Occupational Exposure to Environmental Tobacco Smoke and Health Risk Assessment

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This article addresses concepts of environmental tobacco smoke (ETS) exposure assessment relevant for health risk assessment based on human studies. We present issues that should be considered when selecting a method for ETS exposure assessment for the purposes of health risk assessment and review data on ETS exposure levels in the workplace and in home environments. Two types of estimates are needed for a quantitative risk assessment of the health effects resulting from occupational ETS exposure: *a*) an unbiased estimate of the exposure–effect (or dose–response) relation between ETS and the health effect of interest, and *b*) estimates of the distribution of ETS exposure in different workplaces. By combining the estimated exposure–effect relation with information on exposure distribution for a population of interest, we can calculate the proportions of disease cases attributable to occupational ETS exposure as well as the excess number of cases due to specified exposure conditions. Several dimensions of the exposure profile should be considered when assessing ETS exposure for estimating the exposure–effect relation, including the magnitude of exposure and the biologically relevant time specificity of exposure. The magnitude of exposure is determined by the ETS source strength, environmental factors modifying concentrations, and duration of exposure. Time specificity considerations include the latency period for each health outcome of interest, the time–exposure profile relevant for different disease mechanisms, and the sensitive age period with regard to health effects. The most appropriate indicator of ETS exposure depends on these factors and on the time period that can be assessed with different methods. *Key words*: exposure assessment, risk assessment, tobacco smoke pollution, workplace exposure. — *Environ Health Perspect* 107(suppl 6):829–835 (1999).
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Exposure assessment is an essential element of the evaluation of health risks of environmental exposures. In this article, we consider exposure assessment for environmental tobacco smoke (ETS), with emphasis on occupational settings. Substantial research on the adverse health effects of ETS has already accumulated (*1–4*). Many of the studies have been directed at exposures in residences and involve the effects of smoking in the household on nonsmoking spouses and children, whereas much less data are available on the effects of occupational ETS exposure. This workshop evaluated the evidence on potential health effects resulting from ETS exposure in the workplace.

In this article, we consider the exposure information needed to conduct an informative risk assessment of the health effects of ETS based on human studies, whether epidemiologic or clinical. We present concepts of ETS exposure assessment relevant for health risk assessment based on human studies. We introduce the most commonly used ETS exposure assessment methods and present biologically driven approaches to guide selection of the most appropriate ETS exposure assessment method for assessing health risk. We also review data on ETS exposure levels in the workplace and in the residence and data on their relations with reported smoking by others, with the goal of evaluating their usefulness for assessing the risk of ETS exposure in the workplace.

Fundamental Concepts in ETS Exposure Assessment

Definitions Related to ETS Exposure

ETS exposure of nonsmokers is often referred to as passive smoking or involuntary smoking. It constitutes the exposure of a nonsmoking person to tobacco combustion products from the smoking of others (*5*). First, we consider fundamental concepts related to exposure. Concentration refers to the amount of a contaminant at a particular location in a particular medium, e.g., in a specified volume of air (*6*). ETS is a mixture of gaseous compounds and particles composed of concentrations of several individual constituents. Air pollutant concentrations are usually expressed as mass per unit volume, e.g., micrograms per cubic meter. Exposure is defined as the contact of pollutant with a susceptible surface of the human body (*6–8*). For ETS this definition implies contact with the eyes, the epithelium of the nose, mouth, and throat, and the lining of the airways and alveoli. With respect to time, exposure can be expressed on several time scales, including instantaneous exposure, average exposure over a specified time period, and cumulative exposure (*8,9*). Dose is defined as the amount of contaminant that crosses a boundary of the body (*6,7,10*). Dose depends on the concentration at which exposure is received, the time course of exposure, and the physiologic state of the individual. The amount of the pollutant absorbed constitutes the dose to the body, and the amount that reaches the target organ of the adverse effect is the biologically effective dose.

The Chain Linking ETS Sources to Health Effects

Figure 1 [from Jaakkola and Jaakkola (*9*)] presents the chain that links the sources of ETS to the exposure of an individual and finally to the biologically effective dose. The number of smokers and their smoking patterns in a given space determine the source strength for ETS. The source strength is a major determinant of the concentration of ETS in a given space, but the concentration is also determined by characteristics of the space, including the volume of the space, the ventilation rate, and other factors affecting removal of ETS such as air cleaning. The contact of the pollutant with the relevant surfaces of an individual results in exposure. Dose depends on the concentration of ETS and the physiologic factors that modify the uptake of ETS, including lung morphometry and activity level of the individual. Activity level determines the breathing pattern and thus the ventilation of the lungs and the sites of deposition of ETS components. The biologically effective dose further depends on processes occurring in the body after uptake, including metabolism and elimination of the compounds. The difference between the dose and the biologically effective dose varies according to the health outcome: for asthma the dose received in the lung is probably equivalent to the biologically effective dose, whereas for lung cancer patterns of carcinogen metabolism and elimination affect the biologically effective doses of particular carcinogens.

