Title
Inhalation of hazardous air pollutants from environmental tobacco smoke in US residences

Journal
Journal of Exposure Analysis and Environmental Epidemiology, 14(Supplement 1)

Authors
Nazaroff, William W
Singer, B C

Publication Date
2004

Peer reviewed

Submitted to *Journal of Exposure Analysis and Environmental Epidemiology* April 1, 2003
Revised: July 11, 2003

Inhalation of Hazardous Air Pollutants from Environmental Tobacco Smoke in US Residences

William W Nazaroff [a,b,1] and Brett C. Singer [b]

[a] *Department of Civil and Environmental Engineering, University of California, Berkeley, California, USA*

[b] *Environmental Energy Technologies Division, Ernest Orlando Lawrence Berkeley National Laboratory, Berkeley, California, USA*

Index Terms

Exposure, Health risk, Indoor air quality, Passive smoking, Secondhand smoke, Toxic air contaminants

Running Head

Inhalation of HAPs from ETS in US Residences

[1] Address all correspondence to: Prof. William W Nazaroff, Department of Civil and Environmental Engineering, 633 Davis Hall, University of California, Berkeley, CA 94720-1710 USA. Tel.: +1-510-642-1040. Fax: +1-510-642-7483. E-mail: nazaroff@ce.berkeley.edu

Abstract

In the United States, 48 million adults smoke 3.5-5 × 10^{11} cigarettes per year. Many cigarettes are smoked in private residences causing regular environmental tobacco smoke (ETS) exposure to roughly 31million nonsmokers (11% of the US population), including 16 million juveniles. (Upper bound estimates are 53 million exposed nonsmokers including 28 million juveniles.) ETS contains many chemical species whose industrial emissions are regulated by the US federal government as hazardous air pollutants (HAPs). In this paper, average daily residential exposures to and intakes of 16 HAPs in ETS are estimated for US nonsmokers who live with smokers. The evaluation is based on material-balance modeling; utilizes published data on smoking habits, demographics, and housing; and incorporates newly reported exposure-relevant emission factors. The ratio of estimated average exposure concentrations to reference concentrations is close to or greater than one for acrolein, acetaldehyde, 1,3-butadiene and formaldehyde, indicating potential for concern regarding noncancer health effects from chronic exposures. In addition, lifetime cancer risks from residential ETS exposure are estimated to be substantial (~ 2-500 per million) for each of five known or probable human carcinogens: acetaldehyde, formaldehyde, benzene, acrylonitrile, and 1,3-butadiene. Cumulative population intakes from residential ETS are compared for six key compounds against ambient sources of exposure. ETS is found to be a dominant source of environmental inhalation intake for acrylonitrile and 1,3-butadiene. It is an important cause of intake for acetaldehyde, acrolein, and formaldehyde, and a significant contributor to intake for benzene.

Introduction

Cigarette smoking is an important source of indoor air pollution. Exposure to environmental tobacco smoke (ETS) has been linked to an increased risk of many adverse health outcomes, including lung cancer, asthma onset and exacerbation, and acute respiratory illness (National Cancer Institute, 1999). Concerns have led to restrictions on smoking in public places, including state regulations that severely limit workplace smoking California, Delaware, Maine, Connecticut, New York, and Florida. However, regulatory approaches have limited utility for reducing ETS exposures in private residences. Instead, public education may be best suited to reduce exposures, possibly augmented by technological interventions.

Tobacco smoke comprises a large number of chemical constituents, partitioned to various degrees between the gas and condensed phases. Among the constituents of ETS are chemical compounds that are regulated as hazardous air pollutants (HAPs) by the US federal government (US Environmental Protection Agency, 2003a) and as toxic air contaminants by California (Air Resources Board, 2003). HAPs are species that are known or suspected carcinogens, or that have been shown to cause other serious health effects, such as reproductive problems or birth defects. Characterization and control of HAPs has focused on outdoor sources. However, because of the close proximity between smokers and nonsmokers, and because of the slow dilution of pollutants emitted into indoor spaces, the inhalation intake per unit emission is 100 or more times higher for ETS than for typical outdoor sources (Smith, 1993; Lai et al., 2000).

This paper reports an analysis of exposures to specific ETS-constituent HAPs for nonsmokers who live with smokers. Using concentration guidelines and risk factors, individual HAPs in ETS that pose relatively high risks are identified. The significance of cumulative population intake is also explored in relation to intake from ambient air pollution.

Methods

Overview of Approach

The approach combines a material balance model with published data for key input variables: cigarette consumption patterns, emission factors for HAPs from ETS, residence volume and air-exchange rates, and population statistics. The exposed population of interest is nonsmokers who live with smokers. A primary goal is to provide an estimate of the mean daily exposure to and inhalation intake of specific hazardous air pollutants by members of this exposed population. The assessment only considers the indoor exposure of nonsmokers caused by the cigarette smoking of household members. The cumulative intake by the entire population so exposed is estimated. Health risk information is considered so as to identify specific contaminants, among those assessed, which pose the greatest health risk. The health concerns considered here are long-term risks associated with chronic exposure, including cancer, rather than acute concerns such as odor and irritation.

