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Indoor Air - Assessment

A Review of Indoor Air Quality Risk Characterization Studies

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DISCLAIMER

This document has been reviewed in accordance with U.S. Environmental Protection Agency policy and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

PREFACE

In October of 1986 Congress passed the Superfund Amendments and Reauthorization Act (SARA, PL 99-499) that includes Title IV—the Radon Gas and Indoor Air Quality Research Act. The act directs that EPA undertake a comprehensive indoor air research program.

Research program requirements under Superfund Title IV are specific. They include identifying, characterizing, and monitoring (measuring) the sources and levels of indoor air pollution; developing instruments for indoor air quality data collection; and studying high-risk building types. The statute also requires research directed at identifying effects of indoor air pollution on human health. In the area of mitigation and control the following are required: development of measures to prevent or abate indoor air pollution; demonstration of methods to reduce or eliminate indoor air pollution; development of methods to assess the potential for contamination of new construction from soil gas; and examination of design measures for preventing indoor air pollution. EPA's indoor air research program is designed to be responsive in every way to the legislation.

In responding to the requirements of Title IV of the Superfund Amendments, EPA-ORD has organized the Indoor Air Research Program around the following categories of research: (A) Sources of Indoor Air Pollution, (B) Building Diagnosis and Measurement Methods, (C) Health Effects, (D) Exposure and Risk (Health Impact) Assessment, and (E) Building Systems and Indoor Air Quality Control Options.

EPA is directed to undertake this comprehensive research and development effort not only through in-house work but also in coordination with other Federal agencies, state and local governments, and private sector organizations having an interest in indoor air pollution.

The ultimate goal of SARA Title IV is the dissemination of information to the public. This activity includes the publication of scientific and technical reports in the areas described above. To support these research activities and other interests as well, EPA publishes its results in the INDOOR AIR report series. This series consists of five subject categories: Sources, Measurement, Health, Assessment, and Control. Each report is printed in a limited quantity. Copies may be ordered while supplies last from:

U.S. Environmental Protection Agency
Center for Environmental Research Information
26 West Martin Luther King Drive
Cincinnati, OH 45268

When EPA supplies are depleted, copies may be ordered from:

National Technical Information Service
U.S. Department of Commerce
5285 Port Royal Road
Springfield, VA 22161

ABSTRACT

Risk assessment methodologies provide a mechanism for incorporating scientific evidence and judgments into the risk management decision process. A risk characterization framework has been developed to provide a systematic approach for analysis and presentation of risk characterization study results. This framework was used as a tool to review published studies that provide quantitative risk estimates associated with exposure to indoor air pollutants. Comparisons of both the methods and the resulting risk estimates are presented. Critical assumptions concerning risk estimates and exposure estimates for each study are recorded on the framework.

Fourteen risk characterization studies were reviewed, including three studies for radon, six for environmental tobacco smoke, three for volatile organics, one for formaldehyde, and one for asbestos. The quality and rigor of analysis varied greatly among the studies reviewed. Some of the studies clearly state that they are intended to be preliminary analyses or screening studies, others are reported as sensitivity analyses, and others are detailed risk assessments. Studies which are technically rigorous in some risk components (e.g., dose-response relationships) are often less rigorous in other components (e.g., exposure assessment).

Summary figures are presented which compare individual lifetime cancer risks estimated for each pollutant category and the annual cancer mortality attributable to each pollutant category.

This report is the second in a series of EPA/Environmental Criteria and Assessment Office monographs:

I. DEVELOPMENT OF A RISK CHARACTERIZATION FRAMEWORK
II. A REVIEW OF INDOOR AIR QUALITY RISK CHARACTERIZATION STUDIES
III. USE OF BENZENE MEASUREMENT DATA IN RISK CHARACTERIZATION ESTIMATES: A PRELIMINARY APPROACH
IV. INDOOR CONCENTRATIONS OF ENVIRONMENTAL CARCINOGENS
V. METHODS OF ANALYSIS FOR ENVIRONMENTAL CARCINOGENS

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AUTHORS, CONTRIBUTORS, AND REVIEWERS

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INTRODUCTION

Risk management has emerged as a principal analytical activity for environmental managers within the Federal government, the private sector, and local governments. Risk assessment methodologies provide a mechanism for incorporating scientific evidence and judgments into the risk management decision process. There are four main uses of risk assessments as input for risk management decision making:

1. To estimate an ambient concentration for a specific chemical that will be protective of human health and the environment;

2. To estimate the human health and environmental effects associated with current ambient concentrations of a particular chemical;

3. To estimate the human health and environmental effects associated with releases of one or more chemicals from one or more sources; and

4. To compare estimated risks from specific chemicals in order to set priorities for regulatory actions or motivation for source mitigation.