Figure 2 presents the sequence from dose to health effects. For any given biologically effective dose, the potential health effects may be modified by individual characteristics that determine susceptibility to the injury-causing

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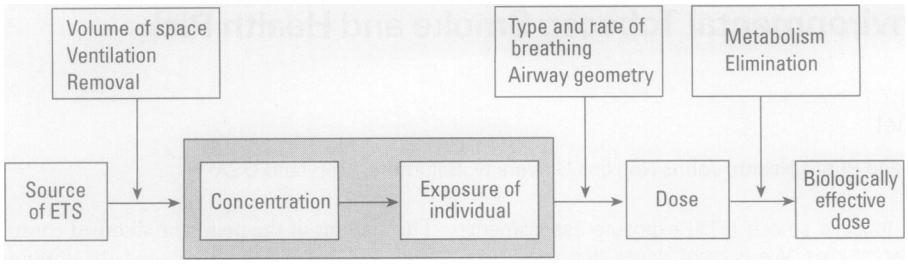

Figure 1. The chain linking ETS sources to the exposure of an individual, and finally to the biologically effective dose. Different environmental and intrinsic factors that influence or modify exposure and dose are illustrated. Reprinted from Jaakkola and Jaakkola (*9*) with permission of the *European Respiratory Journal*.

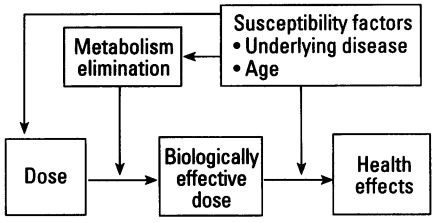

Figure 2. The sequence from dose to health effects. The intrinsic and physiologic factors that determine susceptibility to detrimental effects of ETS are illustrated.

agents in ETS. These factors have not been studied extensively, but at least underlying respiratory or cardiovascular disease and age appear to be important determinants of susceptibility. Genetic factors may also modify responses to tobacco smoke. In addition to modifying the biologic responses of the body to the biologically effective dose, factors determining susceptibility may also modify the dose itself and the biologically effective dose, e.g., by affecting the breathing pattern and thus the uptake of ETS or by influencing the metabolism of absorbed compounds.

Although concentrations of ETS may differ in homes and workplaces, the exposures are expected to be qualitatively similar in chemical composition. Factors that may lead to different concentrations in the workplace compared to the residential environment include differences in source strength resulting from different densities of smokers and different smoking patterns in the two types of environments, and differences in environmental factors that modify concentrations. The most significant of these environmental characteristics are the volume of the space and the air change rate for uncontaminated air. At any given exposure, doses may differ in the home and workplace settings. Factors that may lead to different doses in occupational and residential settings include those affecting uptake, particularly differences in activity levels that affect breathing rate and pattern (mouth vs nose) and thus the ventilation rate of the lungs. Physical activity increases rate of breathing and lung ventilation and hence the delivered dose of ETS.

ETS Exposure Assessment for the Purposes of Health Risk Assessment

The basic aim in assessing personal ETS exposure is to measure the concentrations of ETS that an individual encounters at different times as he/she moves through various microenvironments such as home, workplace, and public places (*9*). Data on the time spent in these microenvironments are also needed to calculate total personal exposure. To conduct a quantitative risk assessment of the health effects resulting from occupational ETS exposure, we need two types of estimates:

- An unbiased estimate of the exposure–effect (or dose–response) relation between ETS and the health effect of interest. Such an estimate can be derived either from individual studies or from a meta-analysis or pooled analysis of individual data. The exposure or dose data should reflect total personal exposure or exposure from one source of interest if we can control for the other potential sources. Control for other potential sources can be accomplished as part of the study design, e.g., by studying working persons married to nonsmokers or housewives with no workplace exposure, or it can be accomplished by adjusting in the data analysis. Home and work environments are the major sources of potential ETS exposure, since most time is spent in these two environments.
- Estimates of the distribution of ETS levels in different workplaces. For risk assessment we need the full characterization of workplace exposure distribution. We need an estimate of the proportion of the work force of interest that is exposed to ETS in the workplace and information on the range of occupational ETS exposures. Ideally, we prefer to derive these estimates from random samples of the work forces providing representative frequency distributions of ETS exposure levels. To extend risk estimates from the home environment to the workplace, we need information on the levels of occupational ETS exposure compared to residential exposure levels. To use risk estimates based on total personal exposure, we need information on the proportion of total exposure contributed by occupational exposure.