Cigarette Smoking Habits in the United States

The prevalence of smoking among noninstitutionalized US adults was determined from the Behavioral Risk Factor Surveillance System (BRFSS), a state-based, random-digit-dialed telephone survey. Current smokers are defined as those who reported having smoked more than 100 cigarettes during their lifetime and who currently smoke every day or on some days. We combined state-by-state, gender-specific information from BRFSS with census data to estimate that 47.7 million adults (24.9 million males) smoke cigarettes in the US (Centers for Disease Control and Prevention, 2001; US Census Bureau, 2000), an overall adult smoking prevalence of 22.8%.

The quantity of cigarettes consumed by smokers was estimated from 1997 records reporting an adult, per capita, tax-paid sales rate of 117 packs per year (Tobacco Institute, 1997). Assuming this rate applies to the 2000 US adult population of 209 million, we estimate an annual consumption of about half a trillion (4.9×10^{11}) cigarettes by US smokers, which corresponds to 1.4 packs (= 28 cigs) per day per adult smoker. Lower estimates of total consumption are obtained from surveys such as BRFSS that ask people about their smoking habits. In a nationwide study from the late 1980s, self-reported cigarette consumption was only 72% of tax-based estimates (Hatziandreu et al, 1989). Applying this factor to the data here would indicate a lower bound estimate on annual cigarette consumption of 3.5×10^{11} cigs y^{-1} by US smokers, or 20 cigs d^{-1} $smoker^{-1}$. Recent (2002) tax-based data indicate US sales of 20.4 billion cigarette packs per year, suggesting an intermediate consumption rate of 4.1×10^{11} cigs y^{-1} (National Center for Tobacco-Free Kids, 2003).

How Many People are Exposed to ETS at Home?

We next seek to estimate the number of nonsmokers who are regularly exposed to environmental tobacco smoke (ETS) in their residences. Comprehensive, unbiased estimators do not exist; however, estimates can be constructed from good proxies. The Third National Health and Nutrition Examination Survey (NHANES III) collected ETS exposure data during 1988-1991 on a nationally representative cross-section of 7074 juveniles (aged 2 mo to 16 y) and 9744 adults (aged 17 and older). In this study, exposure to ETS at home was assumed to occur if any household member smoked, a condition that was reported for 42% of nonsmoking juveniles and 17% of nonsmoking adults (Pirkle et al., 1996). Applying these proportions to the year 2000 total US population of nonsmoking juveniles (66 million, aged 16 or less) and nonsmoking adults

(150 million, aged 17 and over) yields an estimate of 53 million nonsmokers who would be exposed to ETS in their homes of whom 28 million are juveniles. We take this estimate to represent an upper bound, since it does not exclude homes with smokers who do not smoke indoors.

As part of the BRFSS in 1996, data were collected on the prevalence of households with current adult cigarette smokers and any children and adolescents in the home (Centers for Disease Control and Prevention, 1997). The same investigation collected information on whether smoking was permitted in some or all areas of the home. This study estimated that the number of juveniles (age < 18 y) exposed to ETS at home was 15 million, equal to 22% of all US juveniles at that time. The total US population of juveniles in 2000 was 73 million and 22% of this total is 16 million. Correcting the NHANES III adult data in the same way, the proportion of nonsmoking adults regularly exposed to ETS in their own homes would be estimated to be 9%, which corresponds to 15 million people. Thus, this estimate suggests that 31 million nonsmokers are regularly exposed to ETS in their own homes because they live with smokers, and 16 million of those exposed are juveniles. We take this to represent a central estimate of the current size of the exposed population. Note that Schuster et al. (2002) reported that 21 million US children lived in households in which smoking regularly occurred by occupants or visitors, a value that is consistent with our two estimates for juveniles.

Exposure-Relevant Emission Factors

Important input into this analysis is the effective rate at which HAPs are emitted in ETS when a cigarette is smoked. Emission factors have been measured for many ETS constituents in special test chambers. However, recent work indicates that airborne concentrations of some ETS

constituents can be significantly affected by sorptive interactions with indoor surfaces (Singer et al., 2002; Singer et al., in press). Sorption can reduce concentrations and short-term exposures, relative to those predicted using an emission factor measured under low-sorption conditions (e.g., an unfurnished metal chamber). We address this issue with the concept of an exposure-relevant emission factor (EREF) that implicitly incorporates sorption effects under realistic furnishing and ventilation conditions. For this analysis, we are interested in ETS concentrations that result from conditions of regular daily smoking. For the central tendency, we use average EREFs that were derived by mass balance from gas-phase ETS concentrations measured during the fourth (and final) week of two experiments conducted in a furnished 50-m^3 room. In one experiment, 10 cigarettes were smoked each day, 6 days per week, and the ventilation rate was 0.6 h^{-1}. In the second experiment, 5 cigarettes were smoked each day, 7 days per week, and the ventilation rate was 0.3 h^{-1}. We believe that these emission factors represent the best currently available basis for this analysis. However, some uncertainty remains because the EREF experiments were conducted with machine smoked cigarettes in a one-room test facility over only a one-month period. The influence of these conditions on effective emissions is unknown as compared with chronic smoking by humans in residences. However, the method presented here can easily be updated when improved emissions information becomes available.

