method. Multiple measurements were made at most buildings. For 22 office buildings, the data conform reasonably well to a lognormal distribution with a geometric mean of 1.1 h^{-1} and a GSD of 1.84. Relative to the Persily study,

this work has the advantage of being California specific. Key disadvantages are that the buildings studied were selected for convenience rather than to be representative, and the measurements at each building were only made on a single day. Persily made a very large number of measurements in each of his 14 buildings.

Equation (5.3) requires the ventilation rate in flow per person, q_{avg}, rather than in terms of the whole-building air-exchange rate. The conversion was made assuming that one air change per hour is equivalent to a ventilation rate of 50 m^3 per person per hour. The basis for this factor is presented by Persily (1989). It assumes that office building volume is allotted at a rate of 50 m^3 per occupant, based on a standard occupancy of 7 persons per 100 m^2 and a 3.5-m ceiling height (including the air plenum). Thus, seven building occupants will be stationed in 350 m^3 of volume, and an air-exchange rate of 1 h^{-1} will correspond to a ventilation rate of 350 m^3 h^{-1} or 50 m^3 $person^{-1}$ h^{-1}. Thus, the GM for ventilation rate per person of 36.9 m^3 $pers^{-1}$ h^{-1} corresponds to the GM for air-exchange rate of 0.74 h^{-1}.

5.1.3.2.2. Tracer Method Three studies contain substantial data on concentrations of ETS tracers in workplaces; we interpreted all three to obtain distributions for the TL, TM, and TH scenarios.

For scenario TL, we used data from Jenkins et al. (1996). In this study, the personal exposure was measured at work during a single shift for a sample (not a statistically representative sample) of 379 people who reported observing the use of tobacco products at their workplace. Table 9 of that paper reports that the median nicotine concentration (8-hour average value) from this sample was 0.200 μg m^{-3}, the mean was 1.69 μg m^{-3} and the 95th percentile was 7.66 μg m^{-3}. Corresponding numbers for 730 subjects in nonsmoking workplaces were 0.0264 μg m^{-3}, 0.109 μg m^{-3}, and 0.342 μg m^{-3}. We estimated parameters for a lognormal distribution as follows. The AM is taken as the difference between the smoking and nonsmoking means: 1.69 - 0.109 = 1.58 μg m^{-3}. The GM is taken as the difference between the smoking and nonsmoking medians: 0.200 - 0.0264 = 0.174 μg m^{-3}. Given these values of AM and GM, the GSD is estimated from equation (5.8) to be 8.2. The GM for ETS-only benzene is then found to be 0.077 μg m^{-3} by applying equation (5.6). (The corresponding AM is 0.70 μg m^{-3}.)

For scenario TM, the results of Turner et al. (1992) were utilized. Here, 585 office environments were sampled during 1989 over one-hour periods for ETS tracers. The building sites are unspecified but the authors state that they "have no reason to suspect that the buildings in this sample are not representative of office buildings throughout North America." We used the nicotine results reported in Table 2 of that paper. The arithmetic mean contribution of smoking to indoor nicotine is estimated as the difference between mean values for smoking and nonsmoking environments: 6.7 – 0.2 = 6.5 μg m^{-3}. The arithmetic standard deviation is estimated from equation (5.9); given that the reported standard deviations for smoking and nonsmoking

environments are 14.8 and 0.8 μg m^{-3}, respectively, we find $SD_{ETS\text{-}only}$ = 14.8 μg m^{-3}. We converted these results to ETS-only benzene by applying equation (5.6); the estimated arithmetic mean contribution of ETS to benzene levels in offices is 2.9 μg m^{-3} with a standard deviation of 6.5 μg m^{-3}. Then, we applied the spreadsheet program described in §5.1.3.1.2 to determine the lognormal parameters for these conditions, with the result GM = 1.06 μg m^{-3} and GSD = 4.1.

For scenario TH, we used data from Hammond et al. (1995). In this study, passive nicotine samplers were placed for week-long periods in 25 worksites in Massachusetts. We used measurements reported in the last column of p. 957. The nicotine concentrations in open offices where smoking was permitted showed a median of 8.6 μg m^{-3}, an arithmetic mean of 14 μg m^{-3}, and a 90th percentile value of 34 μg m^{-3}. Where smoking was banned, the corresponding values were 0.3, 0.7, and 1.7 μg m^{-3}. We converted these results to ETS-only benzene by taking the difference between the smoking permitted and smoking banned measurements and the applying equation (5.6). On this basis, we estimate the AM contribution of smoking to benzene levels in offices to be 5.9 μg m^{-3}. We assume that the GM is given by the median; for ETS-only benzene this yields 3.7 μg m^{-3}. The GSD is then found to be 2.6, using equation (5.8).

Not all work occurs in offices. The Hammond et al. data provide a basis for estimating exposure in nonoffice worksites separately from office environments. They made 221 measurements of nicotine in production areas and fire stations. The time-averaged nicotine concentrations where smoking was permitted showed a median of 2.3 μg m^{-3}, an arithmetic mean of 4.4 μg m^{-3}, and a 90th percentile value of 7.2 μg m^{-3}. Where smoking was banned, the corresponding values were 0.2, 0.2, and 0.6 μg m^{-3}. Applying the same method described in the previous paragraph, we estimate the ETS-only benzene level in blue-collar worksites to be described by a lognormal distribution with a GM of 0.92 μg m^{-3} and a GSD of 3.2. In the TH scenario, those respondents whose occupations had category codes 61-97 were treated as working at a blue-collar site;[1] all other occupational category codes were treated according to the white-collar distribution (see Table 3.2). For adolescents and children, for whom exposure in the "occupational/office" microenvironment group was small, only the office tracer data were used.

There is some ambiguity in the Hammond et al. measurements. The samplers were left in place for a full week. However, the buildings were typically occupied for only 45 hours during the week. Hammond et al., assumed that the nicotine concentrations were zero during the unoccupied periods and estimated the time-averaged concentration during work hours by dividing the nicotine exposure, measured by the samplers, by an assumed 45 hour exposure period.

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[1] Adult occupation category codes 61-97 include the following occupations: farming, forestry, fishing, craftsmen, repairmen, precision production, machine and vehicle operators and fabricators, helpers, laborers and related.

Whether this approach accurately determines exposure during occupied periods is obscured by the potential adsorption and desorption of nicotine from indoor surfaces. (See Ogden, 1996.)

5.1.3.3 Schools

Explicit data for schools needed to make an accurate assessment of ETS exposure are lacking. Instead, we used the data and approach from occupational (white-collar) settings as the best surrogate. When a subject reported ETS exposure both at school and at work, those sites were modeled separately (i.e., separate parameter values or tracer concentrations were selected).

Some air-exchange rate data are available for schools in California (Lagus, 1995). Fourteen schools were measured, yielding a GM of 2.1 h^{-1} and a GSD of 1.8. By themselves, these data are insufficient to model exposure using either equation (5.2) or equation (5.3). Additional data on parameters such as number of smokers in the indoor environment and room volume would be required. Such data are unavailable. We also know of no data on ETS tracers measured in schools. In the absence of adequate data, we decided that the best approach would be to use the "office/occupational" microenvironment as a surrogate for schools. We modeled exposure following the same method in each scenario used in offices. Although this approach is not ideal, we judged it to be the best possible given the current state of information. We also note that schools contribute only a minor fraction of the total time of self-reported proximity of nonsmokers to others smoking (see Figure 3.2).

5.1.3.4 Retail/Other Indoor

This category of microenvironments, which includes shopping malls, beauty parlors, and barber shops (see Tables 3.2-3.4) appears potentially important as a site of ETS exposure. For example, in the APCR, more than 10% of nonsmoking adults report ETS exposure in this microenvironment group (see Figure 3.3), similar to the other three most common sites of exposure: residences, occupational, and restaurants.

Unfortunately, little information exists from which to estimate ETS concentrations in these settings. The environments are highly diverse, even within a single subclass. Data needed to apply the CMR model are generally lacking and ETS tracer measurements are sparse.

Air-exchange rates for a sample of 13 California buildings in the "retail/other" category have been reported (Lagus, 1995). The sample included a "church conference and meeting building, a nursing home, a funeral home, two automobile dealerships, a truck stop, community college common buildings ..., a large non-mechanically ventilated store and 3 large modern detached retail store[s]." The investigators noted that "it is unlikely that the buildings tested are representative of all retail buildings in California....There were no malls, neither large nor strip, ..., movie theaters, etc." The results show a GM of 1.8 h^{-1} and a GSD of 1.9.

Because of the lack of adequate data to separately represent the "retail/other" class of microenvironments, for three of the four scenarios — TL, TM, and CM — we used the occupational microenvironment as a surrogate. In these cases, we applied the same method used to estimate occupational ETS concentrations for the retail/other settings where exposure was reported. The product of this concentration estimate times the self-reported proximity interval served as the means to estimate exposure.

We identified one direct study of ETS in this set of microenvironments. Löfroth (1993) reported nicotine concentrations measured in the air of one shopping mall, in Sweden, in which smoking was permitted. He reports (in Table 1) time-averaged concentrations over periods of 4–6 hours for six separate days during the winter of 1990–91. We computed lognormal parameters directly from these six measurements and converted the GM to ETS-only benzene by equation (5.6). The result is GM = 4.5 $\mu g\ m^{-3}$ and GSD = 1.4. Because this was a small study located outside of California (and even outside of the U.S.), we only used these data in one scenario, TH.

5.1.3.5 Restaurants

Until recently, smoking in restaurants was almost always permitted (and practiced) and, consequently, ETS exposures could be high. During most of the past few decades, restaurants typically have either had no restrictions on smoking, or else divided their space into smoking and nonsmoking sections. In the latter case, some research has questioned whether the partitioning has actually reduced exposure to ETS, or merely placated the nonsmoking patrons (Lambert et al., 1993).

No suitable data were found on restaurant volumes or air-exchange rates, parameters needed for a CMR model calculation. However, several studies report on the concentrations of ETS tracers in restaurant environments. Thus, we used the tracer method to estimate microenvironmental concentrations in restaurants for all four scenarios.

For scenario TL, we based our analysis on data from Lambert et al. (1993) who measured nicotine and RSP in the smoking and nonsmoking sections of seven Albuquerque restaurants. We generated a lognormal distribution of ETS-only benzene, based on the reported concentrations of nicotine in the smoking sections of the restaurants (Figure 2, erratum). We read the data from the figure, computed the lognormal statistics directly (by taking the mean and standard deviation of the natural logarithm of the measurements, then exponentiating), and converted the GM to ETS-only benzene by applying equation (6). The resulting lognormal parameters for benzene from ETS is a GM of 1.2 $\mu g\ m^{-3}$ and a GSD of 1.5.

Scenarios TM and CM were based on the measurement results of Repace and Lowrey (1980). They report RSP measurements in six restaurants while smoking was observed (eight measurements, with two at each of two sites) and in three restaurants in the absence of smoking (four measurements reported, but two are at the same site). The data are presented in their Tables 5 and 3, and reproduced here. For the smoking environments, the RSP concentrations ($\mu g\ m^{-3}$) were 414 at site E, 158 at site K (avg. of 2 meas.), 136 at site L, 110 at site M (smoking section), 109 at site N (Sample 1), 86 at site R (smoking section), and 107 at site S (Sample 1). Sites M and R were the same restaurant (Repace, 1998), measured on separate days, and so are averaged here to represent a single site at 98 $\mu g\ m^{-3}$. Overall, the arithmetic mean ± standard deviation in the six restaurants with smoking was 170 ± 121 $\mu g\ m^{-3}$. For the nonsmoking environments, the RSP levels ($\mu g\ m^{-3}$) were 29 at crepes, 53 at sandwich (avg. of 2 meas.), and 38 at fast food. So, the RSP levels in restaurants in the absence of smoking was AM ± SD = 40 ± 12 $\mu g\ m^{-3}$. The net contribution of ETS to RSP is estimated to have an AM of 130 $\mu g\ m^{-3}$, given by the difference in these means, and a standard deviation of 122 $\mu g\ m^{-3}$, from equation (5.9). These estimates were converted to ETS-only benzene values (AM ± SD = 6.5 ± 6.1 $\mu g\ m^{-3}$) by application of equation (5.5). Then, the spreadsheet program described in §5.1.3.1.2 was applied to estimate the corresponding lognormal parameters for benzene from ETS: GM = 4.7 $\mu g\ m^{-3}$ and GSD = 2.2.

The TH scenario is based on measurements reported by Löfroth (1993). Table 3 of that paper reports nicotine concentrations, measured over periods of 1-6 h in 4 restaurants and a cafeteria (eight samples, total). Smoking was confirmed to have occurred in these settings during sampling (Löfroth, 1998). The lognormal parameters were computed directly from the eight measurements by first averaging the measurements made at a single site, then computing the GM and GSD of the resulting five measurements (5.0, 9.6, 11.5, 14, and 74 $\mu g\ m^{-3}$). Then the GM was converted to an ETS-only benzene value by applying equation (5.6). The resulting lognormal parameters for benzene from ETS are GM = 6.2 $\mu g\ m^{-3}$ and GSD = 2.7.

We identified a few other candidate studies (Miesner et al., 1989; Oldaker et al., 1990) but did not use their results. The Miesner et al. work was not included because measurements were made in only two restaurants, in contrast to the 5-7 sites for each of the studies we used. The

study of Oldaker et al. covers, by far, the largest number of sites. They made measurements of nicotine and other ETS tracers in at least 30 restaurants in each of 3 major US cities and report that smoking was observed in all of the restaurants. They only report the geometric mean and the range of results (5.1 $\mu g\ m^{-3}$ and 0–23.8 $\mu g\ m^{-3}$, respectively, for nicotine), insufficient information from which to estimate the GSD. The GM of 5.1 $\mu g\ m^{-3}$ corresponds to an ETS-only benzene GM of 2.2 $\mu g\ m^{-3}$, which lies within the range established by the low and high-exposure scenarios we have included. (We were unsuccessful in our attempts to contact Oldaker to obtain additional information that would have permitted us to use the results from this study directly.)

5.1.3.6 Bars/Nightclubs

When categorizing the common locations where Californians are most likely to encounter ETS exposure, certain patterns quickly emerge. One observation is that only a small fraction of nonsmokers visit bars and nightclubs on any given day. However, a large majority of those who do—80% in the APCR study—report proximity to smoking. Furthermore, anyone who has visited a bar or nightclub where smoking is permitted recognizes that the density of smokers and the ETS concentrations are high.

Because of a basic lack of data on smoking intensity, building volume, and ventilation rates, we judged that we could not apply the CMR approach for estimating microenvironmental concentrations in bars and nightclubs. Instead, we relied solely on the ETS tracer method, identifying three separate studies to provide low, mid-range, and high exposure estimates.

For scenario TL, we used measurements of nicotine reported for three bars by Miesner et al. (1989). At one site, separate measurements were made on each of three levels, and we used the arithmetic mean at this site. From the three measurements (4.7, 9.5, and 13.1 $\mu g\ m^{-3}$), the GM and GSD were computed and the GM was converted to an ETS-only benzene estimate by application of equation (5.6). The contribution of ETS to benzene for this scenario was thus estimated to have a GM of 3.7 $\mu g\ m^{-3}$ and a GSD of 1.7.

Scenarios CM and TM were based on RSP data reported in Table 5 of Repace and Lowrey (1980). The AM ± SD at four sites (C, bar and grill; F, bar/cocktail lounge; P, neighborhood restaurant/bar; and Q, hotel bar) was 277 ± 237 $\mu g\ m^{-3}$. To correct for nonETS contributions to RSP, we used the average in nonsmoking restaurants, as described in §5.1.3.5; the AM ± SD of these values is 40 ± 12 $\mu g\ m^{-3}$. The net contribution of ETS to RSP is then estimated to be 237 ± 237 $\mu g\ m^{-3}$. We converted these parameters to ETS-only benzene estimates (AM ± SD = 11.9 ± 11.9 $\mu g\ m^{-3}$) by applying equation (5.5). Then, lognormal parameter estimates were obtained using the spreadsheet program described in §5.1.3.1.2. The resulting lognormal distribution parameters for benzene from ETS are GM = 8.3 $\mu g\ m^{-3}$ and GSD = 2.3.