By combining the estimated exposure–effect relation with information on exposure distribution for a population of interest, we can calculate the proportions of the exposed disease cases (attributable proportion or etiologic fraction) and of all disease cases in this population (population-attributable proportion) attributable to occupational ETS exposure. We can also estimate the excess number of cases due to specified exposure conditions.

Next we discuss important issues that should be considered when selecting an ETS exposure assessment method for estimating the exposure–effect relation and review currently available data on workplace and residential ETS exposures.

Selection of ETS Exposure Assessment Method for Estimating Health Effects

When estimating health effects of ETS exposure, two dimensions in the ETS exposure profile should be considered (*9*): quantitative assessment of exposure and time specificity of exposure in relation to outcome. Quantitative ETS exposure assessment includes the magnitude of ETS concentration, the duration of exposure, and the time pattern of exposure (changes in the exposure level over time). The time period of interest depends on the health outcome.

A key issue concerning the time specificity of exposure is the biologically relevant exposure for each health outcome of interest. Three aspects of the time specificity of exposure should be considered. First, the latency period, or the time period from start of exposure to manifestation of the health outcome, may vary from a few hours for exacerbation of asthma to 20 years or more for lung cancer. Second, the relevant exposure–time profile may vary considerably from one disease to another. For example, high but brief peak levels may be relevant for exacerbations of asthma, whereas development of lung cancer likely reflects cumulative exposure over long time periods. Third, for some health outcomes there may be susceptible age or maturation windows during which exposure may cause disease, whereas similar exposure during another period may have less risk or possibly no effect. For example, the sensitive period may be relevant when assessing the adverse effects of a mother's occupational exposure on the fetus or of household smoking on lung development of infants.

Assessment Methods for ETS Exposure

ETS is a complex mixture of gases and particles. The epidemiologic evidence links the mixture rather than specific components to health effects. Little is known about the role of individual components in causing specific adverse health outcomes. In addition, synergistic effects (interactions) among different compounds are likely to be important in determining health effects. Therefore it is necessary to choose an indicator or a marker for the entire ETS mixture when assessing ETS exposure. In developing an exposure assessment strategy, the most appropriate indicator should be used, considering features of the pathogenesis of the health outcome of interest. The following indicators of ETS have been widely used in previous studies: individual chemical compounds measured in the air, indices derived from questionnaires, and metabolites of ETS components measured in biologic specimens (*1,9,11*). Methods for ETS exposure assessment are divided into direct and indirect methods. These methods are described in more detail in a recent article by Jaakkola and Jaakkola (*9*) that also discusses their strengths and limitations. Personal monitoring of relevant tobacco smoke constituents is considered to be the most direct ETS exposure assessment method. Indirect assessment methods measure concentrations of indoor air tobacco smoke compounds with stationary monitors or collect information on ETS sources in different microenvironments and combine these data with information on time periods spent in each microenvironment, using mathematical formulas or modeling approaches. Biomarkers are usually measures of dose rather than direct or even indirect measures of exposure.

As mentioned, chemical compounds in the air can be measured either with stationary monitors or with personal monitoring using samplers worn for several hours to several days (*9*). Vapor-phase nicotine is the most commonly used marker for the gas-phase constituents of ETS. Other markers of the gas phase include carbon monoxide (CO), nitrogen oxides, formaldehyde, and volatile organic compounds (*1,12*). Respirable suspended particulates (RSPs) have been measured most often as a marker of particle phase constituents of ETS. These are particles less than 2.5 µm in aerodynamic diameter and thus small enough to enter the peripheral airways and alveoli. Other potential markers of the particle phase include tobacco-specific nitrosamines, benzo[*a*]pyrene, and polycyclic aromatic hydrocarbons (PAHs) (*4,12*). The advantages related to using nicotine and RSPs as markers have been availability of validated and easy measurement methods, knowledge of their emission rates from tobacco combustion, and knowledge of their relations to other ETS components (*1,9*). In addition, the presence of nicotine in air is specific to tobacco combustion.

Questionnaire-based assessment is the most widely used exposure assessment method in epidemiologic studies of the health effects of ETS. Questionnaires are relatively inexpensive and can be used to assess long-term ETS exposure, even focusing on past exposures (*9*). Questionnaires have been developed to improve exposure assessment by considering time specificity of exposure as well as quantification of exposure (*13–15*).

Biomarkers can be considered surrogate measures of dose. However, the relation between exposure and the level of a biomarker is often complex, as it is modified by the various factors presented in Figure 1. Cotinine, one of the major metabolites of nicotine, has been used commonly as a biomarker for both active smoking and ETS exposure. In adult nonsmokers, its half-life is from 7 to 40 hr (*1,16*), and it can be measured in plasma, urine, and saliva. Hair nicotine content is a relatively new biomarker of ETS that has the advantage of representing tobacco smoke exposure during the previous 1 to 2 months (*17*). Adsorption of nicotine from the surrounding air onto hair appears to be the primary contributor to the overall nicotine content in the hair, so hair nicotine represents exposure rather than dose (*18*).