Quantifying Exposures to ETS and its Constituents

In keeping with the goal of providing an assessment that is objective, transparent, and as accurate as the empirical data will support, the daily residential exposure to and intake of HAPs from ETS by nonsmokers is estimated by means of a material-balance model. We define the following variables: N is the number of cigarettes smoked per day in a home (cig d^{-1}); V is the

building volume (m^3); A is the number of indoor-outdoor air exchanges per day (d^{-1}); E_i is an exposure-relevant emission factor for the HAP of concern (μg cig^{-1}); Q_B (m^3 d^{-1}) is the rate of air inhalation by a nonsmoker; and f (dimensionless) is an exposure factor that adjusts for potential exposures that do not occur because the nonsmoker is not always at home indoors.

The daily time-averaged residential concentration, C_i, of the HAP caused by ETS for homes where smoking occurs is estimated using equation (1):

$$C_i = \frac{NE_i}{AV} \qquad (1)$$

This equation is based on the principle of mass conservation: the daily mass of a pollutant added to the indoor air by smoking ($N E_i$) is taken to equal the daily mass of that pollutant removed by ventilation (estimated as $C_i A V$).

Note that a similar equation may be obtained when an indoor environment is represented as a single well-mixed volume for which a steady-state approximation is applied. However, these conditions are not needed to derive equation (1). Rather, species concentrations are permitted to vary in time and space, as would be expected with ETS. Equation (1) is developed by means of solving the differential material-balance equation for the time-averaged species concentration. For the equation to be valid, the sorptive interactions in the test chamber when the EREF is measured must be similar to those in the residences where exposure occurs. In addition, the time-averaged species concentration in air ventilated from the building must equal the time-averaged and spatially averaged concentration within the building. And finally, the only important processes that can affect ETS species dynamic behavior must be emissions, sorption, and ventilation.

Having determined the ETS species concentration, we next estimate the average *exposure* concentration. The parameter we seek is the time-average concentration in the breathing zone of the nonsmoker, including not only the periods when they are at home but also when they are away. Because this assessment focuses on the contribution of a specific source — environmental tobacco smoke — we estimate the *attributable* exposure concentration, which is the increment in time-average exposure concentration, X_i, caused by the cigarette smoking in the homes of exposed nonsmokers. Our estimate is obtained by multiplying the result of equation (1) by an exposure factor, *f*,

$$X_i = f \frac{NE_i}{AV} \tag{2}$$

Estimates indicating the likely range of *f* values are discussed below.

Another parameter of interest is the rate of *inhalation intake* of HAPs from environmental tobacco smoke. Expressed in units of mass per time, the intake, I_i, is obtained as the product of the attributable exposure concentration and the average breathing rate:

$$I_i = f \frac{NE_i}{AV} Q_B \tag{3}$$

To generate estimates of exposure concentrations, X_i, and intakes, I_i, parameter values that approximate or bound the expected means are selected for terms on the right-hand sides of equations (1)-(3). We estimate that the average number of cigarettes smoked indoors in smokers homes is in the range N = 14-20 cig d^{-1} (= 0.7-1 pack per day). This estimate incorporates an assumption that, in the absence of household restrictions, the average smoker consumes half of their daily cigarettes indoors at home, which follows from an expectation that habitual smokers

will consume cigarettes at a roughly uniform rate throughout the hours that they are awake. Data show that people in the US spend an average of 69% of their time indoors at home (Klepeis et al., 2001), which, allowing for 8 h of sleep per night, implies that about half of the daily awake hours are spent indoors at home. The average number of smokers in a home that includes a nonsmoker is 1.4 (Pirkle et al., 1996); and the average cigarette consumption rate is 1-1.4 packs per smoker per day. Consequently, the indoor cigarette consumption rate is estimated as $N = 0.5 \times 1.4 \times (1\text{-}1.4) \times 20 = 14\text{-}20$ cigs d^{-1}.

The mean daily number of indoor-outdoor air exchanges is estimated to be $A = 9\ d^{-1}$. This estimate is based a nationwide compilation of 2844 air-exchange rate measurements (Murray and Burmaster, 1995), who reported a good fit of the nationwide data to a lognormal distribution with GM = 0.53 h^{-1} and GSD = 2.3. The value of A corresponds to the harmonic mean of the fitted lognormal distribution. The uncertainty in the GM and GSD for this distribution is small compared with other uncertainties in this analysis, and so is not explicitly incorporated. The harmonic mean of A is the most relevant single value for estimating the mean exposure concentration. Because A appears in the denominator of the right-hand sides of equations (1)-(3), the average of the parameters on the left-hand side vary in proportion to the average of the inverse of A across the distribution in the building stock.