The objectives of this monograph are to review publications that provide quantitative risk estimates associated with exposure to indoor air pollutants and to compare both the methods and estimates presented in these publications. The focus is to pull together the risk characterization work for indoor air pollutants or pollutant categories. Analyses that included exposure assessments, the assessment of dose-response relationships, and the quantitative characterization of risk were of primary interest. The more qualitative health effects and hazard assessments of the numerous studies that have focused on measuring the indoor pollutant concentration were not reviewed in detail and are not discussed in this monograph.

The Risk Characterization Framework presented in Figure 1 has been developed to provide a systematic approach for analysis and presentation of risk characterization study results. This framework is described in detail in a companion monograph (Naugle et al., 1990). It is applied in this monograph as the basis for review and comparison of risk estimates and risk assessment methodologies associated with indoor air pollutants.

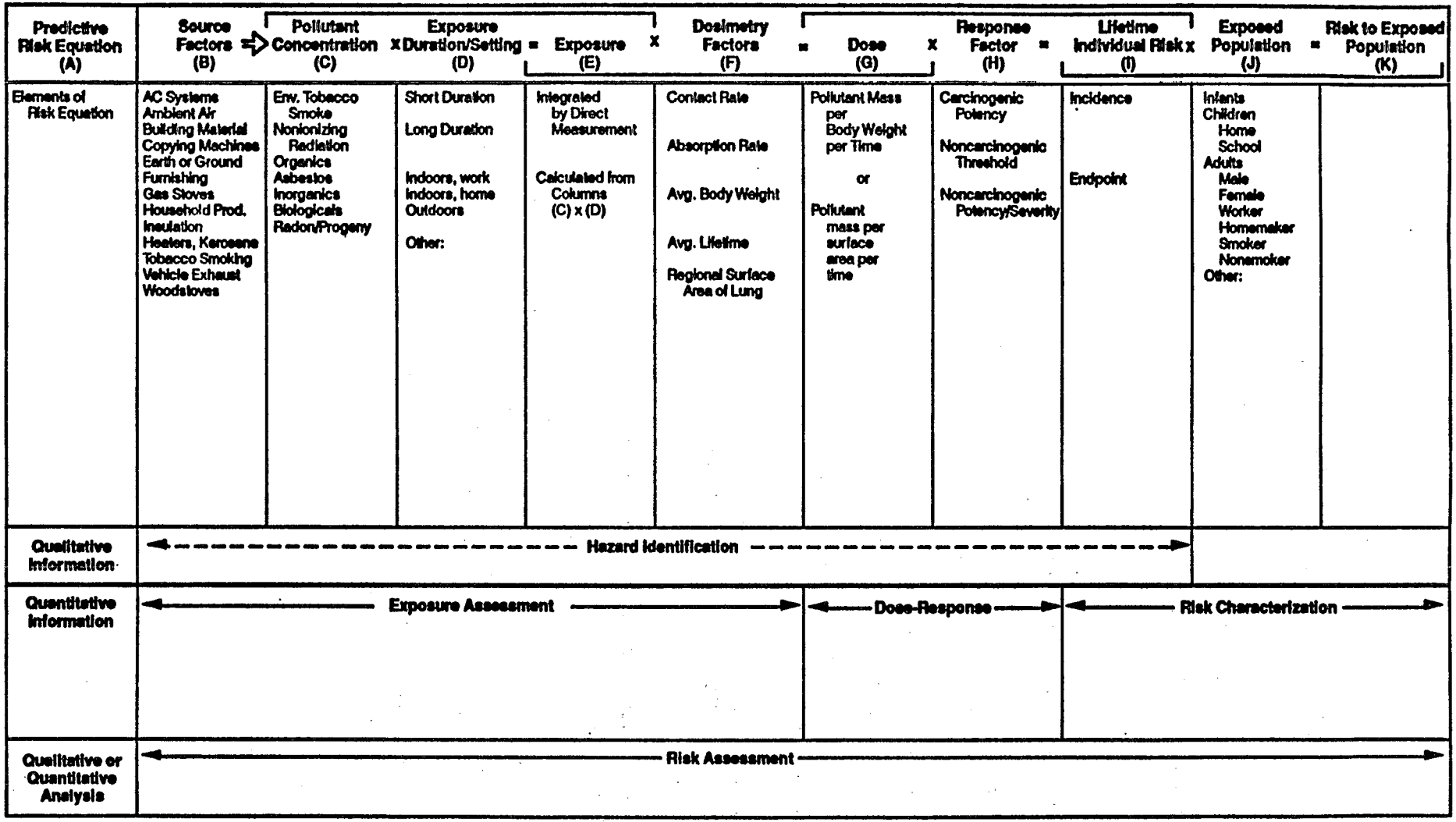

Figure 1. Risk Characterization Framework

SELECTION OF LITERATURE FOR REVIEW

Extensive literature exists on the health effects associated with indoor air pollutants. In order to identify the most relevant information for the purposes of this literature review, the following resources were utilized:

- Recent summary documents on the health effects associated with exposure to indoor air pollutants (i.e., the Samet et al. [1987] articles entitled "Health Effects and Sources of Indoor Air Pollution, Parts I and II", the U.S. Environmental Protection Agency report [1987b] *EPA Indoor Air Quality Implementation Plan* and the appendices, and the *Proceedings of the Fourth International Conference on Indoor Air Quality and Climate* held in Berlin, West Germany, on August 17-21, 1987 [Seifert et al., 1987]);

- The indoor air quality bibliography data base established by the U.S. EPA's Environmental Criteria and Assessment Office (ECAO);

- On-line search using DIALOG MEDLINE and Environmental Abstracts using the key words "indoor air quality and risk or exposure or health effects";

- The bibliographies of documents identified in the previous steps, especially review articles;

- Personal contact with individuals in several Federal agencies, particularly the Centers for Disease Control (CDC) and the National Institute of Occupational Safety and Health (NIOSH), to identify any recent or ongoing research that might be relevant to this project; and

- Individuals who reviewed an earlier draft of this monograph identified additional studies that could be included in this review.

The identified articles were obtained from RTI files, ECAO, or the libraries of local universities and reviewed.

METHODOLOGY