For scenario TH, we used nicotine concentration measurements reported in Table 2 of Collett et al. (1992). This table reports on measurements over 2-h periods in six nightclubs, four taverns, and five neighborhood pubs in Vancouver, British Columbia. The results from all sites were combined to obtain the AM ± SD for nicotine in the entire sample as 48.8 ± 24.4 $\mu g\ m^{-3}$. These results were converted to benzene from ETS estimates by application of equation (5.6), with the result AM ± SD = 21.5 ± 10.7 $\mu g\ m^{-3}$. Then, the spreadsheet program described in §5.1.3.1.2 was applied to determine the lognormal distribution parameters for benzene from ETS: GM = 19.2 $\mu g\ m^{-3}$ and GSD = 1.6.

We also reviewed a paper by Eatough et al. (1989) that reported measurements of ETS tracers in one disco. Because only one site was measured, we consider this source less valuable than the other three papers addressing this microenvironment and did not include the data in our analysis.

5.1.3.7 Transportation

Many Californians are exposed to ETS while using various modes of transportation. Because cigarette smoking has been prohibited for some time in most modes of public transportation, such as buses and subways, our model assumes that exposures to ETS occur in private vehicles only. In the APCR study, well over 90% of the reported ETS exposures in enclosed vehicles occurred in either an automobile or a van.

This microenvironment was the only one for which adequate ETS tracer measurements were completely unavailable and no suitable surrogate microenvironment could be specified. One published study reported differences between in-vehicle concentrations of CO in the presence and absence of smoking (Koushki et al., 1992). We chose not to use these data, since CO is a poor tracer of ETS, especially in the transportation environment where automobiles are themselves a major source of CO. A second study measured the concentrations of RSP and CO in an automobile in the presence of smoking (Ott et al., 1992). However, this study was designed to validate a microenvironmental model rather than to generate data that are representative of smoking exposures in motor vehicles. Consequently, we have used only the CMR model to predict ETS constituent concentrations in motor vehicles, equation (5.4).

A key limitation in applying the CMR model is that ventilation rates in motor vehicles are not well known. The available data shows that air-exchange rates are high, relative to buildings, and also highly variable, depending primarily on vehicle speed, whether vents are open or closed, and whether or not windows are open.

For all four scenarios, we selected vehicle ventilation rates from a lognormal distribution GM of 104 $m^3\ h^{-1}$ and a GSD of 3.1. These parameters are based on an evaluation of experimental data in which vehicle air-exchange rates were measured under a range of conditions

using tracer gases (Rodes et al., 1998). In this study, measurements were made in three vehicles (1991 Caprice, 1997 Taurus, and 1997 Explorer) under a range of driving speeds (0-55 miles per hour), ventilation system conditions (vent open or closed, low fan speed, and windows partly open or closed). A total of 15 conditions were tested. We converted the air-exchange rates to ventilation rates by multiplying individual experimental results by estimated vehicle volumes (2.87 m^3 for the Taurus, 3.04 m^3 for the Caprice, and 3.79 m^3 for the Explorer). Then the GM and GSD of the ventilation rates were computed, yielding the results cited above.

This distribution is consistent with limited data contained in papers by Peterson and Sabersky (1975), Ott et al. (1992), and Park et al. (1996). The latter two papers report volumes for four vehicles in the range 2.4–3.7 m^3. All three papers report air-exchange rates based on tracer-gas decay that, in aggregate, vary from a 1-3 per hour in a stationary car with the windows closed (Park et al., 1996) to 120 per hour with the windows down and the car moving at 20 miles per hour (Ott et al., 1992). Peterson and Sabersky (1975) report rates in the range of 18–40 air changes per hour in a closed vehicle with the air conditioning operating, and the vehicle speed in the range 0–65 miles per hour.

In our Monte-Carlo simulations of exposure, we enforced a minimum ventilation rate of 3 $m^3\ h^{-1}$, based on the results of Park et al. for a stationary vehicle.

We assumed that there is no more than one smoker in the vehicle (n = 1). The rate of cigarette consumption, N_t, was selected randomly from the lognormal distribution derived from the APCR data (GM = 11.8 cigarettes per smoker per day divided by 16 waking hours per day = 0.74 cig $smoker^{-1}\ h^{-1}$ and GSD = 2.35). The emission factor for benzene, e, is selected from a normal distribution with AM ± SD = 406 ± 71 $\mu g\ cig^{-1}$ (Daisey et al., 1998), as in the other applications of the CMR method.

5.1.3.8 Residential Guest

Exposure in "other's homes" is a potentially significant contributor to total exposure. For example, based on self-reported proximity, this location constitutes about 7%, 22%, and 11% of the total amount of time nonsmoking adults, adolescents, and children spend in the presence of ETS. To estimate exposure for this microenvironment we used the tracer method with the same concentration distributions as we used for residential exposures (see §5.1.3.1.2). This method was used for all four scenarios, with the TM parameters being applied for scenario CM. The exposure period was taken to be the self-reported proximity time.

5.2 Results

The central objective of this phase of our research was to estimate the ETS contribution to air toxicant exposures for nonsmoking Californians, and this section summarizes our findings.

Exposure is reported as the time integral of exposure concentration, on a daily basis, in units of μg h m^{-3}. The average exposure concentration (including exposed and nonexposed periods) may be obtained by dividing daily exposure by 24 h.

5.2.1 Toxic Air Contaminant Exposure from ETS

5.2.1.1 Total Exposure

Tables 5.3-5.5 present summary statistics of the probability distribution functions for exposure to all toxic air contaminants studied. Each scenario and each population age group is considered separately. The statistics displayed in these tables apply to the part of the nonsmoking population that is exposed to ETS on a given day in the microenvironments studied (52% for adults, 63% for adolescents, and 33% for children; see Figure 3.4). Figure 5.1 displays similar information in a graphical format, showing for each population group and scenario the arithmetic mean plus selected percentiles for the distributions of exposure to benzene from ETS. For those exposed, the average contribution of ETS to benzene exposure is in the range 9-31 μg h m^{-3} for adults, 9-20 μg h m^{-3} for adolescents, and 12-24 μg h m^{-3} for children. The corresponding ranges for the 90th percentiles of each distribution are 24-71 μg h m^{-3}, 22-44 μg h m^{-3}, and 30-57 μg h m^{-3}.

The cumulative distributions of exposure to benzene from ETS for the exposed nonsmoking California population are shown in Figure 5.2, again segregated by age group and scenario. The coordinate axes are constructed so that a lognormal distribution would appear as a straight line. The distributions conform approximately to lognormality, but all exhibit a bowing downward such that the best-fit lognormal distribution tends to overpredict the high-percentile concentrations and underpredict the median.

Differences among the four scenarios are smaller than the variability within each scenario. For example, based on the arithmetic means, ETS exposure for the four different scenarios agree to within a factor of approximately 2-3 for each age group. Furthermore, and despite the fact that they are based on substantially independent estimates of microenvironmental concentrations, scenarios CM and TM agree very closely, especially for adolescents and children. On the other hand, the ratio of 90th percentile to 10th percentile of exposure within a given scenario ranges from a minimum of 30 (CM, adult) to a maximum of about 225 (TL, adolescents). These observations indicate that the variability in exposure among members of the public is large compared to the uncertainty in estimating the central tendency of exposure.

Table 5.3. Statistical parameters for total daily exposure of the California adult nonsmoking population to toxic air contaminants from environmental tobacco smoke, late 1980's. [1,2]

species/ scenario	*AM* ($\mu g\ h\ m^{-3}$)	*SD* ($\mu g\ h\ m^{-3}$)	*GM* ($\mu g\ h\ m^{-3}$)	*GSD*
acetaldehyde				
TL	49	120	12	8.4
TM	85	180	29	5.4
CM	101	120	53	3.8
TH	160	200	79	4.1
acetonitrile [3]				
TL	38	94	9	8.4
TM	65	140	22	5.4
CM	78	90	41	3.8
TH	130	160	61	4.1
acrylonitrile				
TL	2.3	5.6	0.5	8.4
TM	3.9	8.3	1.3	5.4
CM	4.6	5.4	2.4	3.8
TH	7.6	9.3	3.7	4.1
benzene				
TL	9.3	23	2.2	8.4
TM	16	34	5.5	5.4
CM	19	22	10	3.8
TH	31	38	15	4.1
1,3-butadiene				
TL	3.5	8.6	0.8	8.4
TM	6.0	13	2.1	5.4
CM	7.1	8.2	3.7	3.8
TH	12	14	5.6	4.1
2-butanone				
TL	6.7	17	1.6	8.4
TM	12	24	3.9	5.4
CM	14	16	7.2	3.8
TH	22	27	10.8	4.1
o-cresol				
TL	0.80	2.0	0.19	8.4
TM	1.4	2.9	0.47	5.4
CM	1.6	1.9	0.86	3.8
TH	2.7	3.3	1.3	4.1
m,p-cresol				
TL	1.9	4.7	0.45	8.4
TM	3.3	7.0	1.1	5.4
CM	3.9	4.5	2.0	3.8
TH	6.3	7.8	3.1	4.1

[1] AM–arithmetic mean, SD–arithmetic standard deviation, GM–geometric mean, GSD–geometric standard deviation; TL - tracer low exposure, TM - tracer mid-range exposure, CM - completely mixed room model, TH - tracer high exposure.

[2] Results apply to the proportion of the adult nonsmoking population in California that report some exposure during a day to ETS.

[3] Parameters estimated based on the ratio of emission factors for acetonitrile (1145 $\mu g\ cig^{-1}$) to benzene (280 $\mu g\ cig^{-1}$) reported by Martin et al., 1997; acetonitrile emissions not measured by Daisey et al.

Table 5.3. (continued)

species/ scenario	AM ($\mu g\ h\ m^{-3}$)	SD ($\mu g\ h\ m^{-3}$)	GM ($\mu g\ h\ m^{-3}$)	GSD
ethyl acrylate				
TL	< 0.07	—	< 0.02	—
TM	< 0.12	—	< 0.04	—
CM	< 0.14	—	< 0.07	—
TH	< 0.23	—	< 0.11	—
ethylbenzene				
TL	3.0	7.4	0.7	8.4
TM	5.1	10.9	1.8	5.4
CM	6.1	7.0	3.2	3.8
TH	9.9	12	4.8	4.1
formaldehyde				
TL	30	74	7	8.4
TM	52	110	18	5.4
CM	61	71	32	3.8
TH	100	120	48	4.1
n-nitrosodimethylamine				
TL	0.013	0.032	0.003	8.4
TM	0.022	0.048	0.008	5.4
CM	0.027	0.031	0.014	3.8
TH	0.044	0.053	0.021	4.1
phenol				
TL	6.4	16	1.5	8.4
TM	11	24	3.8	5.4
CM	13	15	6.9	3.8
TH	22	26	10.4	4.1
styrene				
TL	3.4	8.3	0.8	8.4
TM	5.8	12	2.0	5.4
CM	6.9	8.0	3.6	3.8
TH	11	14	5.4	4.1
toluene				
TL	15	37	3.6	8.4
TM	26	55	8.9	5.4
CM	31	36	16	3.8
TH	50	61	24	4.1
o-xylene				
TL	1.5	3.8	0.36	8.4
TM	2.6	5.6	0.91	5.4
CM	3.1	3.6	1.7	3.8
TH	5.1	6.3	2.5	4.1
m,p-xylene				
TL	6.8	17	1.6	8.4
TM	12	25	4.1	5.4
CM	14	16	7.4	3.8
TH	23	28	11	4.1

Table 5.4. Statistical parameters for daily exposure of Californian adolescent nonsmokers to toxic air contaminants from environmental tobacco smoke, late 1980's. [1, 2]

species/ scenario	*AM* ($\mu g\ h\ m^{-3}$)	*SD* ($\mu g\ h\ m^{-3}$)	*GM* ($\mu g\ h\ m^{-3}$)	*GSD*
acetaldehyde				
TL	47	79	12	8.9
TM	69	101	30	4.8
CM	69	90	34	4.1
TH	106	120	53	4.0
acetonitrile [3]				
TL	36	61	9	8.9
TM	53	78	23	4.8
CM	53	70	26	4.1
TH	82	90	41	4.0
acrylonitrile				
TL	2.2	3.7	0.5	8.9
TM	3.2	4.6	1.4	4.8
CM	3.2	4.1	1.6	4.1
TH	4.9	5.4	2.4	4.0
benzene				
TL	8.9	15	2.2	8.9
TM	13	19	5.7	4.8
CM	13	17	6.4	4.1
TH	20	22	10.0	4.0
1,3-butadiene				
TL	3.3	5.6	0.8	8.9
TM	4.9	7.1	2.1	4.8
CM	4.9	6.4	2.4	4.1
TH	7.5	8.2	3.7	4.0
2-butanone				
TL	6.4	10.8	1.6	8.9
TM	9.3	14	4.1	4.8
CM	9.3	12	4.6	4.1
TH	14.3	16	7.2	4.0
o-cresol				
TL	0.77	1.3	0.19	8.9
TM	1.1	1.6	0.49	4.8
CM	1.1	1.5	0.55	4.1
TH	1.7	1.9	0.86	4.0
m,p-cresol				
TL	1.8	3.1	0.45	8.9
TM	2.7	3.9	1.2	4.8
CM	2.7	3.5	1.3	4.1
TH	4.1	4.5	2.0	4.0

[1] AM–arithmetic mean, SD–arithmetic standard deviation, GM–geometric mean, GSD–geometric standard deviation; TL - tracer low exposure, TM - tracer mid-range exposure, CM - completely mixed room model, TH - tracer high exposure.

[2] Results apply to the proportion of the adolescent nonsmoking population in California that report some exposure during a day to ETS.

[3] Parameters estimated based on the ratio of emission factors for acetonitrile (1145 $\mu g\ cig^{-1}$) to benzene (280 $\mu g\ cig^{-1}$) reported by Martin et al., 1997; acetonitrile emissions not measured by Daisey et al.