The residence volume is taken to be $V = 375\ m^3$, based on an analysis of data from the American Housing Survey for US single-family detached and mobile homes (US Census Bureau, 2002). A lognormal distribution was fitted to the population-weighted home floor area; households occupied by only one person were excluded. The result was a GM of 1830 ft^2 (170 m^2) and a GSD of 1.6. The result was converted to volume assuming a ceiling height of 8 ft (2.4

m) and the harmonic mean of 375 m^3 was computed from the properties of a lognormal distribution. Without weighting for number of occupants and without excluding single-person households, the harmonic mean volumes are estimated to be 340 m^3 for all US households and 360 m^3 for single-family detached and mobile homes. The fractional differences among these three numbers are small compared with other uncertainties and so are not explicitly included in developing bounding estimates.

The volume of air breathed daily is taken to be $Q_B = 12\ m^3\ d^{-1}$, which represents the average of the lifetime average values for males ($14\ m^3\ d^{-1}$) and females ($10\ m^3\ d^{-1}$), following Layton (1993).

The exposure factor, *f*, depends on the fraction of time spent indoors at home by nonsmokers, and on the timing of their presence, relative to smoking events. This factor also depends on the distribution and mixing of ETS constituents within the home. We estimate that the average value of *f* very likely lies in the range 0.64-1. The lower bound corresponds to the average fraction of air breathed in one's home, based on our analysis of Layton's data in combination with activity pattern data (Klepeis et al., 2001). This case would apply if the average indoor air breathed by a nonsmoker contained the same ETS concentrations as the average ventilation air leaving the building. The upper bound corresponds approximately to the condition that would obtain if the nonsmoker were present in the home during, and for several hours after cigarette consumption by the smoking resident(s). Relative to this range, to the extent that a nonsmoker is not present during and shortly after smoking, the factor *f* would be reduced for that individual. Conversely, if a nonsmoker were regularly present in the same room as the smoker during smoking events, that individual's exposure concentration could be higher than the range estimated here.

Health-Risk Information: Reference Concentrations and Unit Risk Factors

For hazardous air pollutants that do not cause cancer, the health hazard of chronic exposure is commonly determined by comparing an exposure concentration to a concentration guideline. For this purpose, the US Environmental Protection Agency maintains a list of reference concentrations (RfC), defined to be "an estimate of a continuous inhalation exposure to the human population that is likely to be without an appreciable risk of deleterious effects during a lifetime" (US Environmental Protection Agency, 2003b). California's Environmental Protection Agency has developed chronic inhalation reference exposure levels (REL) for 120 individual air toxicants (Office of Environmental Health Hazard Assessment, 2003), which represent an estimate of the airborne concentration to which individuals may be indefinitely or routinely exposed with no associated significant health risk. A hazard index is computed for each compound as the ratio of the exposure concentration to the respective RfC or REL. Since ETS is just one of many sources of pollution exposure, a hazard index near or in excess of unity would constitute a potential cause for concern.

The risk of exposure to carcinogens is commonly determined by multiplying an exposure concentration times a risk factor. The product represents an estimate of the incremental increase in lifetime cancer risk caused by a lifetime of exposure to that carcinogen at the indicated level. Both the USEPA and CalEPA maintain lists of risk factors for carcinogenic HAPs (US Environmental Protection Agency, 2003b; Office of Environmental Health Hazard Assessment, 2002). Lifetime cancer risks owing to environmental air-pollutant exposure within or above the range of $(1\text{-}100) \times 10^{-6}$ can trigger regulatory or permitting action to restrict emissions.

Intake of Hazardous Air Pollutants from Ambient Sources

Perspective on the significance of ETS exposure to HAPs can be gained by comparing ETS intake to the intake associated with ambient sources. The USEPA has published a national-scale air toxics assessment to "identify those air toxics which are of greatest potential concern, in terms of contribution to population risk." (US Environmental Protection Agency, 2003a). The assessment considered 32 air toxics plus diesel particulate matter. Among the published results are estimated mean exposure concentrations for the population of the contiguous United States. The study did not consider exposures other than those resulting from ambient air pollution. We estimated the total inhalation intake of ambient HAPs by the US population as the product of three terms: the US population (280 million), the daily average breathing rate (12 m^3 d^{-1}), and the average exposure concentration for ambient HAPs, as reported by the USEPA.

Results

Table 1 summarizes the general input data used throughout the evaluation. We report a range of values for two parameters that have a relatively large uncertainty —average number of cigarettes smoked in residences in which nonsmokers live (N), and the exposure parameter (f). We also report a central plus upper bound estimate for population of nonsmokers exposed in their residences. Other parameters are reported as point estimates because they contribute much less than these three to the overall uncertainty in our results.