The Risk Characterization Framework was used as a tool for review and analysis of each document. The components of the risk characterization process identified in the Framework served as a guide in the literature review and subsequent analyses. Relevant information on each of these components and the critical assumptions concerning risk estimates and exposure estimates were abstracted from each document and recorded on forms provided to each reviewer.

The use of the Risk Characterization Framework as the guide for the review of each study required a detailed understanding of the risk characterizations presented by the authors, the critical assumptions used in the analysis, and the equations used to estimate risk. During the review process it became clear that authors frequently do not provide key information on all components of the Risk Characterization Framework. For example, the dosimetry factors used to convert exposure to dose are often not explicitly stated. Also, some risk characterizations lack detail in one or more of the ten elements in Figure 1 required to fully evaluate the study. In these instances, the Risk Characterization Framework served as a tool for identifying studies found to be inadequately documented.

For several of the indoor air pollutants, there are numerous studies in the literature that report risk estimates, while for others there are few or no such studies. Radon, for example, is the mostly widely studied of the indoor air pollutants with regard to the characterization of risk. Since many of these radon studies rely on the same basic data for developing each component of the risk characterization process, this report presents a detailed review of three that were viewed as representative of the best available studies. This is also true of the studies reviewed for environmental tobacco smoke (ETS).

The quality and rigor of analysis varied greatly among the studies reviewed. Some studies clearly state that they are intended to be preliminary analyses or screening studies, others are reported as sensitivity analyses, and still others are presented as detailed risk assessments. Even refined risk estimation studies cannot account for all variances in source parameters, exposure duration, and concentrations for various populations, human activities, and complex human physiological factors. Studies which are technically rigorous for some risk components (such as dose-response relationships) are often very weak in other areas

(such as exposure assessments). Although various levels of analysis presented by different authors were recognized, the three "Quality Levels" suggested in the Risk Characterization Framework (Figure 1, column A) were not reported since the criteria for uniform reporting of such levels have not yet been developed. However, from the detailed analysis of the components of the risk characterizations provided, the reader will be able to see the strengths and weaknesses of each study.