Table 5.4. (continued)

species/ scenario	*AM* ($\mu g\ h\ m^{-3}$)	*SD* ($\mu g\ h\ m^{-3}$)	*GM* ($\mu g\ h\ m^{-3}$)	*GSD*
ethyl acrylate				
TL	< 0.07	—	< 0.02	—
TM	< 0.10	—	< 0.04	—
CM	< 0.10	—	< 0.05	—
TH	< 0.15	—	< 0.07	—
ethylbenzene				
TL	2.8	4.8	0.7	8.9
TM	4.2	6.1	1.8	4.8
CM	4.2	5.4	2.0	4.1
TH	6.4	7.0	3.2	4.0
formaldehyde				
TL	29	48	7	8.9
TM	42	61	18	4.8
CM	42	55	21	4.1
TH	65	71	32	4.0
n-nitrosodimethylamine				
TL	0.012	0.021	0.003	8.9
TM	0.018	0.027	0.008	4.8
CM	0.018	0.024	0.009	4.1
TH	0.028	0.031	0.014	4.0
phenol				
TL	6.2	10	1.5	8.9
TM	9.0	13	3.9	4.8
CM	9.0	12	4.4	4.1
TH	14	15	6.9	4.0
styrene				
TL	3.2	5.4	0.8	8.9
TM	4.7	6.9	2.1	4.8
CM	4.7	6.2	2.3	4.1
TH	7.2	8.0	3.6	4.0
toluene				
TL	14	24	3.6	8.9
TM	21	31	9.2	4.8
CM	21	28	10.3	4.1
TH	32	36	16	4.0
o-xylene				
TL	1.7	2.5	0.36	8.9
TM	2.2	3.1	0.94	4.8
CM	2.2	2.8	1.06	4.1
TH	3.3	3.6	1.7	4.0
m,p-xylene				
TL	6.6	11	1.6	8.9
TM	9.6	14	4.2	4.8
CM	9.6	13	4.7	4.1
TH	15	16	7.4	4.0

Table 5.5. Statistical parameters for total daily exposure of the California children population to toxic air contaminants from environmental tobacco smoke, late 1980's. [1,2]

species/ scenario	*AM* ($\mu g\ h\ m^{-3}$)	*SD* ($\mu g\ h\ m^{-3}$)	*GM* ($\mu g\ h\ m^{-3}$)	*GSD*
acetaldehyde				
TL	64	110	21	7.4
TM	90	130	38	5.1
CM	95	150	40	4.5
TH	130	150	64	4.8
acetonitrile [3]				
TL	49	86	16	7.4
TM	70	98	29	5.1
CM	74	120	31	4.5
TH	98	120	49	4.8
acrylonitrile				
TL	2.9	5.1	1.0	7.4
TM	4.1	5.9	1.8	5.1
CM	4.4	6.8	1.9	4.5
TH	5.9	6.8	2.9	4.8
benzene				
TL	12	21	3.9	7.4
TM	17	24	7.2	5.1
CM	18	28	7.6	4.5
TH	24	28	12	4.8
1,3-butadiene				
TL	4.5	7.9	1.5	7.4
TM	6.4	9.0	2.7	5.1
CM	6.7	10.5	2.8	4.5
TH	9.0	10.5	4.5	4.8
2-butanone				
TL	8.6	15	2.8	7.4
TM	12	17	5.2	5.1
CM	13	20	5.4	4.5
TH	17	20	8.6	4.8
o-cresol				
TL	1.03	1.8	0.34	7.4
TM	1.5	2.1	0.62	5.1
CM	1.6	2.4	0.66	4.5
TH	2.1	2.4	1.03	4.8
m,p-cresol				
TL	2.5	4.3	0.80	7.4
TM	3.5	4.9	1.5	5.1
CM	3.7	5.7	1.6	4.5
TH	4.9	5.7	2.5	4.8

[1] AM–arithmetic mean, SD–arithmetic standard deviation, GM–geometric mean, GSD–geometric standard deviation; TL - tracer low exposure, TM - tracer mid-range exposure, CM - completely mixed room model, TH - tracer high exposure.

[2] Results apply to the proportion of children in California for whom some exposure to ETS is reported during a day.

[3] Parameters estimated based on the ratio of emission factors for acetonitrile (1145 $\mu g\ cig^{-1}$) to benzene (280 $\mu g\ cig^{-1}$) reported by Martin et al., 1997; acetonitrile emissions not measured by Daisey et al.

Table 5.5. (continued)

species/ scenario	*AM* ($\mu g\ h\ m^{-3}$)	*SD* ($\mu g\ h\ m^{-3}$)	*GM* ($\mu g\ h\ m^{-3}$)	*GSD*
ethyl acrylate				
TL	< 0.09	—	< 0.03	—
TM	< 0.13	—	< 0.05	—
CM	< 0.13	—	< 0.06	—
TH	< 0.18	—	< 0.09	—
ethylbenzene				
TL	3.8	6.7	1.2	7.4
TM	5.4	7.7	2.3	5.1
CM	5.8	9.0	2.4	4.5
TH	7.7	9.0	3.8	4.8
formaldehyde				
TL	39	68	13	7.4
TM	55	77	23	5.1
CM	58	90	25	4.5
TH	77	90	39	4.8
n-nitrosodimethylamine				
TL	0.017	0.029	0.005	7.4
TM	0.024	0.034	0.010	5.1
CM	0.025	0.039	0.011	4.5
TH	0.034	0.039	0.017	4.8
phenol				
TL	8.3	15	2.7	7.4
TM	12	17	5.0	5.1
CM	13	19	5.3	4.5
TH	17	19	8.3	4.8
styrene				
TL	4.3	7.6	1.4	7.4
TM	6.2	8.7	2.6	5.1
CM	6.5	10.1	2.8	4.5
TH	8.7	10.1	4.3	4.8
toluene				
TL	19	34	6.3	7.4
TM	28	39	12	5.1
CM	29	45	12	4.5
TH	39	45	19	4.8
o-xylene				
TL	2.0	3.5	0.64	7.4
TM	2.8	4.0	1.2	5.1
CM	3.0	4.6	1.3	4.5
TH	4.0	4.6	2.0	4.8
m,p-xylene				
TL	8.8	16	2.9	7.4
TM	13	18	5.3	5.1
CM	13	21	5.6	4.5
TH	18	21	8.8	4.8

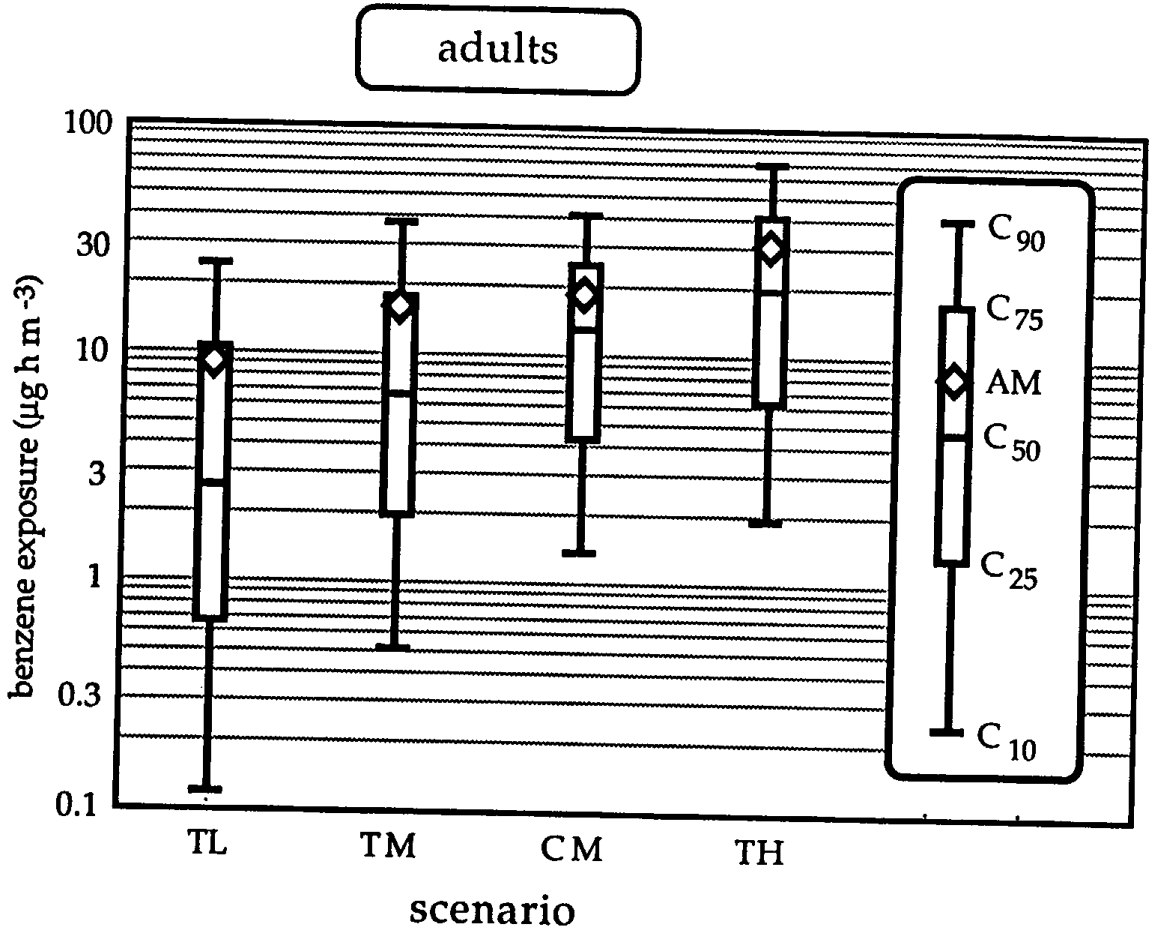

Figure 5.1a A whisker diagram summarizing distributional results for total exposure to benzene from ETS for nonsmoking Californian adults (late 1980's) according to four modeling scenarios. (Scenarios: TL - tracer low-range, TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range. AM is the arithmetic mean and C_i represents the i^{th} percentile of the distribution.)

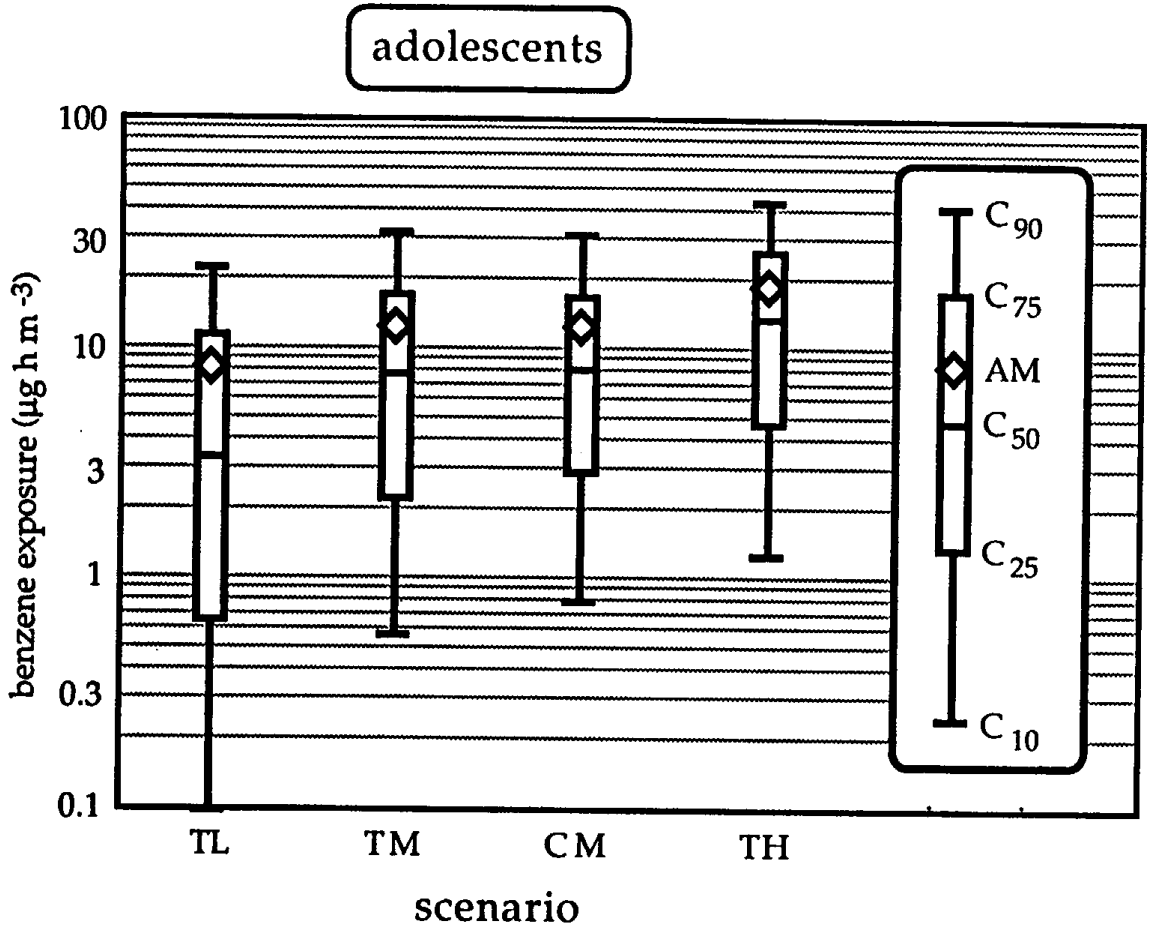

Figure 5.1b A whisker diagram summarizing distributional results for total exposure to benzene from ETS for nonsmoking Californian adolescents (late 1980's) according to four modeling scenarios. (Scenarios: TL - tracer low-range, TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range. AM is the arithmetic mean and C_i represents the i^{th} percentile of the distribution.)

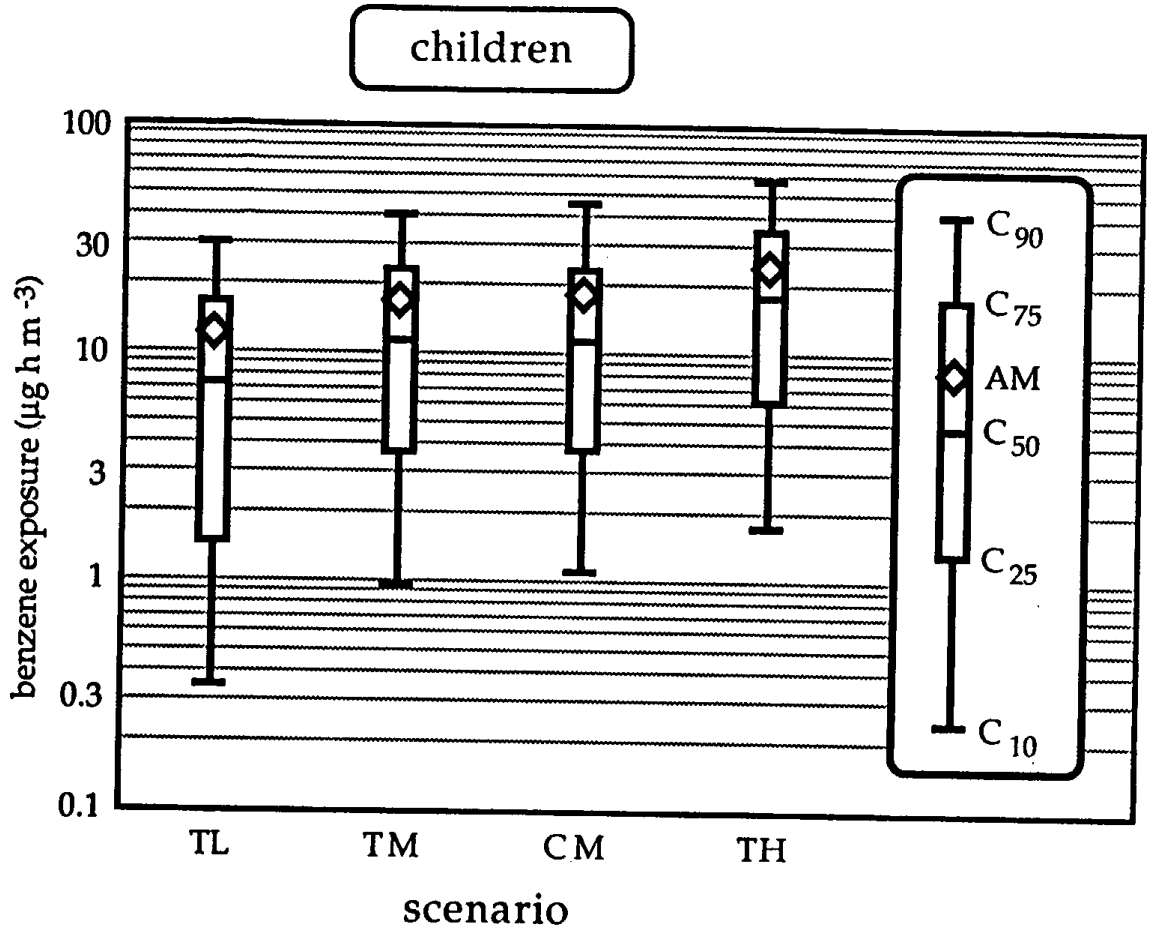

Figure 5.1c A whisker diagram summarizing distributional results for total exposure to benzene from ETS for nonsmoking Californian children (late 1980's) according to four modeling scenarios. (Scenarios: TL - tracer low-range, TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range. AM is the arithmetic mean and C_i represents the i^{th} percentile of the distribution.)