Table 2 presents a summary of exposure and intake estimates for HAPs in residential ETS at the level of an individual nonsmoker. The exposure concentration, evaluated using equation (2), represents the estimated mean increase in HAP concentration in the breathing zone of nonsmokers owing to ETS in their homes. The population for which this estimate applies is

all nonsmokers who live with one or more smokers in homes where smoking is permitted. Only the contribution to ETS exposures from occupants is considered; smoking by visitors is not included. Likewise, exposures to ETS outside the home are not included in this evaluation. The range represents a bounding estimate, accounting for the full ranges of values reported here for N and f. The inhalation intake is obtained as the product of the exposure concentration and the average breathing rate.

Table 3 presents risk estimates from residential exposure to HAPs in ETS. A range is reported for the hazard index and the cancer risk for each species. This range accounts for the uncertainty in mean exposure, reflected in the range of exposure concentrations reported in Table 2 and reproduced in Table 3. The range in risk estimates also reflects the differences for some species in reference concentrations or risk factors between the USEPA and the California EPA.

Presented in bold face in Table 3 are the species for which at least one of these criteria is met: (a) the upper bound hazard index exceeds 1.0; or (b) the lifetime cancer risk exceeds 10^{-6}. The six species so highlighted have the potential to pose significant risk owing to residential ETS exposure. For noncancer effects, the greatest concern among these species is for acrolein, with a hazard index in the range 80-180 for mean exposures. Acrolein irritates the mucous membranes in the eyes and respiratory tract. The limiting concern for chronic exposures is histological lesions in the upper respiratory tract. Acetaldehyde, 1,3-butadiene, and formaldehyde exhibit a hazard index that is of the order of magnitude of unity and, in each case, exceeds 1.0 for part or all of the range.

Five of the HAPs considered in this study are classified as known or probable human carcinogens. For each of the five, lifetime exposure at the average residential ETS level is estimated to yield a lifetime cancer risk well in excess of 10^{-6}. The largest risks are from 1,3-

butadiene (42-530 per million) and acrylonitrile (34-350 per million). Risks are also large for formaldehyde (15-73 per million), acetaldehyde (14-38 per million), and benzene (2-73 per million).

For the six highlighted compounds, we have estimated both the cumulative population intake and the mean individual intake in the United States owing either to residential ETS or to all ambient sources (see Figure 1). Considering cumulative population intake (Figure 1a), only for benzene does the contribution from ambient sources greatly exceed that from residential ETS. For formaldehyde, population intake from ambient sources appears to be moderately higher than from residential ETS, a situation that is reversed for acetaldehyde and acrolein. For 1,3-butadiene and acrylonitrile, the intake from residential ETS substantially exceeds that from ambient sources.

Figure 1b illustrates that, among nonsmokers who live with smokers, ETS can be a dominant source of inhalation intake for five of the six highlighted compounds. Only for benzene are ambient sources even comparable. The entire burden of population intake from residential ETS is borne by the estimated 11% of the US population so exposed, in contrast to the burden of intake owing to ambient sources which is borne by the entire population. This difference leads to the order-of-magnitude shift in the relative contributions of ambient sources and residential ETS between Figures 1a and 1b.

Discussion

Most health-risk evaluations of exposure to environmental tobacco smoke are based on epidemiological investigations that use questionnaires or marker compounds (e.g., cotinine in body fluids) to estimate exposure. The approach presented here is complementary, as it identifies some specific compounds in ETS that contribute significantly to overall health risks.

The results of our study indicate that six hazardous air pollutants in ETS — acetaldehyde, acrolein, acrylonitrile, benzene, 1,3-butadiene, and formaldehyde — should be of particular concern as contributors to health risk from chronic, residential ETS exposure. Future studies may usefully be focused on better characterizing exposure to these compounds from ETS and on the effectiveness of intervention measures to reduce such exposures.

Acknowledgments

This research was supported by the Cigarette and Tobacco Surtax Fund of the State of California through the Tobacco-Related Research Program of the University of California, Award 7RT-0099. Additional support was provided by the U.S. Department of Energy under Contract No. DE-AC03-76SF00098.

References

Air Resources Board. *California Air Toxics Program.* 2003. California Environmental Protection Agency. Accessed at http://www.arb.ca.gov/toxics/toxics.htm.

Centers for Disease Control and Prevention. State-specific prevalence of cigarette smoking among adults, and children's and adolescent's exposure to environmental tobacco smoke — United States, 1996. *MMWR* 1997; 46:1038-1043.

Centers for Disease Control and Prevention. State-specific prevalence of current cigarette smoking among adults, and policies and attitudes about secondhand smoke — United States, 2000. *MMWR* 2001; 50:1101-1106.

Hatziandreu EJ, Pierce JP, Fiore MC, Grise V, Novotny TE, Davis RM. The reliability of self-reported cigarette consumption in the United States, *Am J Public Health* 1989; 79:1020-1023.