OVERVIEW OF RESULTS

Figure 2 summarizes estimates of lifetime risk of cancer or mortality obtained from the risk characterization literature reviewed in this work. For many of the studies included in this review, lifetime risk estimates refer exclusively to lung cancer. However, three of the articles addressing risk attributable to exposure to environmental tobacco smoke (ETS) included endpoints other than lung cancer. The Russell et al. (1986) and Wells (1988) articles include endpoints such as heart disease, bronchitis, and emphysema in addition to lung cancer in the risk estimates. The Fong (1982) article includes emphysema as well as lung cancer in the risk estimates. Also, Mauskopf (1987) provides risk estimates for mesothelioma as well as lung cancer that are attributable to exposure to asbestos. All data shown in Figure 2 are from column I of each Risk Characterization Framework described later in Figures 4 through 19. The ranges shown in this figure are for different point estimates of risk and do not represent a statistical confidence interval or range of uncertainty. Lifetime risk estimates typically refer to the probability of developing cancer over a lifetime for the average individual in a defined population and for a specified exposure scenario over the entire lifetime. Assumptions often are required concerning the exposure scenario and the dose-response relationship. The risk estimates refer only to the probability of developing cancer over a lifetime that is attributable to exposure from the pollutant shown. As can be seen, such estimates have been developed for radon and its decay products, ETS, a number of volatile organics (including formaldehyde), and asbestos. The individual studies from which these estimates are taken are described more fully in the next section.

Figure 3 presents the range of estimates of population risk of cancer obtained from the literature for each pollutant or pollutant category for which such estimates exist. Population risk estimates typically refer to the number of cancer cases that are projected for a defined population and for a specified exposure scenario during one year. Such an estimate is based on one of two sources of data: epidemiological studies (as in the case of ETS), or animal studies which are extrapolated to give an estimate of individual lifetime risk to humans and then combined with some estimate of the exposed population. Assumptions often are required concerning the exposed population, the exposure scenario, and the dose-response relationship. The pollutants shown in Figure 3 were the only ones for which nationwide