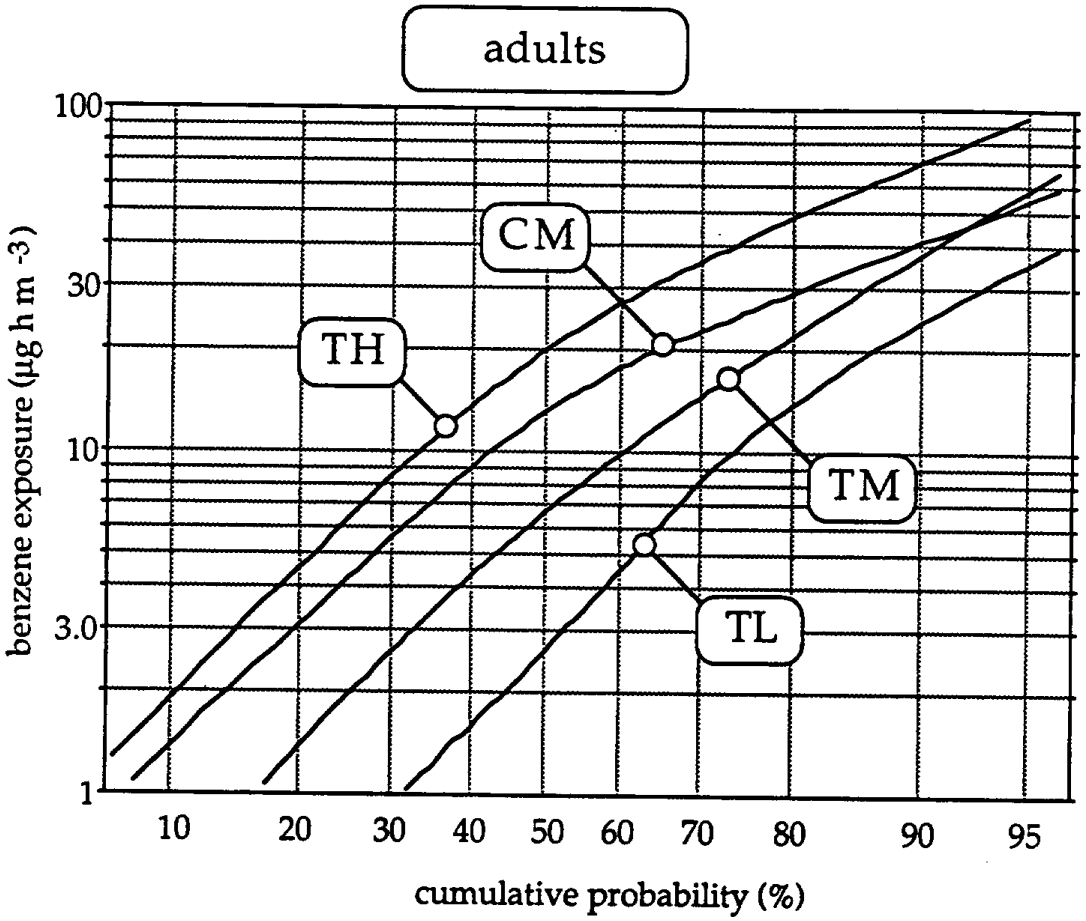

Figure 5.2a Lognormal-probability plot of the distributions of total exposure (late 1980's) to benzene from ETS, separated by scenario, for nonsmoking adults. (Scenarios: TL - tracer low-range, TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range.)

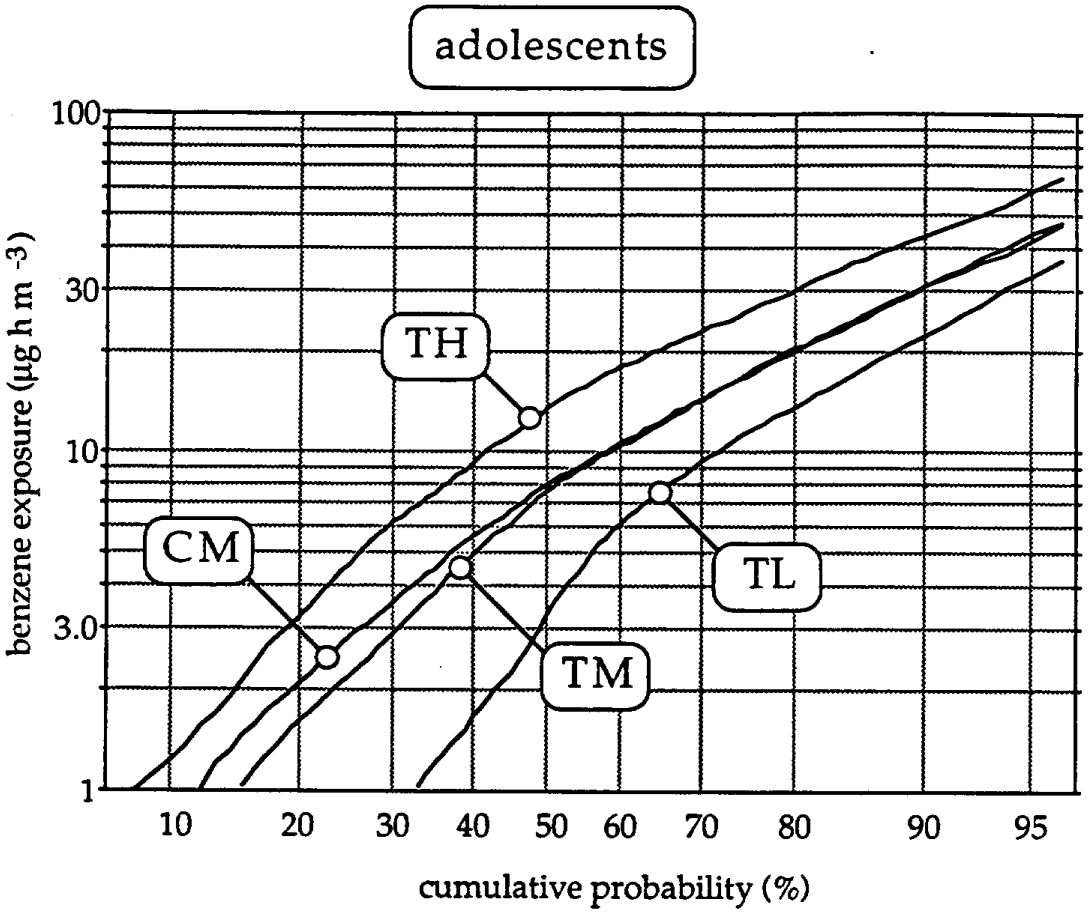

Figure 5.2b Lognormal-probability plot of the distributions of total exposure (late 1980's) to benzene from ETS, separated by scenario, for nonsmoking adolescents. (Scenarios: TL - tracer low-range, TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range.)

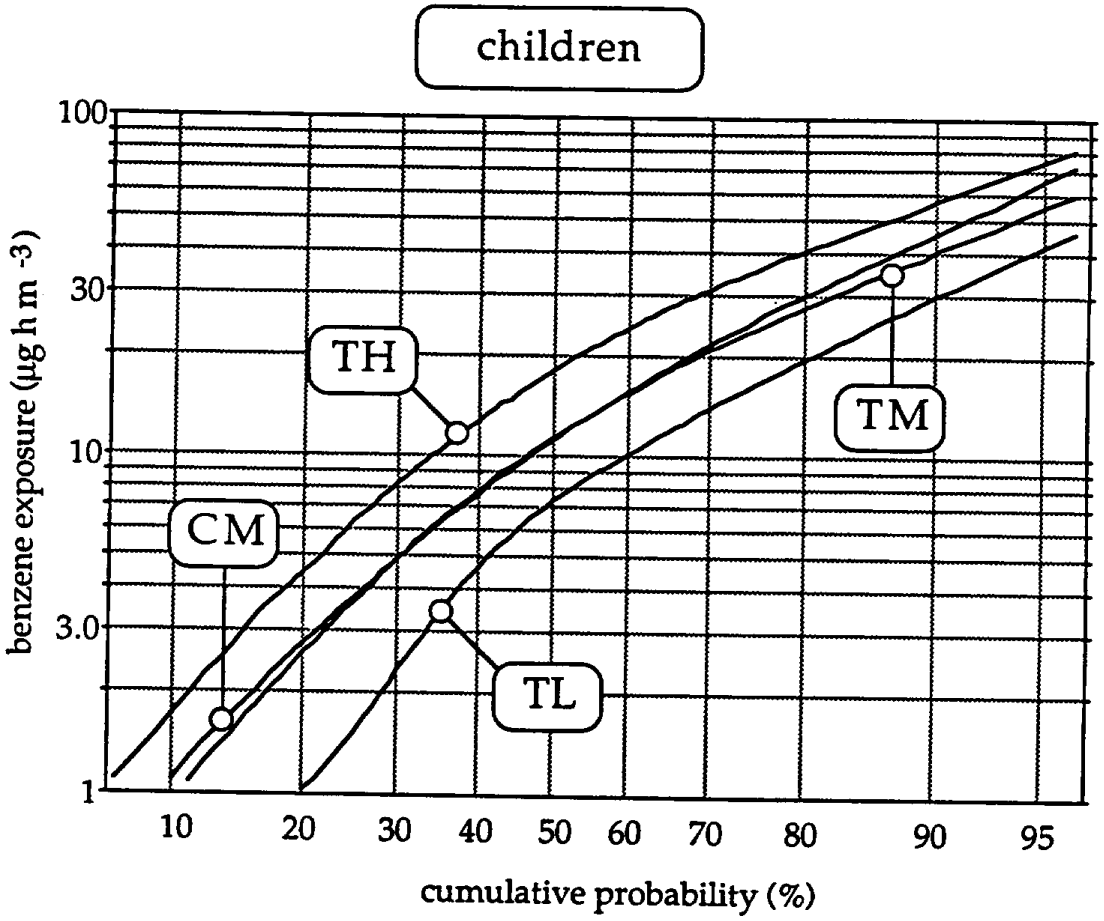

Figure 5.2c Lognormal-probability plot of the distributions of total exposure (late 1980's) to benzene from ETS, separated by scenario, for children. (Scenarios: TL - tracer low-range, TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range.)

5.2.1.2 Contributions of Microenvironments

Figures 5.3-5.5 show the apportionment of the arithmetic mean (AM) exposure to ETS among different microenvironments. Although the mean total exposures for exposed adults, adolescents and children are similar, the locations where these exposures occur vary markedly.

For adults, significant ETS exposure occurs in many distinct environments, and no single microenvironment dominates. For all scenarios, personal residences and workplaces are the primary settings for exposure, contributing 58-61% to that total. The contributions to exposure in each of the remaining five microenvironments are potentially significant, ranging from a low of 4% for residential guest in three scenarios and transportation in scenario TH to a high of 15% for transportation in scenario TL. In general, these findings substantiate other published evidence concerning the importance of residential and occupational locations for adult ETS exposures. A study by Cummings et al. (1990) assessed ETS exposure for 663 never- and exsmokers using urinary cotinine measurements and questionnaires. A total of 76% of the subjects reported exposure to ETS over the course of the four previous days and the most frequently mentioned sources of exposure were at home—27%, and at work—28%. A study by Emmons et al. (1992) found that the primary source of ETS exposure was the workplace (50% compared to 10% in residence), except when there was a smoker in the household, in which case the household was the primary source.

For adolescents, the proportion of mean exposure that occurs in residences grows in importance. Summing "residential" and "residential guest," these environments contribute 62-74% of the total mean exposure (Figure 5.4). On the other hand, the average contribution of occupational exposure is negligible for this population group. The microenvironment groups "retail/other" and "transportation" are also significant contributors, adding 4-15% and 8-18% of total mean exposure, respectively.

The results for children (Figure 5.5) show strong dominance of the residential microenvironments, especially one's own home. This single setting contributes 70-73% of the mean exposure to ETS of children. Transportation is a distant second in importance, adding 9-18%, and residential guest contributes 6-7%.

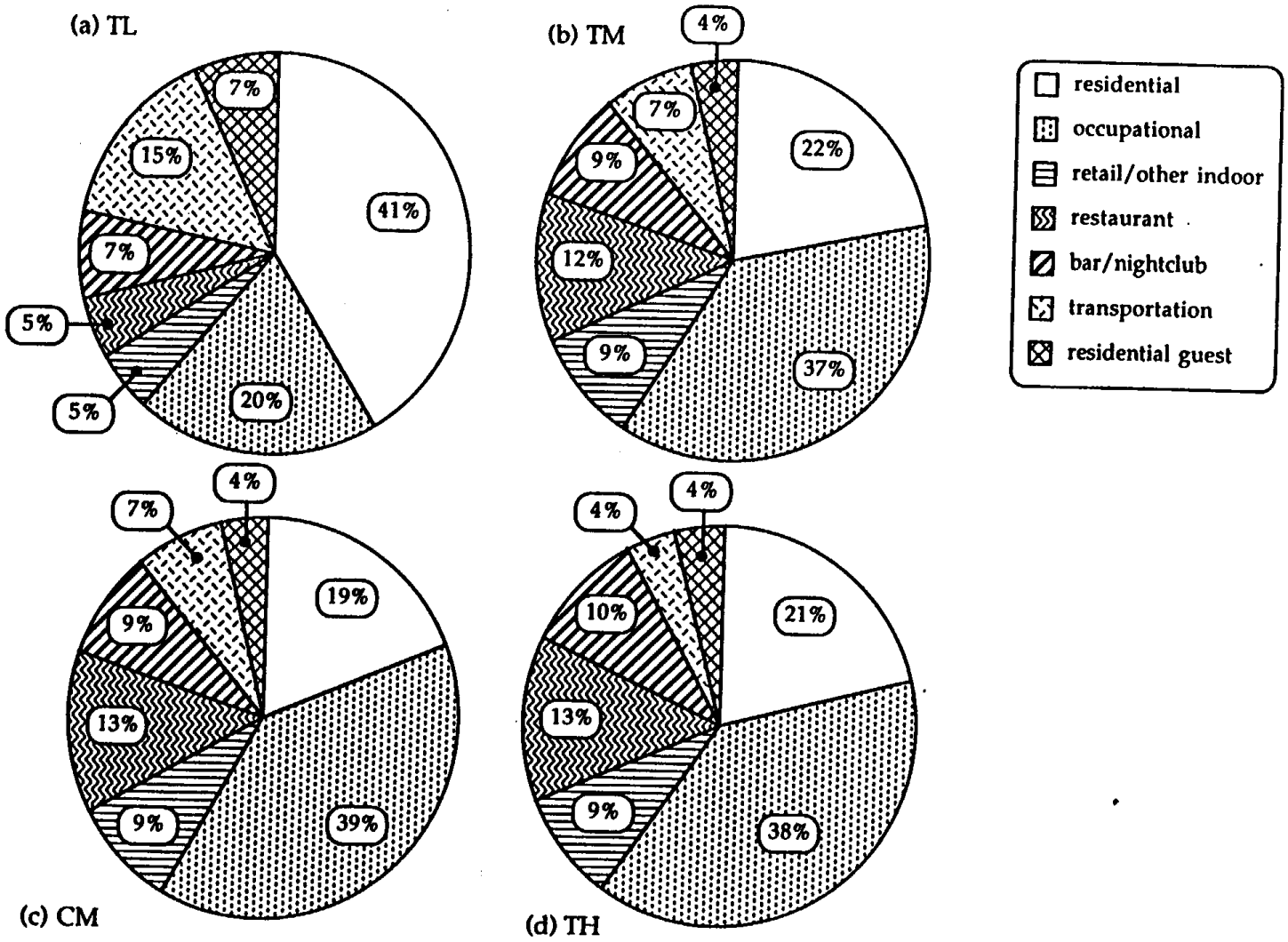

Figure 5.3 Proportion of arithmetic mean ETS exposure (late 1980's) among microenvironments for each of the four scenarios for adult nonsmokers: (a) TL (tracer low-range), (b) TM (tracer mid-range), (c) CM (completely mixed room model), and (d) TH (tracer high-range).