Klepeis NE, Nelson WC, Ott WR, Robinson JP, Tsang AM, Switzer P, Behar JV, Hern SC, Engelmann WH. The National Human Activity Pattern Survey (NHAPS): A resource for assessing exposure to environmental pollutants. *J Exposure Anal Environ Epidemiol* 2001; 11:231-252.

Lai ACK, Thatcher TL, Nazaroff WW. Inhalation transfer factors for air pollution health risk assessment. *J Air Waste Manage Assoc* 2000; 50:1688-1699.

Layton DW. Metabolically consistent breathing rates for use in dose assessments. *Health Phys* 1993; 64:23-36.

Murray DM, Burmaster DE. Residential air exchange rates in the United States: Empirical and estimated parametric distributions by season and climatic region. *Risk Anal* 1995; 15:459-465.

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

National Center for Tobacco-Free Kids. *State Cigarette Tax Rates & Rank, Date of Last Increase, Annual Pack Sales & Revenues, and Related Data.* 2003. Accessed at http://tobaccofreekids.org/research/factsheets/pdf/0099.pdf.

Office of Environmental Health Hazard Assessment. *Hot Spots Unit Risk and Cancer Potency Values*. 2002. California Environmental Protection Agency. Accessed at http://www.oehha.ca.gov/air/hot_spots/pdf/TSDlookup2002.pdf.

Office of Environmental Health Hazard Assessment. *Technical Support Document for the Determination of Noncancer Chronic Reference Exposure Levels*. 2003. California Environmental Protection Agency. Accessed at http://www.oehha.ca.gov/air/chronic_rels/agency.html.

Pirkle JL, Flegal KM, Bernert JT, Brody DJ, Etzel RA, Maurer KR. Exposure of the US population to environmental tobacco smoke. *JAMA* 1996; 275:1233-1240.

Schuster MA, Franke T, Pham CB. Smoking patterns of household members and visitors in homes with children in the United States. *Arch Ped Adolescent Med* 2002; 156: 1094-1100.

Singer BC, Hodgson AT, Guevarra KS, Hawley EL, Nazaroff WW. Gas-phase organics in environmental tobacco smoke. 1. Effects of smoking rate, ventilation, and furnishing level on emission factors. *Environ Sci Technol* 2002; 36:846-853.

Singer BC, Hodgson AT, Nazaroff WW. Gas-phase organics in environmental tobacco smoke. 2 Exposure-relevant emission factors and indirect exposures from habitual smoking. *Atmos Environ* (in press).

Smith KR. Fuel combustion, air pollution exposure, and health: The situation in developing countries. *Annu Rev Energy Environ* 1993; 18:529-566.

Tobacco Institute. *The Tax Burden on Tobacco: Historical Compilation*. 1997; 32:33. Accessed at http://www.tobaccoinstitute.com/.

US Census Bureau. *American Fact Finder.* 2000; Geographic Comparison Table, GCT-P5. Age and sex: 2000. Accessed at http://factfinder.census.gov/.

US Census Bureau. *American Housing Survey for the United States: 2001*. 2002. Series H150/01. US Government Printing Office, Washington DC. Accessed at http://www.census.gov/hhes/www/ahs.html.

US Environmental Protection Agency. *Air Toxics Website*. 2003a. Accessed at http://www.epa.gov/ttn/atw.

US Environmental Protection Agency. *Integrated Risk Information System*. 2003b. Accessed at http://www.epa.gov/iris/.

Figure Caption

Figure 1. Estimated inhalation intake of six hazardous air pollutants in the United States, comparing all ambient sources (circles) with residential environmental tobacco smoke (rectangles). (a) Cumulative intake summed over entire exposed populations of 280 million (ambient) and 31 million (residential ETS). (b) Mean individual intake for members of the exposed populations.

Table 1. Summary of parameters used in exposure and intake analysis

Parameter	Symbol	Value
Total US population	—	281 million
Exposed nonsmokers, juveniles	—	16 million (28 million) [1]
Exposed nonsmokers, adults	—	15 million (25 million) [1]
No. cigarettes smoked indoors at home	N	14-20 d^{-1}
Air-exchange rate	A	9 d^{-1}
Residence volume	V	375 m^3
Breathing rate by nonsmoker	Q_B	12 m^3 d^{-1}
Exposure parameter	f	0.64-1.0

[1] Value in parenthesis represents upper bound estimate of exposed population.

Table 2. Exposure to and individual intake of HAPs from ETS for nonsmokers who live with a smoker.