Recent reports have assessed protein and DNA adducts as markers of ETS exposure (*4*). The compound 4-aminobiphenyl (4-ABP) is a known human carcinogen, and its adduct of hemoglobin has a half-life of approximately 4 months (*1*). Elevated levels of 4-ABP adduct have been observed in association with ETS exposure (*19,20*). Albumin adducts of PAHs is another group of protein adducts measured as markers of ETS (*21*). Reports of other lung carcinogens as potential biomarkers of ETS, such as 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol and its glucuronide, have also been published (*4,22*).

Selection of the Most Appropriate Method for ETS Exposure Assessment

Table 1 presents an overview of the time frames that can be assessed with different indicators of ETS exposure. The time period that can be assessed with a single measurement and with repeated measurements is indicated for each marker. For air concentration measurements, continuous monitoring is also considered. The last column indicates whether a given method can be applied for assessment of current or past exposure. Air concentration measurements of ETS markers provide assessment of current exposure on a time frame of hours to months, depending on whether the measurements are taken once, repeatedly, or by continuous monitoring. Measurements of biomarkers provide assessment of past exposure. The exposure window spans hours with cotinine measurements and months with hair nicotine and 4-ABP adduct. With repeated measurements of hair nicotine or 4-ABP adduct, exposures over years can be assessed. Questionnaires can be used to assess both current and past exposures and to provide assessment for periods ranging from hours to years.

Table 2 presents, according to our judgment, the most likely latency periods for effects of ETS exposure on four major diseases of interest. Development of new disease and exacerbation of established disease are considered separately for asthma and coronary heart disease (CHD). The latency periods cover a full spectrum—from hours for exacerbation of asthma and angina, to days for exacerbation of asthma, to months for induction of asthma and low birth weight, and to years for induction of asthma, CHD, and lung cancer.

Table 1. Exposure period that can be assessed with different indicators of ETS.

Indicator of ETS	Frequency of measurements	Hours	Days	Months	Years	Current or past exposure
Air concentrations of nicotine or RSPs	Once	X				C
	Repeated		X	X		C
	Continuous monitoring	X	X			C
Urine, salivary plasma cotinine	Once	X				P
	Repeated		X	X		P
Hair nicotine	Once			X		P
	Repeated			X	X	P
4-ABP-Hb adduct	Once			X		P
	Repeated			X	X	P
Questionnaire	Once	X	X	X	X	C, P
	Repeated		X	X	X	C, P

Abbreviations: C, current; P, past; 4-ABP-Hb adduct, hemoglobin adduct of 4-aminobiphenyl.

Table 2. Latency period for different health outcomes.

Health outcome	Hours	Days	Months	Years
Asthma				
Exacerbation	X	X		
Induction			X	X
Low birth weight			X	
Coronary heart disease				
Angina	X			
Induction				X
Lung cancer				X

Table 3. Appropriate exposure assessment methods for different health outcomes.

Health outcome	Air conc. (nicotine or RSP)	Urine, salivary plasma cotinine	Hair nicotine	4-ABP-Hb adduct	Questionnaire
Asthma					
Exacerbation	O,C	O			O
Induction	R	R	O,R	O,R	O,R
Low birth weight	R	R	O, R	O, R	O, R
Coronary heart disease					
Angina	O,C	O			O
Induction			R	R	O,R
Lung cancer			R	R	O, R

Abbreviations: C, continuous monitoring, conc., concentrations, O, one measurement; R, repeated measurements.

Table 3, which combines information from Tables 1 and 2, synthesizes the exposure assessment methods suitable for different health outcomes. Single and continuous measurements of air concentrations of ETS markers are appropriate for conditions with a short latency period such as exacerbation of asthma and angina. Repeated measurements can be used to assess exposure related to induction of asthma and low birth weight. Single measurements of cotinine in body fluids can be used in estimating risk for exacerbation of asthma and angina, whereas repeated measurements are suitable for induction of asthma and low birth weight. Hair nicotine content and the hemoglobin adduct of 4-ABP provide an assessment of exposure during months and are suitable for induction of asthma and low birth weight. Repeated measurements of these biomarkers can provide an assessment of exposure for years and can thus be used for induction of asthma, low birth weight, CHD, and lung cancer. Finally, since questionnaires can be focused to provide exposure assessment for any time period, they are useful for all the health outcomes. They are particularly suited to studying diseases with very long latency periods such as CHD and lung cancer. In addition to consideration of the latency period and the need for assessment of current or past exposure, the relevant time–exposure profile for the health outcome of interest also should be considered when choosing the appropriate exposure assessment method. For example, high peak values could be registered by continuous monitoring of marker concentrations in the air, whereas average levels over longer time periods could be measured with repeated air concentration determinations combined with questionnaire-derived time–activity data. Cumulative exposure over long time periods could be assessed with hair nicotine content, 4-ABP hemoglobin adduct levels, or indices based on questionnaire information.