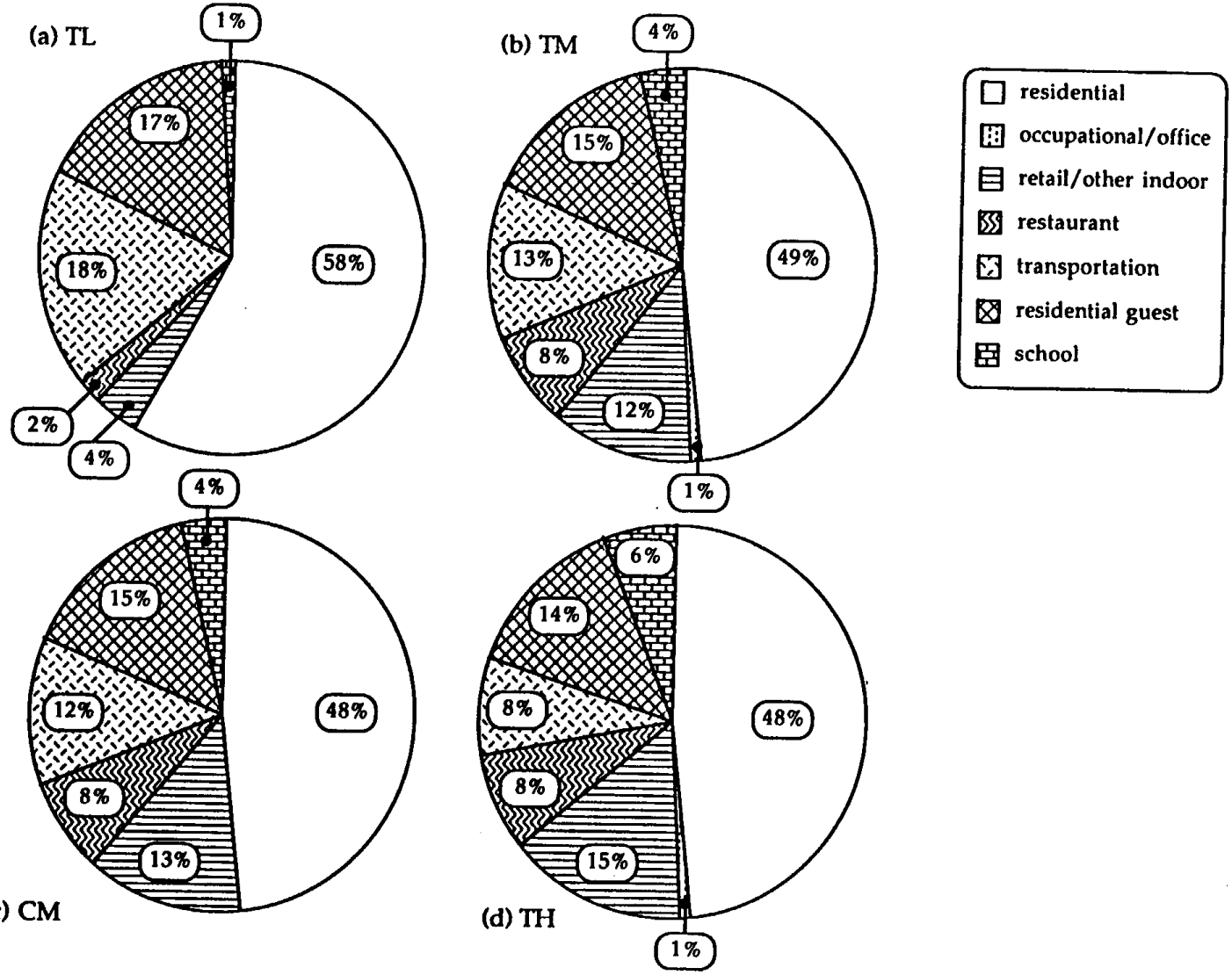

Figure 5.4 Proportion of arithmetic mean ETS exposure (late 1980's) among microenvironments for each of the four scenarios for adolescent nonsmokers: (a) TL (tracer low-range), (b) TM (tracer mid-range), (c) CM (completely mixed room model), and (d) TH (tracer high-range).

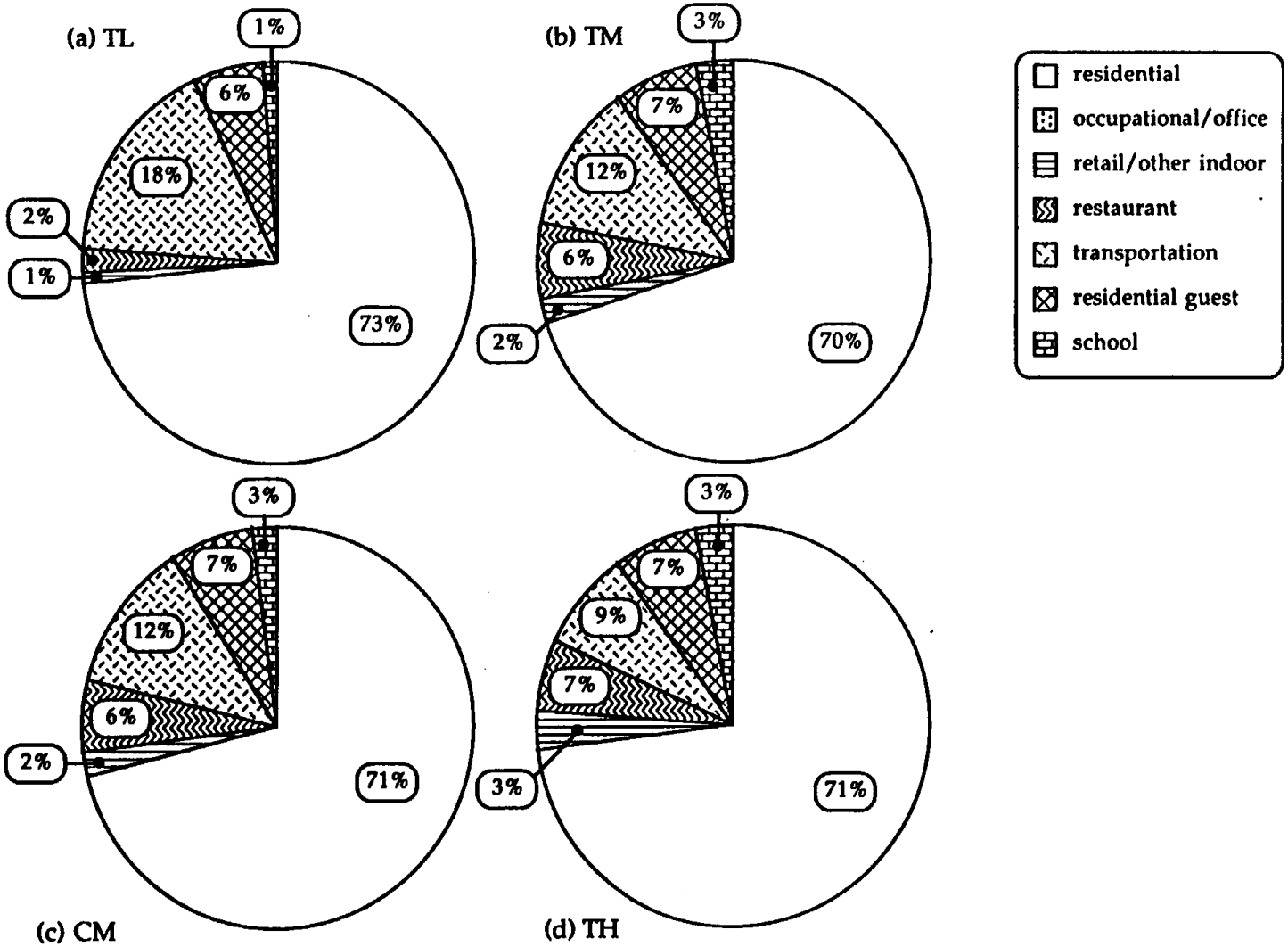

Figure 5.5 Proportion of arithmetic mean ETS exposure (late 1980's) among microenvironments for each of the four scenarios for children: **(a)** TL (tracer low-range), **(b)** TM (tracer mid-range), **(c)** CM (completely mixed room model), and **(d)** TH (tracer high-range).

Tables 5.6-5.8 and Figure 5.6 present greater detail on the contribution of each microenvironment to exposure. The tables present summary statistics for the distribution of exposures to benzene from ETS in each microenvironment. The results in this table only apply to those exposed in that particular setting, with the percentages of the nonsmoking population so exposed shown in Figure 3.3. For adults, Table 5.6 shows that for all scenarios and microenvironments, the arithmetic mean contributions to exposure (when such exposures occur) are contained within a fairly narrow range, from 1.7 $\mu g\ h\ m^{-3}$ for retail/other in scenario TL to 47 $\mu g\ h\ m^{-3}$ for bars/nightclubs in scenario TH. Uncertainty in the central estimates is indicated by the change in AM values within a single microenvironment across scenarios. Table 5.6 shows that the ratio of the maximum to minimum scenario means ranges from a factor of 2 for residential exposure to a factor of 8 for restaurants. Although substantial, this uncertainty is smaller than the variability within a microenvironment. For a lognormal distribution, the ratio of the 95th to 5th percentile concentrations is given by $GSD^{3.3}$. Table 5.6 shows that the GSD for residential exposures is in the range 2.1-2.5, which suggests a variability for the central 90% of the distribution in the range 12–20. For occupational exposures, the GSD range is 2.8-7.3, indicating variability ranging from 30 to 700. (The one very high GSD value for offices derives from the results of Jenkins et al. (1996).) For adolescents and children, focusing on residences, similar observations apply. The range of arithmetic means among the four scenarios spans a factor of 2. The variability, estimated as $GSD^{3.3}$, is in the range 16-34. Focusing on scenario CM, Figure 5.6 depicts information about the distribution of exposure to benzene from ETS in each microenvironment.

5.2.2 Mean Exposure for All Nonsmokers to Toxic Air Contaminants from ETS

Using the arithmetic mean values for each scenario, we estimated the contribution of ETS to the average exposure of nonsmoking Californians to seventeen air toxicants. Table 5.9 presents these results, along with a summary of results from Phase I. In each case, to estimate mean exposure for the entire nonsmoking population, we multiplied the estimates of mean exposure for those exposed by the fraction of the nonsmoking population that was exposed in each evaluation. This approach assumes that the contribution to exposure is negligible for those who do not report being in the proximity of a smoker during the day. The range of results is defined by scenario TL at the low end and scenario TH at the high end; scenarios CM and TM produce intermediate values.

Table 5.6. Statistical parameters for daily exposure of the California adult nonsmoking population to benzene from environmental tobacco smoke in different microenvironments, late 1980's.[1,2]

microenvironment	AM ($\mu g\ h\ m^{-3}$)	SD ($\mu g\ h\ m^{-3}$)	GM ($\mu g\ h\ m^{-3}$)	GSD
residential				
tracer method				
low exposure	17	17	12	2.4
mid-range	20	18	15	2.3
high exposure	30	22	23	2.1
CMR method[3]				
mid-range	16	15	11	2.5
occupational				
tracer method				
low exposure	4.9	27	0.84	7.3
mid-range	20	41	8.1	4.4
high exposure	32	40	16	3.9
CMR method[3]				
mid-range	20	14	14	2.8
retail/other indoor				
tracer method				
low exposure	1.7	14	0.12	11
mid-range	6.9	24	1.5	6.4
high exposure	11	13	5.8	3.6
CMR method[3]				
mid-range	6.9	9.5	3.1	3.9
restaurant				
tracer method				
low exposure	1.8	2.0	1.1	2.7
mid-range	8.7	14	4.4	3.4
high exposure	14	27	5.9	3.9
bar/nightclub				
tracer method				
low exposure	9.4	10	5.9	2.7
mid-range	26	39	13	3.2
high exposure	47	48	31	2.6
transportation				
CMR method[3]				
mid-range	9.0	23	2.8	5.1
residential guest				
tracer method				
low exposure	4.4	8.0	1.7	4.6
mid-range	5.5	9.8	2.0	4.7
high exposure	8.1	12	3.5	4.1

[1] AM–arithmetic mean, SD–arithmetic standard deviation, GM–geometric mean, GSD–geometric standard deviation.

[2] Results apply to that portion of the adult nonsmoking population in California that report some exposure in that microenvironment during a day (see Figure 3.3).

[3] CMR - completely mixed room model.

Table 5.7. Statistical parameters for daily exposure of the California adolescent nonsmoking population to benzene from environmental tobacco smoke in different microenvironments, late 1980's.[1,2]

microenvironment	AM (μg h m^{-3})	SD (μg h m^{-3})	GM (μg h m^{-3})	GSD
residential				
tracer method				
low exposure	14	17	8.2	2.9
mid-range	17	21	11	2.6
high exposure	26	26	18	2.4
CMR method [3]				
mid-range	17	20	11	2.7
occupational/office				
tracer method				
low exposure	0.90	2.9	0.094	9.7
mid-range	3.9	7.8	0.92	6.1
high exposure	7.8	12	2.7	4.8
CMR method [3]				
mid-range	3.6	4.5	1.4	4.4
retail/other indoor				
tracer method				
low exposure	1.1	5.8	0.082	13
mid-range	5.2	13	1.04	8.4
high exposure	10.0	9.2	4.1	5.5
CMR method [3]				
mid-range	5.8	6.8	2.1	5.9
restaurant				
tracer method				
low exposure	0.69	0.52	0.48	2.0
mid-range	3.3	4.4	2.0	2.7
high exposure	5.4	9.4	2.7	3.2
school				
tracer method				
low exposure	0.53	2.7	0.056	9.5
mid-range	2.5	6.1	0.70	5.6
high exposure	5.3	7.9	2.3	4.2
CMR method [3]				
mid-range	2.6	3.9	1.1	4.4
transportation				
CMR method [3]				
mid-range	6.0	11	2.4	4.4
residential guest				
tracer method				
low exposure	4.3	10	1.8	3.6
mid-range	5.9	12	2.2	4.3
high exposure	8.7	15	3.9	3.8

[1] AM–arithmetic mean, SD–arithmetic standard deviation, GM–geometric mean, GSD–geometric standard deviation.
[2] Results apply to that portion of the adolescent nonsmoking population in California that report some exposure in that microenvironment during a day (see Figure 3.3).
[3] CMR - completely mixed room model.