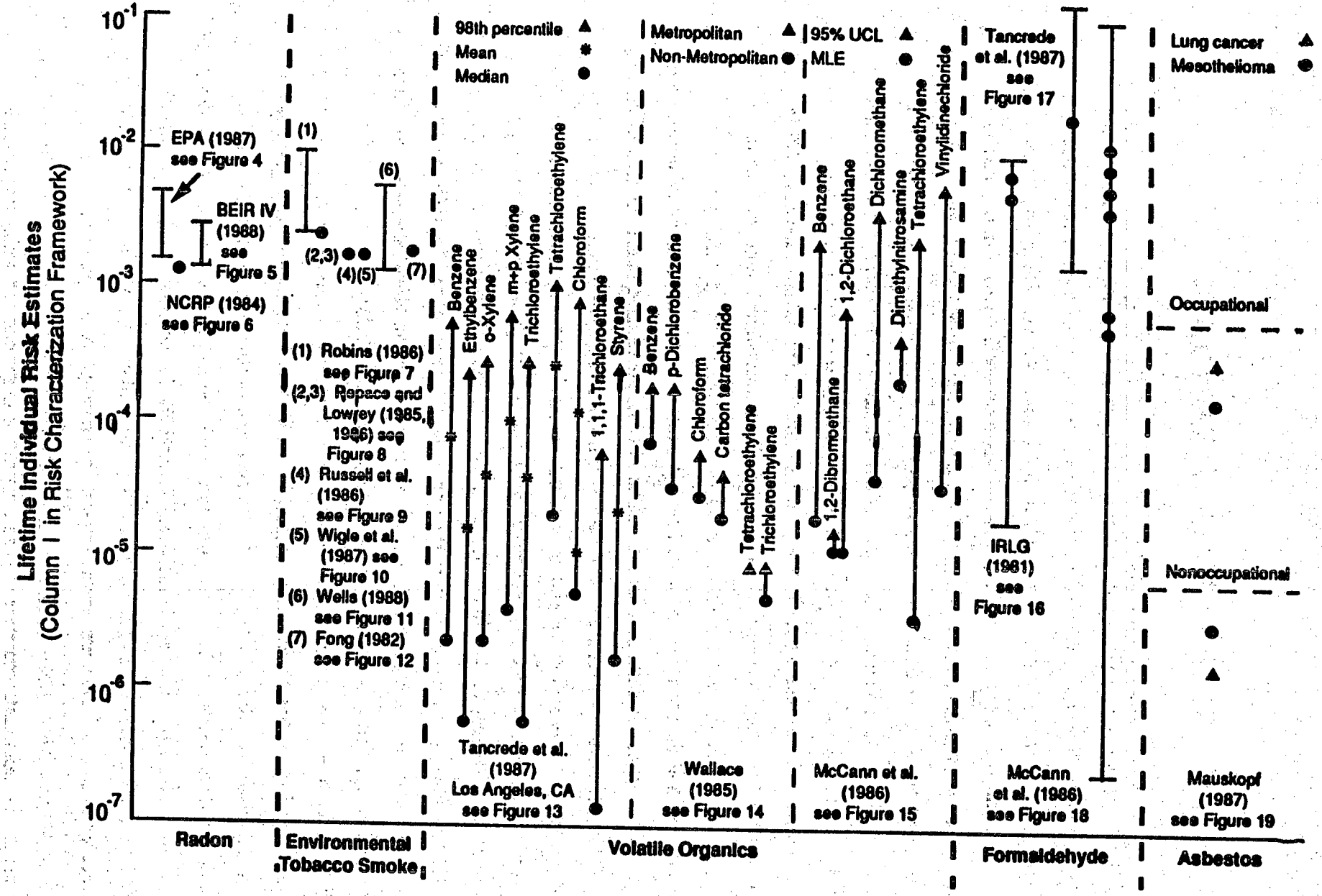

Figure 2. Comparison of Individual Lifetime Cancer Risk due to Indoor Air Pollutants