Some markers might be particularly relevant for specific health effects depending on the phase of ETS represented by the marker, the site of deposition, and the target tissue ultimately reached by the marker. Information on specific compounds biologically relevant for different health outcomes is sparse, with the exception of carcinogenic effects (*1,4,23*). Hypotheses can be formed; but in the case of ETS, interactions among different compounds may be essential for the health effects. Carcinogen adducts, even if measured in blood, may be the most appropriate indicator for cancer risk. For asthma, both gas-phase and particulate-phase markers may be relevant; exacerbations might be triggered by deposition of particles and gaseous compounds in the upper airway and bronchi. In addition to other mechanisms, carbon monoxide has been suggested as a factor in exacerbation of angina (*24*). Further research on the pathogenesis of ETS-related diseases is needed before recommendations can be made on the basis of biologic relevance of any individual compound.

Table 4. Summary of indoor air nicotine and RSP concentrations associated with smoking occupancy in residential and work environments in field studies with at least 15 observations.[a]

Microenvironment	Range in mean air nicotine conc. (μg/m^3)	Minimum–maximum of air nicotine conc. (μg/m^3)	Range in mean air RSP[b] conc. (μg/m^3)	Minimum–maximum of air RSP[b] conc. (μg/m^3)
Residences	2.2–19	0.1–292	12–430	0.7–1100
Offices	1.1–10.0	0–71.5	28–133	0–1088
Other workplaces	4.3–14	< 1.6–167	36–300	6–3580
Restaurants and/or bars	4.3–22	0–450	53–126	0–685

[a]Based on review by Guerin et al. (*12*). [b]RSP levels related to smoking occupancy were calculated by subtracting the average level in nonsmoking environments (i.e., the background level) from that observed in the smoking environments for each study (when applicable) before taking it into account in this summary table.

ETS Levels in the Work and Residential Environments

Comparison of the Indoor Air Concentrations of ETS Markers

Since most epidemiologic data on health effects of ETS come from studies assessing ETS exposure at home, we review and compare data available on ETS levels in both work and residential environments. Two reviews on the topic were published in 1992 (*1,12*). In addition, U.S. studies on ETS exposure in the workplace and home were summarized recently by Hammond (*25*).

Guerin and co-workers (*12*) reviewed major studies, defined as those with at least 15 observations, that had measured air nicotine and/or RSP concentrations in different environments. A summary of these findings is presented in Table 4. The average air nicotine levels were comparable between residential environments and work situations other than office work but slightly lower in office environments. The highest nicotine levels have been observed in bars and restaurants or in small enclosed spaces with minimal ventilation, e.g., small offices, cars, and aircraft. Indoor RSP concentrations associated with smoking occupancy were also comparable between residential environments and work environments other than offices, but the upper range of mean concentrations was slightly lower in offices. The RSP values in Table 4 were achieved by subtracting the average level in nonsmoking environments from that observed in the smoking environments for each study. RSP levels in nonsmoking households were about half or less those related to smoking occupancy. Figures 3 and 4 show the range of average nicotine and RSP concentrations as well as the range of maximum and minimum values from smoking occupancy by different indoor environments, according to the review by U.S. Environmental Protection Agency (*1*). Only studies with sampling times of 4 hr or greater were included in the residential and office environments. The mean nicotine concentrations were comparable between home and office environments, whereas the maximum concentrations were higher in office environments. The mean and maximum RSP concentrations were somewhat higher in residential than in office environments.

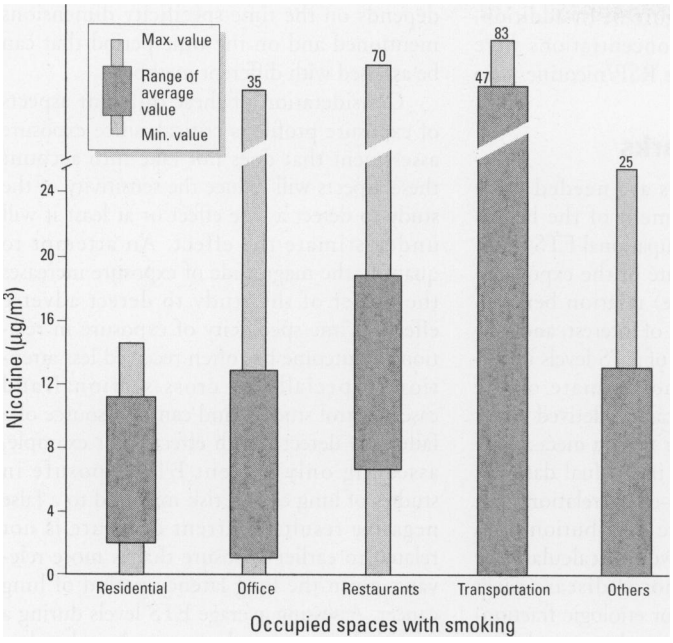

Figure 3. Range of average nicotine concentrations from selected studies and maximum and minimum values related to smoking occupancy in different indoor environments. Abbreviations: max., maximum; min., minimum. For residential and office environments, only studies with sampling time of 4 hr or greater were included. Reprinted from the report by the U.S. Environmental Protection Agency (*1*).