Table 5.8. Statistical parameters for daily exposure of California children to benzene from environmental tobacco smoke in different microenvironments, late 1980's.[1,2]

microenvironment	AM ($\mu g\ h\ m^{-3}$)	SD ($\mu g\ h\ m^{-3}$)	GM ($\mu g\ h\ m^{-3}$)	GSD
residential				
tracer method				
low exposure	15	22	9.9	2.4
mid-range	20	24	14	2.5
high exposure	30	26	22	2.3
CMR method [3]				
mid-range	23	30	14	2.9
occupational/office				
tracer method				
low exposure	0.28	1.4	0.016	13
mid-range	1.1	2.9	0.21	6.9
high exposure	2.2	3.8	0.71	4.8
CMR method [3]				
mid-range	0.95	1.2	0.42	3.8
retail/other indoor				
tracer method				
low exposure	0.58	2.7	0.055	11
mid-range	2.8	6.7	0.70	6.4
high exposure	4.8	4.6	2.7	3.5
CMR method [3]				
mid-range	2.8	3.3	1.4	3.9
restaurant				
tracer method				
low exposure	1.2	1.0	0.83	2.3
mid-range	5.8	7.4	3.4	2.9
high exposure	9.3	15	4.5	3.4
school				
tracer method				
low exposure	5.1	25	0.47	8.0
mid-range	18	50	5.1	4.6
high exposure	32	55	16	3.4
CMR method [3]				
mid-range	17	27	7.9	3.5
transportation				
CMR method [3]				
mid-range	6.5	18	2.1	5.0
residential guest				
tracer method				
low exposure	3.4	5.9	1.3	4.7
mid-range	5.6	10	1.8	5.4
high exposure	8.2	13	3.0	4.8

[1] AM–arithmetic mean, SD–arithmetic standard deviation, GM–geometric mean, GSD–geometric standard deviation.

[2] Results apply to that portion of children in California that report some exposure in that microenvironment during a day (see Figure 3.3).

[3] CMR - completely mixed room model.

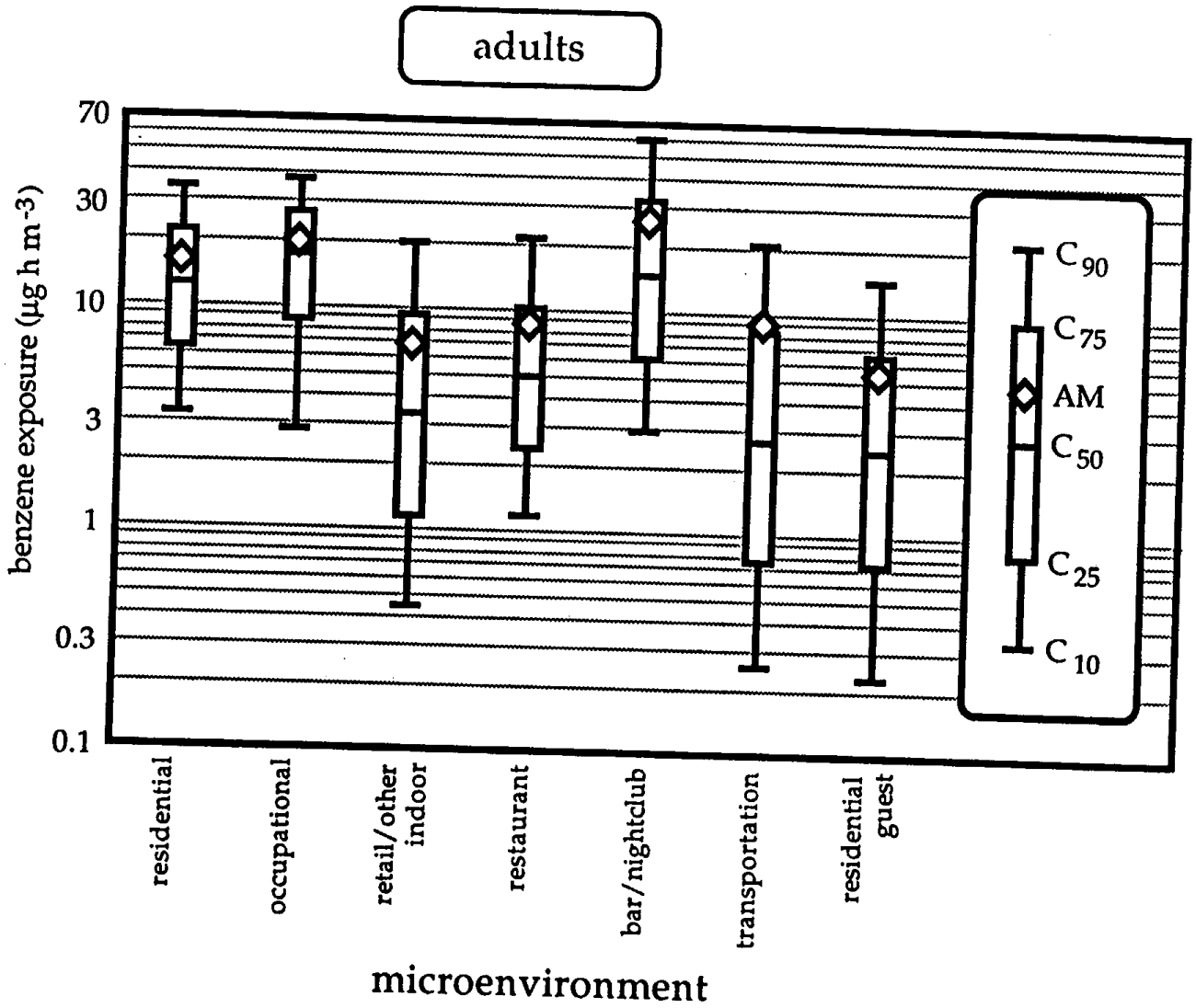

Figure 5.6a Whisker diagram for benzene exposure variability from ETS among microenvironments in scenario CM (completely mixed room model), late 1980's, for adults. (AM is the arithmetic mean and C_i represents the i^{th} percentile of the distribution.)

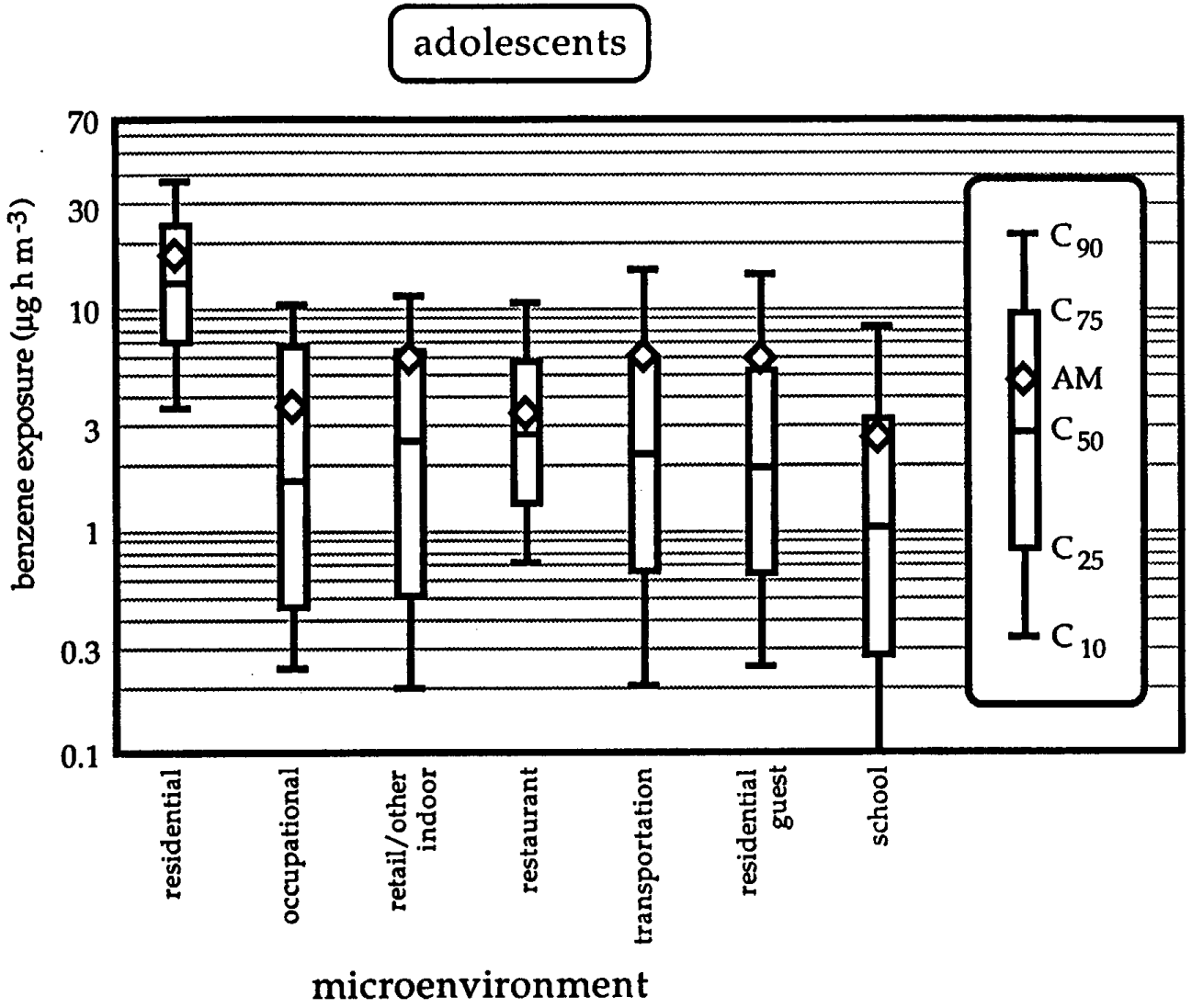

Figure 5.6b Whisker diagram for benzene exposure variability from ETS among microenvironments in scenario CM (completely mixed room model), late 1980's, for adolescents. (AM is the arithmetic mean and C_i represents the i^{th} percentile of the distribution.)

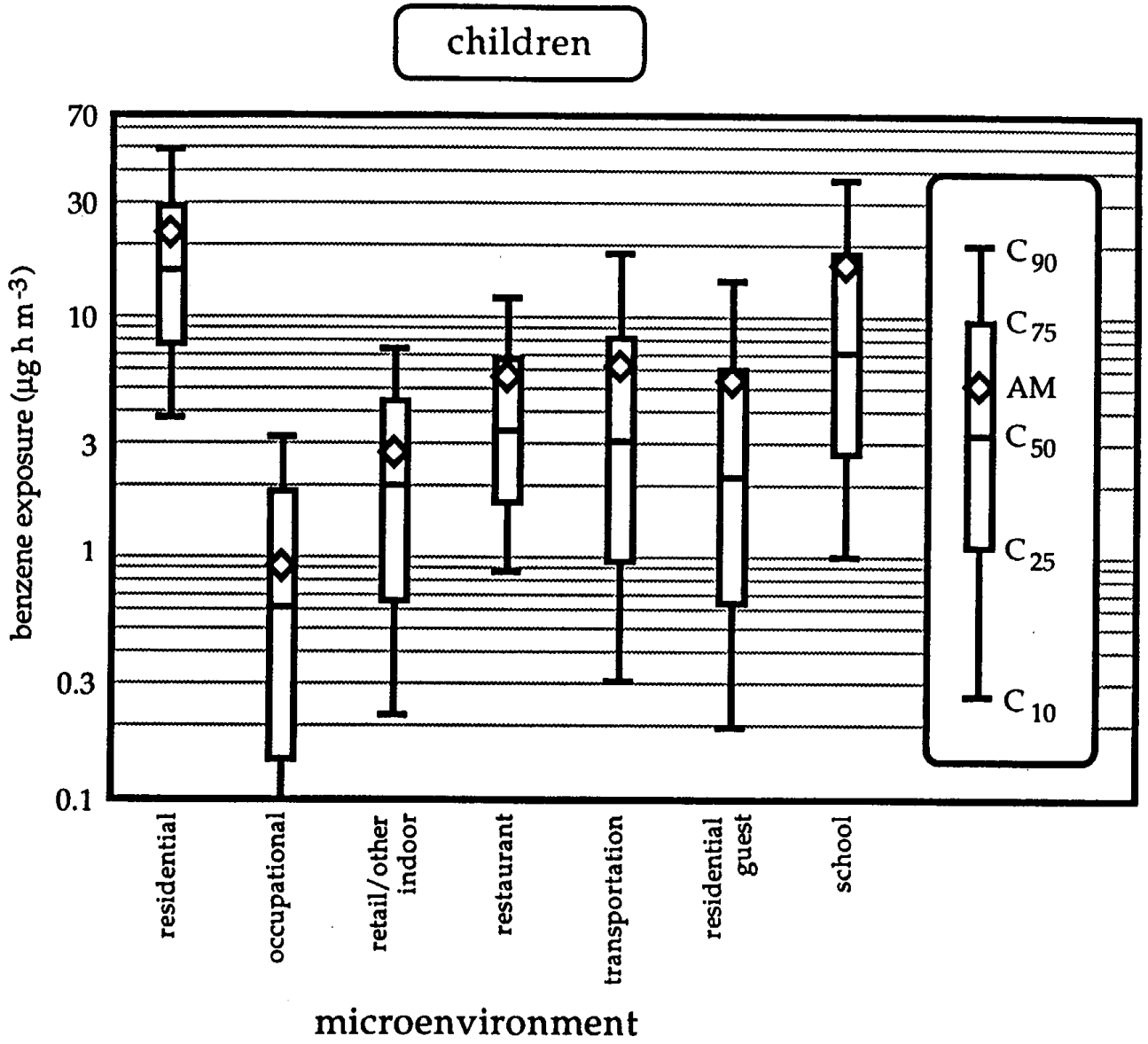

Figure 5.6c Whisker diagram for benzene exposure variability from ETS among microenvironments in scenario CM (completely mixed room model), late 1980's, for children. (AM is the arithmetic mean and C_i represents the i^{th} percentile of the distribution.)

Table 5.9. Comparison of Phase I and Phase II estimates of average daily exposure of all nonsmoking Californians to toxic air contaminants from ETS, late 1980's.

	ETS-only, daily exposure ($\mu g\ h\ m^{-3}$)			
		Phase II [2]		
compound	*Phase I* [1]	*adults*	*adolescents*	*children*
acetaldehyde	150-200	25-83	30-67	21-43
acetonitrile	94	20-68	23-52	16-32
acrylonitrile	6.5	1.2-4.0	1.4-3.1	1.0-1.9
benzene	14	4.8-16	5.6-13	4.0-7.9
1,3-butadiene	10-31	1.8-6.2	2.1-4.7	1.5-3.0
2-butanone (MEK)	19	3.5-11	4.0-9.0	2.8-5.6
o-cresol	2.4	0.4-1.4	0.5-1.1	0.3-0.7
m,p-cresol	5.5	1.0-3.3	1.1-2.6	0.8-1.6
ethyl acrylate	< 0.19	< 0.12	< 0.10	< 0.06
ethylbenzene	6.5-8.6	1.6-5.1	1.8-4.0	1.3-2.5
formaldehyde	86-110	16-52	18-41	13-25
n-nitrosodimethylamine	0.04	0.007-0.023	0.008-0.018	0.006-0.011
phenol	19	3.3-11	3.9-8.8	2.7-5.6
styrene	4.8	1.8-5.7	2.0-4.5	1.4-2.9
toluene	40-43	7.8-26	8.8-20	6.3-13
o-xylene	10.3	0.8-2.7	1.1-2.1	0.7-1.3
m,p-xylene	13	3.5-12	4.2-9.5	2.9-5.9

[1] Estimated by multiplying the ETS-only exposure concentrations in Table 4.6 by 24 h.
[2] Range of arithmetic means for scenarios TL, TM, CM, and TH; obtained by multiplying approrpriate values in Tables 5.3-5.5 by percentage of nonsmokers exposed to ETS on a daily basis in the simulated microenvironments (52% for adults, 63% for adolescents, and 33% for children).