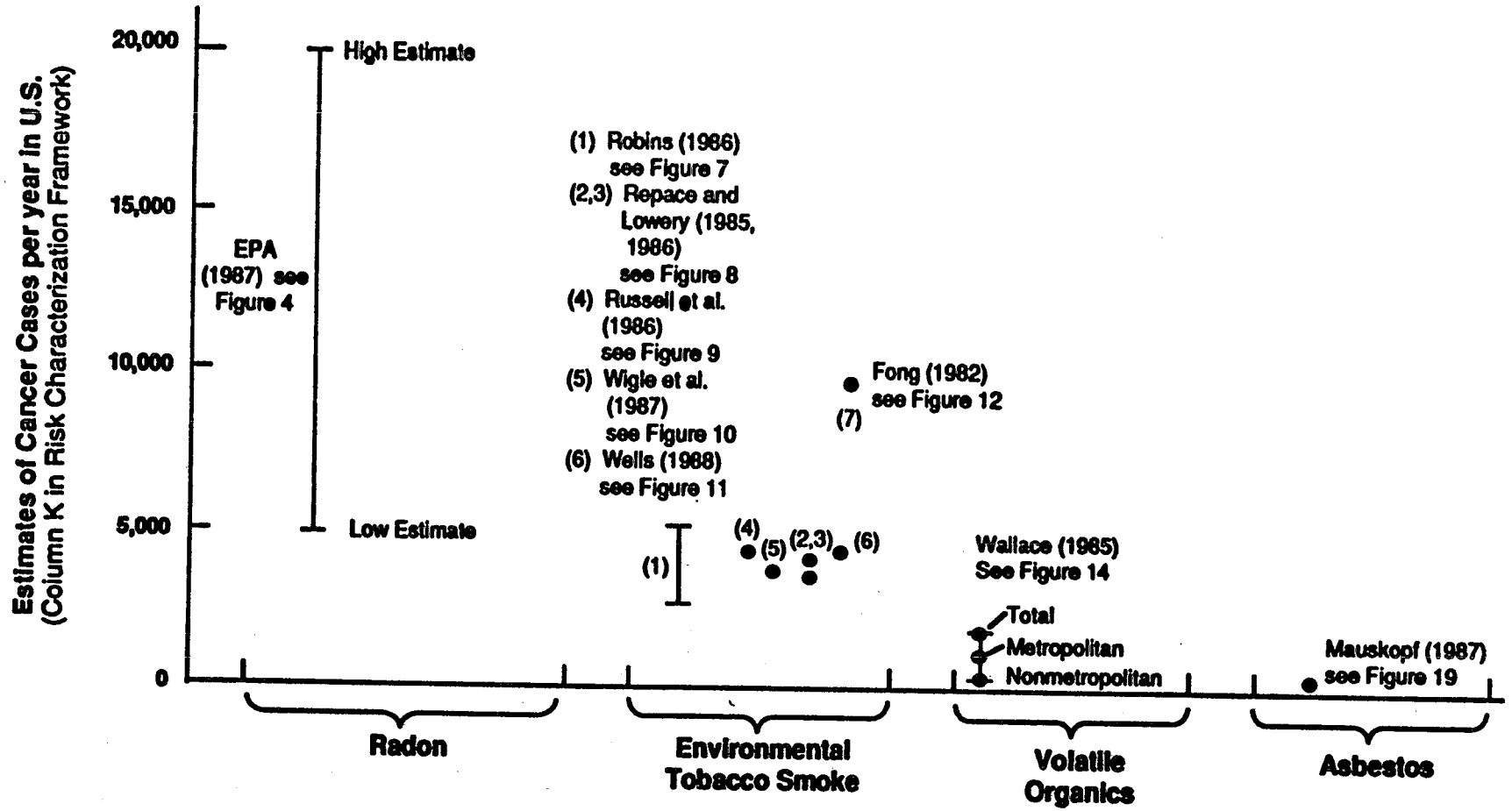

Figure 3. Comparison of Annual Cancer Cases due to Indoor Air Pollutants

RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	Earth or ground	Radon progeny		Lung duration Indoors/home Lifetime = 78.80 years 75% time spent indoors		Calculated from columns (C)x(D)		1 WLM = 170 WLhr Inhalation rate adjustment factor: 16.8/80		Calculated from columns (E)x(F)		Unit risk coefficient (cases per WLM)		Endpoint: lung cancer deaths		All U.S. population		Annual risk of lung cancer Columns (I)x(J) / 78.8
		4.0 E-3 WL		5.0 E+5 hr		2.0 E+3 WLhr		8.0 E-3 WLM/WLhr		6.0 E+0 WLM		2.9 E-4 to 1.1 E-3 WLM		1.8 E-3 to 8.9 E-3		226,545,805		5,000 to 20,000 (deaths/yr)
Qualitative Information	← Hazard Identification →																	
	Based on lung cancer mortality data for 18 groups of miners																	
Quantitative Information	← Exposure Assessment →									← Dose-Response →				← Risk Characterization →				
	EPA assumes an average indoor radon level of 0.004 WL based on a study of New York and New Jersey homes.									Response factor was calculated based on an assumed relative risk of 1% or 4% per WLM.				Estimation of population risk is the focus of EPA's analysis. Lifetime individual risk was back calculated.				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: USEPA, (1987), Radon Reference Manual, Office of Radiation Programs, Washington, DC, EPA 520/1-87-20.
2. Additional comments: See pages 2 and 8. Also, Appendix B for radon unit definitions and conversion factors. The notation, E-3, E-5, E-4, etc. is scientific notation used throughout to mean x 10^{-3}, x 10^{-5}, x 10^{-4}, etc. For example: 4.0 E-3 equals 4.0 x 10^{-3}.

Figure 4. Radon: U.S. Environmental Protection Agency (1987c)
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Key Assumptions and Comments on Data

Column	Data	Comments
A	Equation used in EPA Radon Reference Manual (see further explanation in following text)	$TLCD = CR \times T \times F_{WLM} \times R_{RRM} \times TCR \times POP$ TLCD = total U.S. lung cancer deaths from indoor radon (see Column K below) C_R = mean lifetime indoor radon decay product concentration (Col. C below) T = average lifetime exposure duration (hours, see Column D below) F_{WLM} = conversion for WLhours to WLM, adjusting for difference in breathing rate between miners and average adult (see Column F below) R_{RRM} = relative lung cancer mortality risk for lifetime exposure to radon ranging from 1% to 4% per WLM (see Column H below) TCR = underlying annual average U.S. lifetime lung cancer mortality rate corrected for 10 year latency period (see Column H below) POP = 1980 U.S. population
B	Source Factors	Naturally occurring radon and daughter products (progeny). Building effects on risk calculation not considered.
C	Pollutant Concentration	Based on a study of the distribution of ambient radon and radon progeny in residential buildings in the New York and New Jersey area, EPA assumed an average indoor radon concentration for the entire U.S. of 0.004 WL. This is roughly equivalent to 0.8 pCi/L of radon in air. See Appendix B for unit definitions.
D	Exposure Duration/Setting	Assumes: average lifetime of 73.88 years with 75% of time spent indoors. These have been converted to hours using the following equation: $73.88 \times 365 \times 24 \times 0.75 = 5 \times 10^5$
F	Dosimetry Factors	A dosimetry factor of 0.003 WLM/WLh was used which assumes 1 WLM = 170 WLh and adjusts for breathing rate of miners (breathing rate of miners = 30L/min; breathing rate of average adult = 15.3L/min). 1 WLM/170 WL-hr. x 15.3/30 = 0.003.
G	Dose	The average lifetime dose calculated from Columns E and F is 6 WLM.