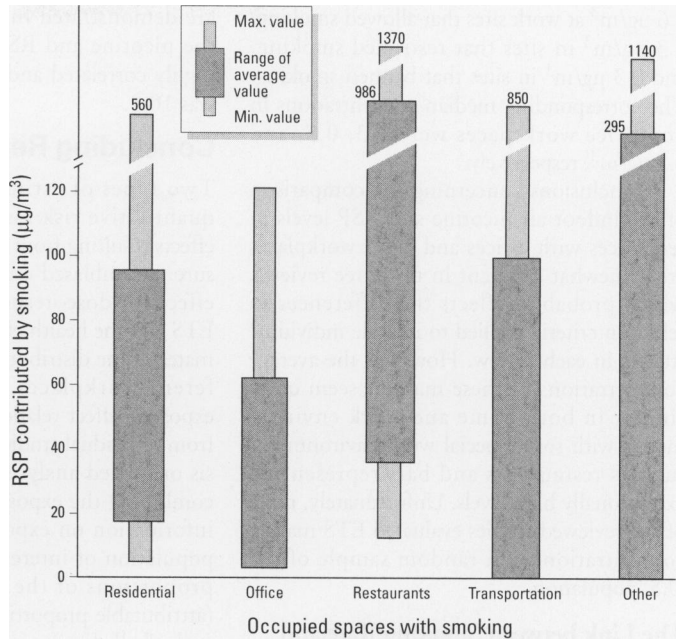

Figure 4. Range of average RSP concentrations from selected studies and maximum and minimum values related to smoking occupancy in different indoor environments. RSP values represent the contribution over background levels without smoking. For residential and office environments, only studies with sampling time of 4 hr or greater were included. Reprinted from the report by the U.S. Environmental Protection Agency (*1*).

The highest values of both nicotine and RSP concentrations were observed in restaurants and transportation.

More recent studies have usually shown somewhat lower concentrations of nicotine in both residential and work environments. In Stockholm, Sweden, personal monitoring of nicotine and RSP exposures was conducted in 1994 among 190 nonsmokers who were either housewives or househusbands or worked in 1 of 12 selected nonindustrial occupations (*26*). Among ETS-exposed housewives and househusbands, the mean 24-hr concentration of nicotine was 3.1 µg/m^3 (range, 0.2–7.5 µg/m^3); the mean 24-hr concentration of RSP was 51 µg/m^3 (range, 15–154 µg/m^3). Among the working subjects, the mean concentrations for ETS exposure in the home (sampling outside of work hours; mean, 15 hr) were 0.3 µg/m^3 (range, 0.1–1.6 µg/m^3) for nicotine and 27 µg/m^3 (range, 7.4–63 µg/m^3) for RSP. The mean concentrations for work exposure (sampling during work hours; mean, 7 hr) were 0.5 µg/m^3 (range, 0.1–3.1 µg/m^3) for nicotine and 24 µg/m^3 (range, 9.7–70 µg/m^3) for RSP. In a large study of 16 metropolitan areas of the United States in 1993–1994, personal sampling was performed in 1,498 subjects representing a variety of occupations in both office and nonoffice environments (*27*). The smoking status of the environment was first classified by response to a screening questionnaire, then confirmed by a diary report. Subjects with salivary cotinine levels of 15 ng/mL or more were excluded as potential active smokers. The mean 8-hr nicotine and RSP concentrations related to ETS exposure in the workplace were 2.4 µg/m^3 (95th percentile, 10.8 µg/m^3) and 49.4 µg/m^3 (95th percentile, 145 µg/m^3), respectively. The corresponding mean 16-hr concentrations related to home exposure (nonwork exposure including possible exposures while shopping, commuting, dining out, etc.) were 2.7 µg/m^3 (95th percentile 7.9 µg/m^3) and 44.1 µg/m^3 (95th percentile, 125 µg/m^3), respectively.