Mean exposures of nonsmoking adolescents and children are estimated to be similar to the mean exposures of nonsmoking adults. For all children, the population mean exposure to air toxics from ETS ranges from 49% (scenario TH) to 82% (scenario CM) of that for all adult nonsmokers. Much of the reduction for children is accounted for by the lower percentage of the population that is exposed in the modeled microenvironments (33% for children vs. 52% for adults). For adolescents, the range of population mean exposures is 78% (scenario TH) to 116% (scenario TL) of that for adults. A higher proportion of adolescents are exposed than adults (63% vs. 52% in the modeled microenvironments), but the average exposure among those exposed is somewhat smaller for adolescents.

For benzene and styrene, the Phase I results lie well within the range of adult exposures predicted in Phase II. Total exposures to most other species are predicted by the Phase I method to be roughly a factor of two larger than the Phase II predictions for adults. The disagreement between the results of the two assessment methods is greatest for o-xylene, where the Phase I predictions are about an order of magnitude greater than those from Phase II. The Phase I and Phase II results are in general agreement, given the large uncertainties in the Phase I arithmetic means. For example, the Phase I 90% confidence bounds on the arithmetic mean exposure from ETS for the entire nonsmoking population are 3-90 $\mu g\ m^{-3}\ h$ for benzene, 0.8-12 $\mu g\ m^{-3}\ h$ for styrene, 2-30 $\mu g\ m^{-3}\ h$ for o-xylene, and 5-360 $\mu g\ m^{-3}\ h$ for m,p-xylene. For benzene and styrene, the Phase II predictions lie entirely within these ranges. For m,p-xylene, the Phase I and Phase II ranges overlap substantially. For o-xylene, the ranges overlap, but only slightly.

In Phase I, to estimate exposure for compounds not measured in the exposures database (CED), we applied an ETS exposure scale factor, Z, that was derived as the average from four measured compounds — benzene, styrene, o-xylene, and m,p-xylene (see Section 4.2.2). Among these four estimates of Z, three agree closely (benzene = 2.51×10^{-3} cig m^{-3}; styrene = 2.45×10^{-3} cig m^{-3}; and m,p-xylene = 3.31×10^{-3} cig m^{-3}), while the result for o-xylene is considerably higher (11.5×10^{-3} cig m^{-3}). If o-xylene were excluded, the average ETS exposure scale factor would decrease by about 43%, from 4.9×10^{-3} cig m^{-3} to 2.8×10^{-3} cig m^{-3}. In turn, this would reduce the estimated ETS-only exposures for Phase I reported in Table 5.9 by 43% for all compounds other than benzene, styrene, o-xylene, and m,p-xylene. With such a reduction, the Phase I estimates would agree well with the Phase II estimates for all compounds other than o-xylene.

5.2.3 Fraction of Nonsmoker Exposure from ETS

Insight into the overall significance of ETS as a source of TACs can be gained by comparing the estimates of exposure caused by ETS to estimates of total exposure from all sources. In Phase I of this project, we synthesized data from the Californian Exposures Database to estimate total

exposure of nonsmoking Californians to four air toxicants: benzene, styrene, o-xylene, and m,p-xylene (Table 4.1). From these data, we computed the arithmetic mean, 24-hour total exposure for these four compounds for the passively exposed population as the average exposure concentration multiplied by 24 hours. For example, for benzene, the mean daily exposure for those exposed to ETS is 12.6 $\mu g\ m^{-3} \times 24\ h = 302\ \mu g\ h\ m^{-3}$. Corresponding values for styrene, o-xylene, and m,p-xylene are 57, 229, and 492 $\mu g\ h\ m^{-3}$, respectively. Assuming these estimates apply for the exposed portion of each population age group, we estimated the fraction of exposure caused by ETS by dividing the appropriate arithmetic mean from Tables 5.3-5.5 by the corresponding total exposure. Table 5.10 presents the intermediate results, showing the total mean inhalation exposure and the estimated mean exposure from ETS for Phase I and Phase II.

Figure 5.7 summarizes the results, showing for those who reported being exposed what fraction of the average total inhalation exposure results from ETS. These results are obtained by dividing the appropriate mean exposure from ETS in Table 5.10 by the mean exposure from all sources. (The values are also reported in parentheses in Table 5.10.) Focusing on Phase II, and including all age groups and scenarios, the fractional contributions of ETS to the total exposure of exposed nonsmokers are in the range 3-10% for benzene, 6-19% for styrene, 0.7-2% for o-xylene, and 1-5% for m,p-xylene. The figure shows that the Phase I predictions lie within the range of Phase II predictions for benzene and styrene, at the top end of the range for m,p-xylene, and above the range for o-xylene.

The fraction of exposure from ETS for *all* nonsmokers (both exposed and nonexposed) are analogously estimated, with the results reported in Table 5.11. The population-weighted average values for the four scenarios in Phase II show that ETS contributes 5-15 $\mu g\text{-}h\ m^{-3}$ of daily exposure to benzene, averaged over all nonsmoking Californians. This corresponds to 2-5% of the total inhalation exposure of nonsmokers. For the other three compounds, the analogous results are as follows: styrene 2-6 $\mu g\text{-}h\ m^{-3}$ (3-10%); o-xylene 0.8-2.5 $\mu g\text{-}h\ m^{-3}$ (0.4-1.1%), and m,p-xylene 4-11 $\mu g\text{-}h\ m^{-3}$ (0.8-2.4%).

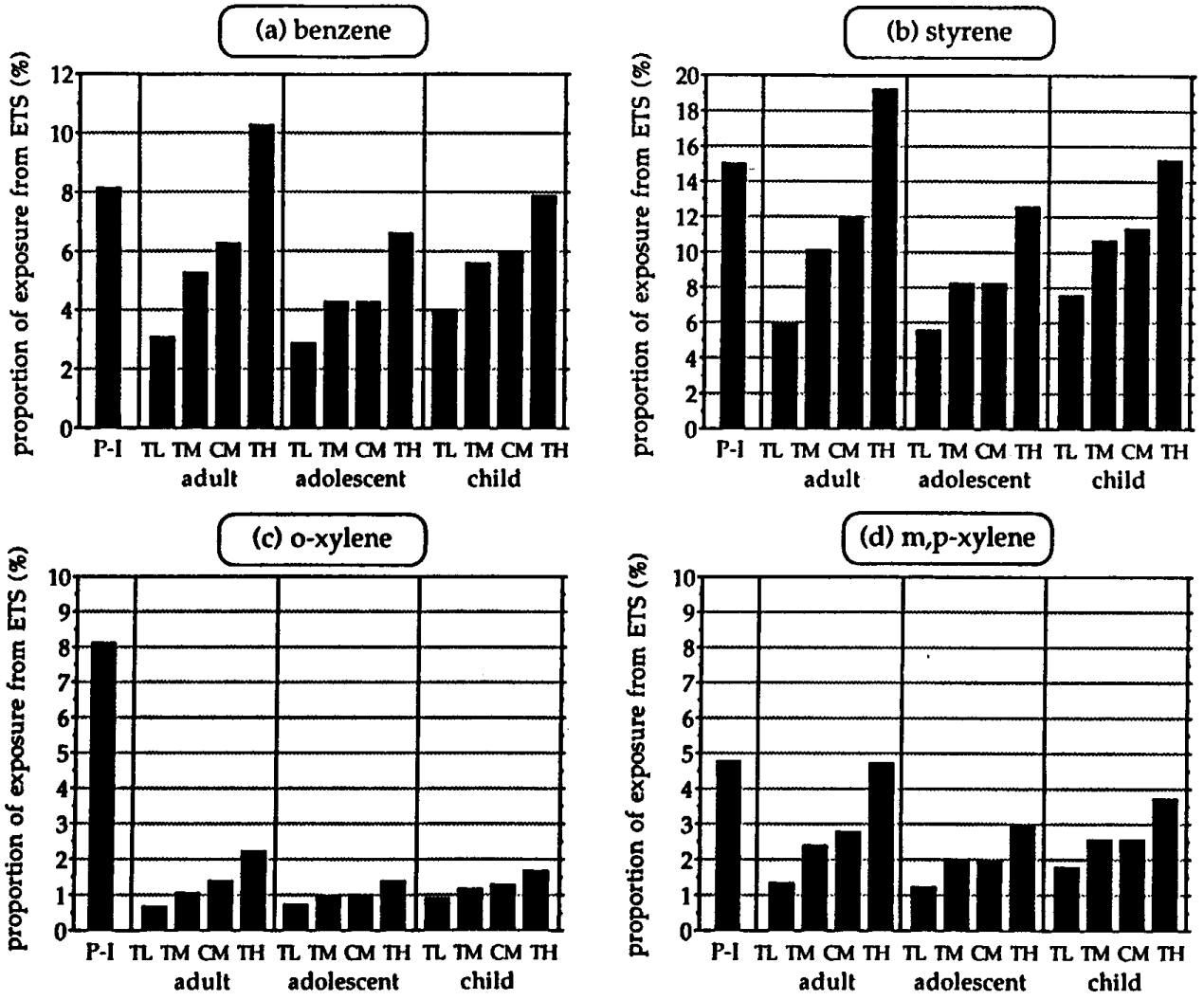

Figure 5.7 Proportion of mean exposure to nonsmoking Californians from environmental tobacco smoke for four air toxicants, late 1980's. Proportions apply to the portion of the population that reports exposure to ETS. (P-I represents Phase I results — see §4; other bars represent Phase II results for different age groups and scenarios: TL - tracer low-range, TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range.)

Table 5.10. Daily mean inhalation exposure (μg h m^{-3}) to selected air toxicants for nonsmoking Californians exposed to environmental tobacco smoke. [1,2]

	species			
	benzene	styrene	o-xylene	m,p-xylene
exposure from all sources				
total exposure [3]	302	57	229	492
exposure from ETS				
Phase I [4]	24.5 (8.1%)	8.6 (15%)	18.5 (8.1%)	24 (4.9%)
Phase II — adults [5]				
scenario TL	9.3 (3.1%)	3.4 (6.0%)	1.5 (0.7%)	6.8 (1.4%)
scenario TM	16 (5.3%)	5.8 (10%)	2.6 (1.1%)	12 (2.4%)
scenario CM	19 (6.3%)	6.9 (12%)	3.1 (1.4%)	14 (2.8%)
scenario TH	31 (10.3%)	11 (19%)	5.1 (2.2%)	23 (4.7%)
Phase II — adolescents [6]				
scenario TL	8.9 (2.9%)	3.2 (5.6%)	1.7 (0.7%)	6.6 (1.3%)
scenario TM	13 (4.3%)	4.7 (8.2%)	2.2 (1.0%)	9.6 (2.0%)
scenario CM	13 (4.3%)	4.7 (8.2%)	2.2 (1.0%)	9.6 (2.0%)
scenario TH	20 (6.6%)	7.2 (13%)	3.3 (1.4%)	15 (3.0%)
Phase II — children [7]				
scenario TL	12 (4.0%)	4.3 (7.5%)	2.0 (0.9%)	8.8 (1.8%)
scenario TM	17 (5.6%)	6.2 (11%)	2.8 (1.2%)	13 (2.6%)
scenario CM	18 (6.0%)	6.5 (11%)	3.0 (1.3%)	13 (2.6%)
scenario TH	24 (7.9%)	8.7 (15%)	4.0 (1.7%)	18 (3.7%)
Phase II — population average [8]				
scenario TL	9.7 (3.2%)	3.5 (6.2%)	1.6 (0.7%)	7.1 (1.4%)
scenario TM	16 (5.2%)	5.7 (10%)	2.6 (1.1%)	12 (2.4%)
scenario CM	18 (6.0%)	6.6 (12%)	3.0 (1.3%)	13 (2.7%)
scenario TH	29 (9.5%)	10.2 (18%)	4.7 (2.1%)	21 (4.3%)

[1] Results in parentheses represent an estimate of the proportion of nonsmoker exposure attributable to ETS, obtained by dividing the exposure from ETS by the exposure from all sources.

[2] Scenarios: TL - tracer low exposure; TM - tracer mid-range exposure; CM - completely mixed room model; TH - tracer high exposure (see Table 5.1).

[3] Obtained from Table 4.1 by multiplying the AM personal exposure concentration for the "passive" population by 24 h per day.

[4] Obtained from Table 4.1 by multiplying the AM personal exposure concentration for "ETS only" by 24 h per day.

[5] Obtained from the AM column of Table 5.3.

[6] Obtained from the AM column of Table 5.4.

[7] Obtained from the AM column of Table 5.5.

[8] Population-weighted average results, derived from entries earlier in the table, with weighting factors of 32.3% for adults (≥ 18 y), 5.6% for adolescents (12-17 y), and 6.6% for children (0-11 y). The weighting factors represent the proportion of the California nonsmoking population in the respective age group that is exposed to ETS on a given day. (For adults: 32.3% = 74% (proportion of total population that is adults) × 78% (proportion of adults that are nonsmokers) × 56% (proportion of nonsmoking adults exposed to ETS); for adolescents, 5.6% = 8.7% × 94% × 68%; and for children 6.6% = 17.3% × 100% × 38%.)

Table 5.11. Daily mean inhalation exposure (μg h m^{-3}) to selected air toxicants for all nonsmoking Californians. [1,2]

	species			
	benzene	styrene	o-xylene	m,p-xylene
exposure from all sources				
total exposure [3]	296	57	222	481
exposure from ETS				
Phase I [4]	13.7 (4.6%)	4.8 (8.4%)	10.3 (4.6%)	13.3 (2.8%)
Phase II — adults [5]				
scenario TL	5.2 (1.8%)	1.9 (3.3%)	0.8 (0.4%)	3.8 (0.8%)
scenario TM	9.0 (3.0%)	3.2 (5.6%)	1.5 (0.7%)	6.7 (1.4%)
scenario CM	10.6 (3.6%)	3.9 (6.8%)	1.7 (0.8%)	7.8 (1.6%)
scenario TH	17 (5.7%)	6.2 (10.9%)	2.9 (1.3%)	13 (2.7%)
Phase II — adolescents [6]				
scenario TL	6.1 (2.1%)	2.2 (3.9%)	1.2 (0.5%)	4.5 (0.9%)
scenario TM	8.8 (3.0%)	3.2 (5.6%)	1.5 (0.7%)	6.5 (1.4%)
scenario CM	8.8 (3.0%)	3.2 (5.6%)	1.5 (0.7%)	6.5 (1.4%)
scenario TH	14 (4.7%)	4.9 (8.6%)	2.2 (1.0%)	10.2 (2.1%)
Phase II — children [7]				
scenario TL	4.6 (1.6%)	1.6 (2.8%)	0.8 (0.4%)	3.3 (0.7%)
scenario TM	6.5 (2.2%)	2.4 (4.2%)	1.1 (0.5%)	4.9 (1.0%)
scenario CM	6.8 (2.3%)	2.5 (4.4%)	1.1 (0.5%)	4.9 (1.0%)
scenario TH	9.1 (3.1%)	3.3 (5.8%)	1.5 (0.7%)	6.8 (1.4%)
Phase II — population average [8]				
scenario TL	5.2 (1.7%)	1.9 (3.3%)	0.8 (0.4%)	3.8 (0.8%)
scenario TM	8.5 (2.9%)	3.0 (5.3%)	1.4 (0.6%)	6.3 (1.3%)
scenario CM	9.6 (3.3%)	3.5 (6.2%)	1.6 (0.7%)	7.1 (1.5%)
scenario TH	15 (5.1%)	5.5 (9.6%)	2.5 (1.1%)	11 (2.4%)

[1] Results in parentheses represent an estimate of the proportion of nonsmoker exposure attributable to ETS, obtained by dividing the exposure from ETS by the exposure from all sources.

[2] Scenarios: TL - tracer low exposure; TM - tracer mid-range exposure; CM - completely mixed room model; TH - tracer high exposure (see Table 5.1).

[3] Derived from Table 4.1 as the weighted average of the AM personal exposure concentration for the "passive" population (56%) and the unexposed population (44%), multiplied by 24 h per day.