Figure 4. Radon: U.S. Environmental Protection Agency (1987c)
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Key Assumptions and Comments on Data

Column	Data	Comments
H	Response Factor	Risk coefficients expressed as cases/WLM of exposure were estimated to range from 2.9 x 10^{-4} cases/WLM to 1.1 x 10-3/WLM. These are computed from the annual average of U.S. lifetime lung cancer risk per person (1980 vital statistics) adjusted for 10 year latency period, multiplied by relative risk of lung cancer mortality from a lifetime exposure to radon, per WLM, of 1% to 4%: (4.584 x 10^{-4}/yr) * (73.88yr - 10y) * (0.01/WLM) or (0.04/WLM).
I	Lifetime Individual Risk	Range of 1.8 x 10^{-3} to 6.9 x 10^{-3} of individual excess relative lifetime risks were calculated.
J	Exposed Population	U.S. population in 1980 = 226,545,805 (as used in this reference).
K	Risk to Exposed Population	Individual risk (I) multiplied by population (J) and converted to cases/yr by dividing by average lifetime of 73.88 yrs.

Figure 4. Radon: U.S. Environmental Protection Agency (1987c)
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RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	Earth or ground	Calculated Radon progeny		Calculated Long duration Indoors, home		Calculated from columns (C)x(D)				Calculated from columns (E)x(F)		Unit risk coefficient (cases per WLM)		Endpoint: lung cancer deaths				
Males:		2.0 E-3 WL		6.1 E+5 hrs		1.2 E+3 WLhr		1 WLM / 170 WLhr		7.0 E+0 WLM		5.1 E-4/WLM		3.50 E-3				
Females:		2.0 E-3 WL		6.7 E+5 hrs		1.3 E+3 WLhr		1 WLM / 170 WLhr		7.6 E+0 WLM		1.9 E-4/WLM		1.40 E-3				
Qualitative Information	← Hazard Identification →																	
	Data from four studies of underground miners exposed to radon																	
Quantitative Information	← Exposure Assessment →									← Dose-Response →				← Risk Characterization →				
	The pollutant concentration is calculated from an assumed exposure of 0.1 WLM/yr used in the example calculation on page 76 of the BEIR IV report.									Unit risk coefficients are taken from example on page 76 of the BEIR IV report.				Lifetime risk of lung cancer mortality due to a lifetime exposure to radon progeny are calculated in the example on page 76 of the BEIR IV report.				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: BEIR IV, (1988), Health Risks of Radon and Other Internally Deposited Alpha Emitters, National Research Council, Commission on Life Sciences, Committee on the Biological Effects of Ionizing Radiation; National Academy Press, Washington, DC.
2. Additional comments: See pages 2 and 3.

Figure 5. Radon: National Research Council (1988)
Page 1 of 3

Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation Used in Bier IV	Age-specific relative-risk time-since-exposure (TSE) model: $r(a) = r_o(a)[1 + \beta\gamma(a)(W_1 + 0.5W_2)]$ $r(a)$ = age-specific lung-cancer mortality rate (from all causes) $r_o(a)$ = age-specific background lung cancer mortality rate from all causes other than radon β = the basic slope of the dose-response relation (0.025) $\gamma(a)$ = effect of age at risk (1.2 for age <55, 1.0 for age 55-64, and 0.4 for age ≥65 years) W_1 = WLM of exposure incurred between 5 and 15 years before current age W_2 = WLM of exposure incurred more than 15 yrs before current age The estimated excess lifetime risk of lung cancer due to exposure to radon progeny is estimated by: $R_e - R_o$ where: R_o is the lifetime probability of lung cancer mortality in a nonexposed person R_e is the lifetime probability of lung cancer mortality in an exposed person. Estimates of R_e based on the TSE model given above will vary with calendar time. The BEIR IV report includes tables which show the ratio of lifetime risk (R_e/R_o), lung cancer risk (R_e), and years of life lost for males and for females by exposure rate in WLM per year, based on the above model when exposure is sustained throughout life. The data shown in the columns of Figure 5 are taken from an example on page 76 of the BEIR IV report and Table 2-13 on that same page.
C	Pollutant Concentration	The assumed exposure of 0.1 WLM/yr used in the example on page 76 of the report was converted to an average concentration by using the following equation: .1 WLM/yr x 170 WLh/1 WLM x 1 day/24 hr x 1 yr/365 days = 0.002 WL The 0.002 WL of radon progeny is roughly equivalent to a concentration of 0.4 pCi/L of gaseous radon in air. See Appendix B for unit conversion factors.
D	Exposure Duration/Setting	Average lifespan for males 69.7 years, females 76.4 years, converted to hours. No adjustment made for percent of time spent indoors.