Table 5 presents a summary of the geometric mean and median air nicotine concentrations as well as the range of concentrations in different occupational settings and in homes in the United States, according to the recent review by Hammond (*25*). The values for occupational concentrations are from measurements in work areas of nonsmokers. The mean nicotine concentrations in offices allowing smoking were generally between 2 and 6 µg/m^3, and in diverse blue-collar occupations mean concentrations were between 1 and 6 µg/m^3, although some workplaces had higher means. In homes of smokers, the mean nicotine concentrations were generally between 1 and 3 µg/m^3, indicating slightly lower ETS exposure levels in residential than in work environments. The mean and median concentrations were somewhat higher in office environments than in other work environments, probably attributable to the larger size and better ventilation of the nonoffice work areas. In a large study of 25 Massachusetts work sites, including office environments and different production work areas, work site smoking policy had a strong effect on indoor air nicotine concentrations (*28*). The median values in offices were

Table 5. Summary of geometric mean and median air nicotine concentrations and minimum and maximum concentrations in different types of workplaces allowing smoking as well as in homes in the United States.[a,b]

Microenvironment	Range in geometric mean air nicotine concentration (µg/m^3)	Range in median air nicotine concentration (µg/m^3)	Minimum–maximum of air nicotine concentrations (µg/m^3)
Offices	0.80–48.32	LD or 0.60–48.35	LD or < 0.05–71.50
Nonoffice workplaces, including manufacturing (chemicals, dyes, paper products, etc.), fire stations, railroads, barber shops, hospitals, and aircraft	0.18–16.80	0.10–10.00	< 0.05–126.00
Homes	1.50–5.80	1.00–3.30	0.10–28.60

LD, limit of detection. [a]Based on review by Hammond (*25*). [b]The concentrations in occupational settings are measured in work areas of nonsmokers.

8.6 μg/m^3 at work sites that allowed smoking, 1.3 μg/m^3 in sites that restricted smoking, and 0.3 μg/m^3 in sites that banned smoking. The corresponding median concentrations in nonoffice workspaces were 2.3, 0.7, and 0.2 μg/m^3, respectively.

Conclusions concerning the comparison of the indoor air nicotine and RSP levels in residences with offices and other workplaces are somewhat different in the three reviews, which probably reflects the differences in selection criteria applied to include individual studies in each review. However, the average concentrations of these markers seem quite similar in both home and work environments, with some special work environments such as restaurants and bars representing exceptionally high levels. Unfortunately, none of the reviewed studies evaluated ETS marker concentrations in a random sample of the U.S. population.

The Link between Smoking Rate and Indoor Air Marker Concentrations

Estimates of the relation between smoking rate in residences or workplaces and indoor air concentrations of nicotine or RSP provide an important link for risk assessment because most of the human studies on health effects of ETS have assessed ETS exposure based on questionnaire reports. Several experimental studies as well as field studies with realistic smoking conditions have applied mass balance models to predict ETS concentrations from cigarette smoking rate in indoor spaces (*29*). Indoor concentrations of RSP, CO, or nicotine have been measured to validate these models, and good agreement was seen between observed and predicted indoor concentrations (*29–32*). In a recent article including theoretical considerations and a review of previous studies, Ott concluded that cigarette smoking is well suited for making accurate predictions of marker concentrations applying the mass balance law (*29*), which is stated as follows: "The average concentration in a well-mixed indoor setting is computed as the source strength divided by the product of the volume of the setting and the air change rate of the setting."

Table 6 presents estimates of the relation between questionnaire-reported smoking rate in residences or workplaces and indoor concentrations of nicotine and/or RSP from selected studies. In 1986 Leaderer and Hammond (*33*) studied 96 residences in the Onondaga and Suffolk counties of New York State. They found linear relations between diary-reported total number of cigarettes smoked during 1 week in the residences and 1-week residential nicotine and RSP levels. Monitoring of nicotine and RSP was conducted in the main living area (living room or family room). These relations are demonstrated in Figure 5. In addition, the nicotine and RSP concentrations were highly correlated and the RSP/nicotine ratio was 10.8.

Concluding Remarks

Two types of estimates are needed for a quantitative risk assessment of the health effects resulting from occupational ETS exposure: an unbiased estimate of the exposure–effect (or dose–response) relation between ETS and the health effect of interest, and estimates of the distribution of ETS levels in different workplaces. The estimate of the exposure–effect relation can be derived either from individual studies or from a meta-analysis or pooled analysis of individual data. By combining the exposure–effect relation with information on exposure distribution for a population of interest, we can calculate the proportions of the exposed disease cases (attributable proportion or etiologic fraction) and of all disease cases in this population (population-attributable proportion) attributable to occupational ETS exposure. We can also estimate the excess number of cases resulting from specified exposure conditions.