Compound [1]	EREF (µg cig^{-1}) [1]	Exp. conc. (µg m^{-3}) [2]	Inhalation intake (µg d^{-1}) [3]
Acetaldehyde	2360	6.3-14	75-170
Acetonitrile	1145	3.0-6.8	36-81
Acrolein	610	1.6-3.6	19-43
Acrylonitrile	195	0.5-1.2	6-14
Benzene	430	1.1-2.5	14-31
1,3-Butadiene	515	1.4-3.1	16-37
2-Butanone	325	0.9-1.9	10-23
Cresol	72	0.2-0.4	2-5
Ethylbenzene	150	0.4-0.9	5-11
Formaldehyde	950	2.5-5.6	30-67
Methylnaphthalenes	50	0.1-0.3	2-4
Naphthalene	40	0.1-0.2	1-3
Phenol	190	0.5-1.1	6-14
Styrene	235	0.6-1.4	7-17
Toluene	925	2.5-5.5	29-66
Xylene	510	1.4-3.0	16-36

[1] Exposure-relevant emission factor. Values averaged from experiments conducted at air-exchange rates of 0.3 and 0.6 h^{-1} (except for acetaldehyde and formaldehyde, for which no data are available at 0.3 h^{-1}; and xylene mixtures, for which no data are available at 0.6 h^{-1}). Source: Singer et al., in press.

[2] Estimated range intended to include the daily average incremental concentration attributable to residential ETS in the breathing zone of nonsmokers who are in their homes; assumes N = 14-20 cig d^{-1}, A = 9 d^{-1}, V = 375 m^3, and f = 0.64-1.0.

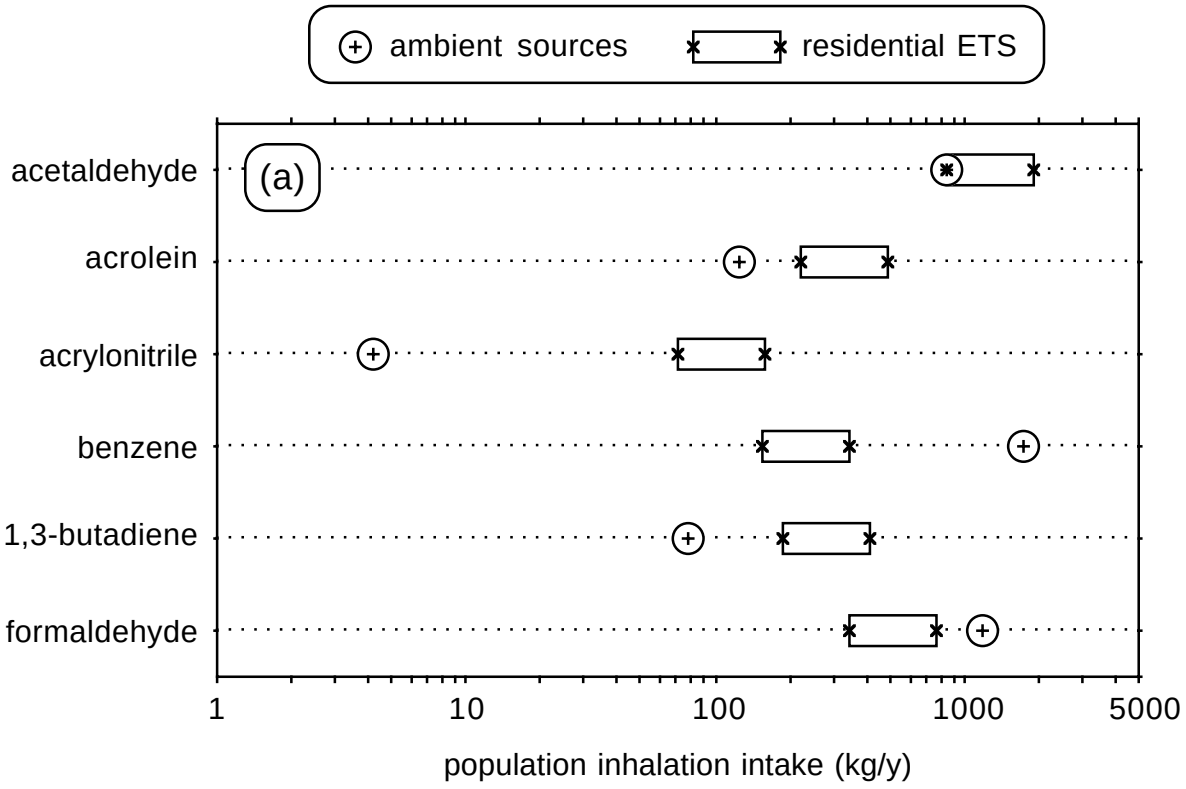
[3] Estimated range intended to include the daily average intake attributable to residential ETS for nonsmokers who are exposed to ETS in their residences; assumes Q_B =12 m^3 d^{-1}.