[4] Obtained from Table 4.1 by multiplying the AM personal exposure concentration for "ETS only" by 56% exposed and by 24 h per day.

[5] Obtained by multiplying the AM column of Table 5.3 by 56%, the proportion of the nonsmoking adult California population exposed to ETS during a day in the late 1980's.

[6] Obtained by multiplying the AM column of Table 5.4 by 68%, the proportion of the nonsmoking adolescent California population exposed to ETS during a day in the late 1980's.

[7] Obtained by multiplying the AM column of Table 5.5 by 38%, the proportion of children (age 7-11 y, inclusive) in California exposed to ETS during a day in the late 1980's.

[8] Population-weighted average results, derived from entries earlier in the table, with weighting factors of 57.7% for adults (≥ 18 y), 8.2% for adolescents (12-17 y), and 17.3% for children (0-11 y). The weighting factors represent the proportion of the California population that are nonsmokers in the respective age group.

5.3 Discussion

5.3.1 Uncertainty and Variability in ETS Exposure

The uncertainty in our results is indicated by differences in results among scenarios and also by comparison between the Phase I and Phase II results. The true mean exposure to nonsmoking Californians caused by ETS probably lies within the range defined by the TL scenario in Phase II (the lower bound) and the Phase I predictions (upper bound for most species) or the TH scenario in Phase II (the upper bound for adult exposure to benzene and styrene). That the results from Phase I are comparable to the Phase II results, even though generated by an almost entirely independent method, adds confidence to the findings. Also, the fact that the CM scenario, based largely on predicting ETS concentrations, agrees well with the T- scenarios, based largely on measured ETS concentrations, further substantiates the findings. However, the input data for this analysis are not of sufficient quality to definitively conclude that the true mean lies within these ranges. Information for estimating microenvironmental concentrations in retail/other and transportation microenvironments is particularly weak.

The variability in exposure among the population is reflected in parameters such as the GSD. The analysis consistently reveals that the variability is much larger than the uncertainty. We can be confident that some individuals experience ETS exposures that are much larger than the mean. For example, for adults in the TM scenario, the population mean exposure to benzene from ETS is estimated to be 16 $\mu g\ h\ m^{-3}$ while the 95th percentile among those exposed to ETS is 60 $\mu g\ h\ m^{-3}$ (see Figure 5.2a). It might be important in developing public policies for ETS exposure control to identify and target interventions at the high end of the distribution.

For methodological reasons, the variability information generated in this phase of the project is superior in quality to that indicated in Phase I. A key challenge encountered in Phase I was to discern a fairly small fractional contribution of ETS to exposure in the presence of large contributions from other sources. This weakness is not present in the approach applied in this second phase. Here, whether using nicotine as a tracer or predicting ETS concentrations using a material balance model, the presence of other sources of air toxics is not a factor. Even when particulate matter is used as a tracer, tobacco smoke is such a strong source that the effect of background on the results is small.

As with uncertainty, the variability revealed by this research should be considered as indicative, rather than definitive. Not every factor that contributes to variability was captured in the analysis. For example, to ensure stability in the results, exposures were predicted 40 times for each of the study subjects. Each time exposure was computed, the same activity pattern was applied. Consider the case of adults, for example, where the number of subjects is 579 in our

model simulation. In reality, if 23,000 (40 × 579) fully independent assessments were made of daily exposure, they should reveal somewhat larger variability because the daily activity patterns would not tend to be as narrowly constrained as in the study group simulated here.

Another important point concerns the difference between daily exposure and long-term average exposure. Health effects from TACs may result from either cumulative exposure over a long period or acute episodic exposure. The long-term average rate of exposure accumulation (i.e., the average exposure concentration) for a population is expected to be the same as for a short-term period. For example, the mean ETS-caused benzene exposure for the California nonsmoking population is estimated to be in the range 4-16 $\mu g\ h\ m^{-3}$ per day (Table 5.9), corresponding to an average exposure concentration of 0.2-0.7 $\mu g\ m^{-3}$. This average exposure concentration should also apply on a long-term basis.

On the other hand, the variability in exposure is a function of the time period of integration. In general, as the period increases, the variability decreases. The magnitude of this effect cannot be easily predicted. The fundamental reason why variability tends to narrow with increasing exposure period is this: the further short-term exposure departs from the mean, the greater the probability that at least part of the departure is caused by a temporal fluctuation. In other words, an exposure that is very high relative to the population mean for one day is more likely to be above rather than below the subject's long-term average exposure. The smaller the day-to-day fluctuations, the less variability will change with increasing exposure period.

5.3.2 Assessment Limitations

This section presents a discussion of the main factors that affect the accuracy and precision of our predictions of TAC exposure caused by ETS in Phase II. It is not possible, in most cases, to quantify the effects of these factors. However, largely on the basis of the judgment and experience of the investigators, some qualitative comments can be made about their significance.

5.3.2.1 Quality of Input Data

The data used in this assessment can be grouped into four broad categories:

(1) activity pattern surveys (Jenkins et al., 1992; Wiley et al., 1991a and 1991b) provided information on who, where, and for what duration individuals were exposed to environmental tobacco smoke; this survey also provided information on the prevalence of smoking and the rates of cigarette consumption by smokers;

(2) measurements of ETS tracers (nicotine and particulate matter) from many sources provided information on the concentrations of environmental tobacco smoke in different microenvironments;

(3) surveys and studies conducted principally for energy conservation and general indoor air quality use provided information on building factors such as ventilation rate and building volume that, in turn, were used in CMR models to predict microenvironmental concentrations of ETS; and

(4) emission factors for toxic air contaminants in environmental tobacco smoke (Daisey et al., 1994 and 1998; Martin et al., 1997) were used to predict microenvironmental concentrations of ETS-caused TACs using both the tracer and CMR model approach.

For five of the microenvironments, the quantity of available data is generally good. For the "retail/other," "transportation," and "school" microenvironments, relevant data are sparse and so one cannot place much confidence in the exposure predictions for these settings.

The activity pattern data have several important strengths. The study population is a statistically representative sample of Californians. For adults and children, the size of the study populations we used—the 579 nonsmoking adults and 413 children who reported some exposure to ETS during the day in the simulated microenvironments—are adequately large to provide good information not only on the central tendencies but also on the variability within the distribution (that is, the 90th percentile exposures). (On the other hand, the sample size of 86 for nonsmoking adolescents is small enough to limit the robustness of our exposure estimates.) The level of detail contained in the survey regarding activities is excellent.

The most important weakness in the APCR and CAP studies for the present purposes arises in discerning precisely which of the study subjects were exposed to environmental tobacco smoke and especially for what duration. The self-reported proximity (SRP) is an important, but ambiguous indicator. It seems likely that complete reliance on SRP as a measure of exposure duration would tend to bias the results towards underpredicting exposure. Especially in indoor settings, ETS exposure is certainly indicated by an answer "yes" to the proximity question: "were you around anyone (else) who was smoking...while...?" However, ETS exposure might also have occurred even if the answer to this question is "no." Cigarette smoke can be transported from one part of a building to another. Also, exposure could occur from smoking that had taken place, but ended, before the activity began in the given location. On the other hand, the answer "yes" to the proximity question does not demonstrate that ETS exposure occurred throughout that activity. And, of course, in addition to the duration of exposure, the intensity of exposure must be known and the SRP response provides no clue about ETS concentrations. Overall, information on duration of exposure may limit the accuracy and precision of our estimates to about the same degree as the information on microenvironmental concentrations.

Several good studies have been published on ETS tracer concentrations in different microenvironments. The best information is available for residences and offices; data quantity and quality are also good for restaurants and bars/nightclubs. Three important weaknesses are

apparent: (1) most studies did not use statistically representative samples of the full set of the particular microenvironment studied; (2) very few of the measurements were made in California; and (3) both nicotine and particulate matter suffer problems as tracers of TACs in ETS. Our approach of including low- and high-range exposure scenarios was designed partly to compensate for the first two weaknesses. For predicting the arithmetic mean, the TL and TH scenario results differ by factors of approximately 2-3 overall. It seems unlikely that the differences in conditions between California and the sites where measurements were made are as large as this. The weaknesses associated with ETS tracers are also diminished by incorporating some studies that measured nicotine and others that measured particulate matter (PM). The primary problem with nicotine—interactions with surfaces—are distinct from the primary problem with PM—the presence of significant nonETS sources. The use of both the CMR model approach and the tracer method for predicting microenvironmental concentrations also reduces the significance of nicotine and PM being imperfect tracers.

The data used in the CMR model for residences and offices are generally very good in quality and quantity. Residential air-exchange rate measurements are approximately specific to California, but do not constitute a representative sample. Residential volume measurements are statistically representative, but for the US as a whole, rather than restricted to California. The ventilation rate information for offices includes a study from California, but better quality data are from a nationwide study; neither study measured a representative sample of buildings.

The ETS emissions factors are based on careful measurements in a room-sized environmental chamber using the cigarettes most commonly smoked in California (Daisey et al., 1994 and 1998). One potentially important limitation applies to these results. The cigarettes were machine smoked and only the sidestream smoke was emitted into the chamber. However, an independent study conducted using human smokers yields largely consistent results (Martin et al., 1997), thereby adding confidence to the use of relative emission factors for estimating exposure to suite of toxic air contaminants.

5.3.2.2 Quality of Model

In the broadest sense, the model consists of five components taken in combination:

(1) the method for predicting the contribution of ETS to benzene concentrations in microenvironments based on tracer measurements;

(2) the method for predicting the contribution of ETS to benzene concentrations in microenvironments based on the principle of material balance (CMR model);

(3) the method for constructing ETS-caused benzene exposure for an individual by combining data on activity patterns and microenvironmental concentrations;

(4) the method for constructing probability distribution functions of ETS-caused benzene exposure as a composite of the exposures of individuals; and

(5) the method of predicting exposure to different TACs by scaling according to the relative emission factors.

The strengths and weaknesses of each of these elements is discussed briefly here, except (4) which, being thoroughly sound, requires no further discussion.

Aspects (1) and (5) depend on the same fundamental premise: that the time-averaged concentrations of TAC and tracer species produced in environmental tobacco smoke are present in consistent proportion from one microenvironment to another. Probably, two conditions must be met to satisfy this premise. First, the relative amounts emitted of each species should be consistent among cigarettes and no more than weakly dependent on how cigarettes are smoked. Second, the indoor dynamic behavior, especially with respect to interactions of pollutants with surfaces, should not significantly alter the ETS mix, at least on a time-averaged basis. Available information does not permit firm conclusions about whether these conditions are met. Nevertheless, the data on emissions factors from Daisey et al. (1994 and 1998) and Martin et al. (1997) indicates that ETS emissions are only weakly dependent on cigarette brand and on whether cigarettes are smoked by machine or by humans. A large portion of ETS is sidestream smoke emitted while the cigarette is idling between puffs, and one would expect the combustion conditions to be relatively constant for idling conditions. The evidence on dynamic behavior is less compelling. Laboratory studies show, for example, that following combustion of a single cigarette, the decay of nicotine concentration follows a different pattern than that of other species, including ideal tracers (Nelson et al., 1992). On the other hand, as noted earlier, in environments where smoking is habitual, nicotine may attain a dynamic balance such that the average airborne concentration is an accurate indicator of the average ETS level (Van Loy et al., 1997a, 1998). The relative consistency of toxic air contaminant concentrations caused by tobacco smoking has not been ascertained in any field studies.

Regarding aspect (2)—as applied, the CMR model raises several potential concerns. First, implicit in the derivation of equations (5.2)–(5.4) and the application to predicting exposure is the well-mixed hypothesis. Strictly, we assume that the average ETS concentration encountered by a nonsmoker in an indoor environment is the same as the average concentration leaving the building via ventilation. This strict condition can be met if the species concentrations are uniform everywhere throughout the space.

Location of the smoker in the indoor environment is another important consideration. Since cigarette smoke is effectively emitted from a point, proximity of a nonsmoker to a smoldering cigarette will influence exposure, and this can work in both directions. Social interactions with the smoker will tend to place the nonsmoker closer to the emissions and increase

exposure. Efforts of nonsmokers to avoid cigarette smoke will tend to reduce exposure, relative to the model predictions.

Other behavioral factors may also significantly affect exposure, but are not captured in the model. For example, actions such as deliberately increasing building ventilation during smoking, or having the smoker step outside to smoke, will decrease exposures relative to predictions.

As was discussed in an earlier paragraph for the tracer method, the CMR model, as formulated here, also depends on the assumption that pollutant interactions with surfaces are not important modifiers of exposure. This assumption is expected to be better for compounds with high volatility than for those with lower volatility.

Given these concerns, it is reassuring that both the residential and occupational microenvironments exhibit results that are largely consistent between the CM and the T- scenarios, despite the very large differences in computational approach. Tables 5.6-5.8 show, for example, that the AM exposure in residences for scenario CM agrees well (within about 20%) of the AM for scenario TM. In the occupational microenvironment, the AM values for exposure vary markedly among the T- scenarios, with the value for TH approximately 7 × that for TL. Again, though, the mean for CM agrees well with the mid-range tracer (TM) estimate.

In concept, aspect (3) is fundamentally sound. However, one may be concerned by the aggregation of distinct microenvironments into groups, as well as by the representation of a broad group of microenvironments by a smaller class. For example, the retail/other category is highly diverse and may not be represented well by a single probability distribution function. Also, we have implicitly represented all workplace environments by offices except in TH, where blue-collar worksites were separately considered. Information needed to more completely relax these approximations is lacking.

5.3.2.3 Biases

An important goal in this assessment was to avoid bias in the results. That is, we have sought to minimize the extent to which our assessment predicts exposures that are systematically greater or less than expected. We only know of one factor that contribute to bias in our results. The effect contributes perhaps a 20% error to the predictions, small compared with the uncertainty range of a factor of 2–3 associated with the different scenarios.

We have excluded from the assessment several microenvironments in which some exposure to tobacco smoke is reported (see Tables 3.2-3.4). According to our assessment of the APCR data 10% of minutes of self-reported proximity of adults to ETS occur in these settings (Figure 3.2). For adolescents and children, the proportion is comparable, 8% and 15%, respectively. If the true exposure duration were the same as indicated by SRP and if the average exposure concentration in the excluded environments matched the average for those included, our

predictions of exposure would be biased by corresponding percentages below the true value because of this factor. However, the true bias is undoubtedly smaller than indicated by these percentages because the excluded microenvironments are almost entirely outdoors where ETS concentrations are expected to be much less than for an average indoor site where smoking occurs.

5.3.3 Comparison with Prior Studies

Benzene exposures have been estimated by MacIntosh et al. (1995) using a population-based exposure model. The methodology of the MacIntosh et al. study is similar to ours in that they conducted a probabilistic simulation of time-activity patterns combined with microenvironmental concentrations. Personal air exposures were estimated for the US Environmental Protection Agency's Region 5 (Illinois, Indiana, Michigan, Minnesota, Ohio, and Wisconsin) and Arizona. Cigarette smoking was included as an indoor source of benzene. Indoor air concentrations resulting from smoking were modeled as lognormal with GM = 1.0 $\mu g\ m^{-3}$ and GSD = 3.3. The 24-hour average benzene exposure concentration distribution due to ETS from MacIntosh et al. has an arithmetic mean of 0.8 $\mu g\ m^{-3}$ and a 90th percentile value of 2.5 $\mu g\ m^{-3}$ (Figure 3 in MacIntosh et al.). These concentrations can be converted to exposures by multiplying by 24 hours, yielding an AM of 19 $\mu g\ h\ m^{-3}$ and a 90th percentile value of 60 $\mu g\ h\ m^{-3}$. These estimates from MacIntosh et al. agree well with our predictions for adults (compare Figure 5.1a).