Several dimensions of the exposure profile should be taken into account when assessing ETS exposure for estimating an exposure–effect relation. These include the magnitude of exposure and the biologically relevant time specificity of exposure (*9*). The magnitude of exposure is determined by the ETS source strength, the environmental factors that modify concentrations, and the duration of exposure. The magnitude of biologically effective dose is determined also by factors affecting uptake of ETS and metabolism of compounds absorbed. Time specificity–related issues include consideration of the latency period for each health outcome of interest, the time–exposure profile relevant for different disease mechanisms, and the sensitive age period with regard to different health effects. The most appropriate indicator of ETS for exposure assessment for each health outcome depends on the time specificity dimensions mentioned and on the time period that can be assessed with different methods.

Consideration of these different aspects of exposure profile is critical, since exposure assessment that does not take into account these aspects will reduce the sensitivity of the study to detect a true effect or at least it will underestimate the effect. An attempt to quantify the magnitude of exposure increases the power of the study to detect adverse effects. Time specificity of exposure in relation to outcome has often received less attention, especially in cross-sectional and case–control studies, and can be a source of a failure to detect health effects. For example, assessing only current ETS exposure in studies of lung cancer risk may lead to a false negative result if current exposure is not related to earlier exposure that is more relevant, given the long latency period of lung cancer. Assessing average ETS levels during a specified time period may not be related to

Table 6. Estimates of the relation between questionnaire-reported smoking rate in residences or workplaces and indoor air concentrations of RSP and nicotine from selected studies.

Quantity of smoking, reference	RSP conc.[a] (μg/m^3)	Nicotine conc.[a] (μg/m^3)
Per 1 smoker in home, Spengler et al. (*34*)	20	–
Per ≥ 1 smokers in home, Coultas et al. (*35*)	17.3	2.1
Per pack[b] (total amount smoked in a week), Leaderer and Hammond (*33*)	6.4	0.6

[a]Sampling periods were 24 hr in Spengler et al. (*34*), 24 hr in Coultas et al. (*35*), 1 week in Leaderer and Hammond (*33*). [b]One pack = 20 cigarettes.

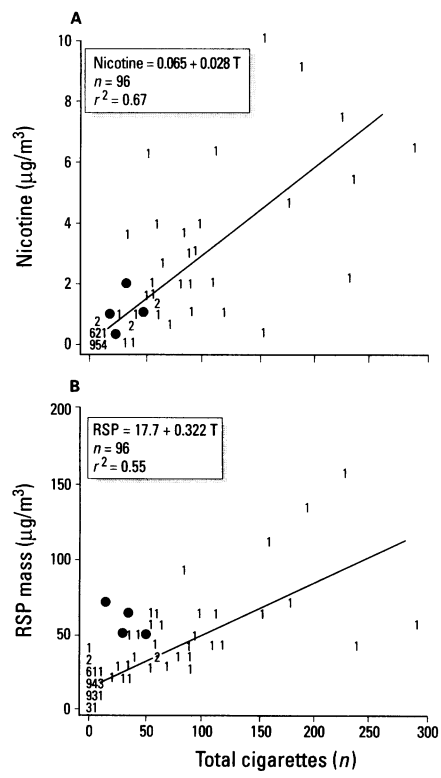

Figure 5. (*A*) Vapor-phase nicotine and (*B*) RSP concentrations measured during 1 week in 96 residences as a function of the number of reported cigarettes smoked (T) in these residences during the sampling period. *n*, the number of residences that were studied. r^2, the proportion of variation in nicotine/RSP concentrations explained by the regression model. Numbers 1–9 refer to the number of observations at the same concentrations. Closed circles indicate that cigar or pipe smoking was reported. Reprinted from Leaderer and Hammond (*33*) with permission of the American Chemical Society.

exacerbations of asthma, whereas high peak exposures may be followed by increased episodes of asthma exacerbation. A third example is studies of effects of fetal exposure; the developmental period during which exposure takes place is essential with regard to detecting health effects.

In three extensive reviews of indoor air nicotine and/or RSP concentrations in different microenvironments, the levels were essentially comparable between work and residential environments in the United States and other countries (*1,12,25*). According to the most recent review of nicotine concentrations, the levels are slightly higher in the workplace compared to residential environments where smoking takes place (*25*). The nicotine levels are somewhat higher in offices compared to blue-collar occupational settings, probably attributable to larger size and better ventilation of nonoffice work areas. In some special work environments such as bars and restaurants, the levels may be extremely high. Data from experimental and field studies show good agreement between ETS marker indoor concentrations predicted from cigarette smoking with models applying mass balance law and measured concentrations of RSP, CO, and nicotine. Some data are available on the relations between questionnaire-reported rate of smoking and indoor ETS marker concentrations. However, more research is needed to achieve more accurate and precise estimates of the relations between questionnaire-reported amount of smoking and indoor air marker concentrations, since most of the health effect studies have based ETS exposure assessment on questionnaires. When better estimates are obtained, information on the distribution of ETS exposure levels in different occupational settings can be better used for assessing health risks due to workplace ETS exposure.

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