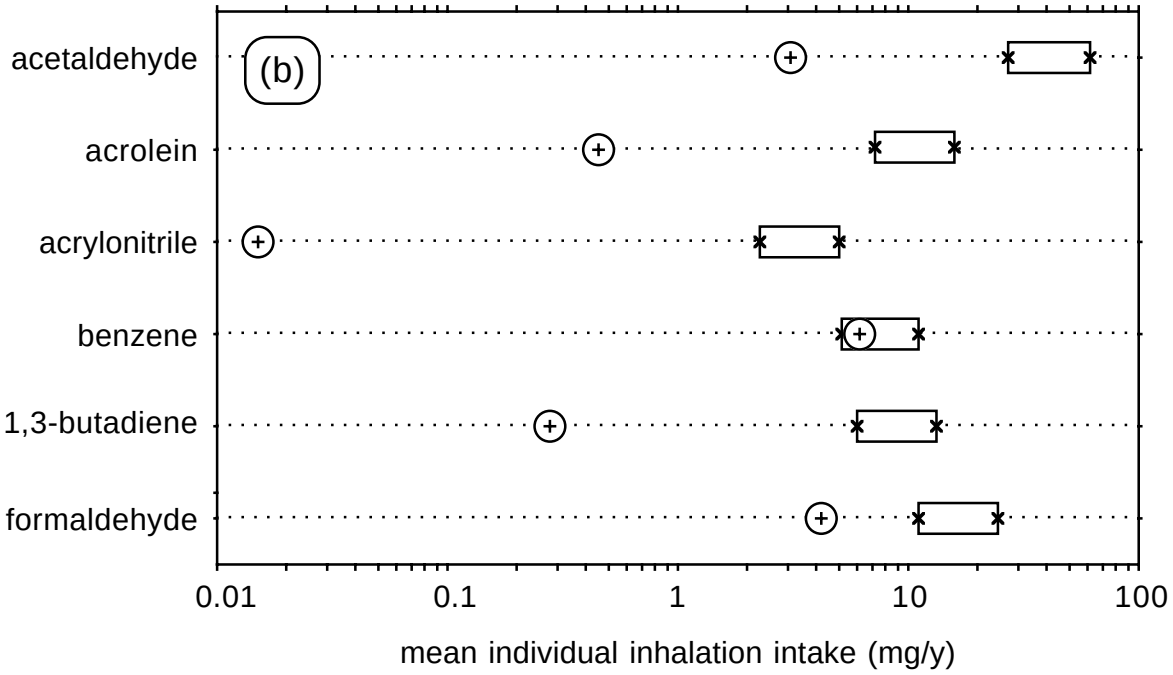
Table 3. Health risk estimates from residential exposure of nonsmokers to HAPs from ETS. [1]

Compound	Exp. conc. (µg m^{-3})	Outcomes other than cancer		Cancer risk	
		RfC, REL (µg m^{-3}) [2]	Hazard index	Risk factor (per µg m^{-3}) [2]	Risk (× 10^{-6})
Acetaldehyde	6.3-14	9	0.7-**1.6**	2.2×10^{-6}, 2.7×10^{-6}	**14-38**
Acetonitrile	3.0-6.8	60	0.05-0.11		
Acrolein	1.6-3.6	0.02	**80-180**		
Acrylonitrile	0.5-1.2	2	0.2-0.6	6.8×10^{-5}, 2.9×10^{-4}	**34-350**
Benzene	1.1-2.5	30, 60	0.02-0.08	2.2×10^{-6}, 7.8×10^{-6}, 2.9×10^{-5}	**2-73**
1,3-Butadiene	1.4-3.1	2, 8	0.2-**1.6**	3.0×10^{-5}, 1.7×10^{-4}	**42-530**
2-Butanone	0.9-1.9	1000	~ 10^{-3}		
Cresol mixtures	0.2-0.4	4	0.05-0.1		
Ethylbenzene	0.4-0.9	1000	< 10^{-3}		
Formaldehyde	2.5-5.6	2	**1.2-2.8**	6×10^{-6}, 1.3×10^{-5}	**15-73**
Methylnaphthalenes	0.1-0.3	na	—		
Naphthalene	0.1-0.2	3, 9	0.01-0.07		
Phenol	0.5-1.1	600	0.001-0.002		
Styrene	0.6-1.4	1000	~ 10^{-3}		
Toluene	2.5-5.5	400	0.006-0.014		
Xylene mixtures	1.4-3.0	100, 200	0.007-0.03		

[1] **Bold-faced** entries either exceed a hazard index of 1.0 (over at least part of the range) or have a cancer risk in excess of 1 per million.

[2] RfC = reference concentration, REL = reference exposure level. References for health risk data: Office of Environmental Health Hazard Assessment, 2002; Office of Environmental Health Hazard Assessment, 2003; US Environmental Protection Agency, 2003b.


ambient sources
residential ETS
(a)
acetaldehyde
acrolein
acrylonitrile
benzene
1,3-butadiene
formaldehyde
1
10
100
1000
5000
population inhalation intake (kg/y)


(b)
acetaldehyde
acrolein
acrylonitrile
benzene
1,3-butadiene
formaldehyde
0.01
0.1
1
10
100
mean individual inhalation intake (mg/y)