Figure 5. Radon: National Research Council (1988)
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Key Assumptions and Comments on Data

Column	Data	Comments
F	Dosimetry Factors	WLhr converted to WLM using the following dosimetry factor: 1 WLM = 170 WLhr. No adjustment made for difference in breathing rate between miners and average person.
H	Response Factor	The unit risk coefficients for males and females shown in Column H were calculated in the following manner. Using the time since exposure model, the lifetime excess risks for males and females were calculated separately assuming a lifetime exposure of 0.1 WLM/yr. The total annual risk for a person's age is calculated from this equation using an appropriate background age specific risk (R_o). This risk estimate is multiplied by the chance of surviving all causes of death to that age and these products are summed over the ages in the desired periods. From these calculations the excess lifetime risk of lung cancer (R_e) can be calculated. The lifetime risk (R_e) for males exposed at 0.1 WLM/yrs is 0.07087; the lifetime risk for unexposed males (R_o) is 0.06734; and the excess risk attributed to radon exposure, $R_e - R_o$ is 0.07087 - 0.06734 = 0.00353. Similarly, for females at 0.1 WLM/yr, $R_e - R_o$ is 0.02663 - 0.02521 = 0.00142. These lifetime excess risks were then divided by the lifetime cumulative exposure calculated separately for males and females. Males were assumed to have a lifespan of 69.7 years which would give a mean cumulative lifetime exposure to male population of 6.97 WLM. Similarly, females were assumed to have an average lifespan of 76.4 years and a cumulative average lifetime exposure of 7.64 WLM. Dividing the lifetime excess risk calculated for males by 6.97 gives 5.1×10^{-4} cases per WLM of lifetime exposure as shown in Column H. Similarly, dividing the lifetime excess risk to females by 7.64 WLM gives the lifetime lung cancer mortality for females per WLM of 1.9×10^{-4}. (These calculations are shown on page 76 of the BEIR IV report.)
I	Lifetime Individual Risk	Lifetime Individual Risks were calculated separately for males and females. Columns G x H = Column I.
K	Risk to Exposed Population	The BEIR IV Report does not characterize risk to the exposed population.

Figure 5. Radon: National Research Council (1988)
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RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	Earth or ground	Radon progeny		Long duration Indoors, home		Calculated from columns (C)x(D)						Unit risk coefficient (cases/WLM)		Calculated from author's data Endpoint: lung cancer deaths		All U.S. population		
		4.0 E-3 WL		6.1 E+5 hrs		2.4 E+3 WLhr		1 WLM/170 WLhr		1.4 E+1 WLM		1.3 E-4/WLM		1.8 E-3				
Qualitative Information	Hazard Identification																	
Quantitative Information	Exposure Assessment: The average exposure to radon decay products in the U.S. is estimated as 0.2 WLM/yr. This converts to 0.004 WL as a concentration.									Dose-Response: A unit risk factor was derived from data presented in Table 10-2 of the NCRP report, assuming lifetime exposure.				Risk Characterization: Lifetime individual risk was calculated by multiplying columns (G) and (H).				
Qualitative or Quantitative Analysis	Risk Assessment																	

Footnotes: 1. Reference: NCRP, Evaluation of Occupational and Environmental Exposures to Radon and Radon Daughters in the United States, National Council on Radiation Protection and Measurement, Report 78, Washington, DC.
2. Additional comments: See pages 2 and 3.

Figure 6. Radon: National Council on Radiation Protection and Measurements (1984)
Page 1 of 3

Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation Used in NCRP (1984) (see further explanation in following text)	The risk equation: $A(t\|t_0) = RC\,(P_t/P_{t_0})\,e^{-\lambda(t-t_0)}$ where: $A(t\|t_0)$ = attributable annual tumor rate at age t ($t \geq 40$) due to a single annual exposure at t_0. If exposure occurs after age 35, risk commences at t_0 + 5 years, if exposure occurs before age 35, risk commences at age 40, RC = risk coefficient 10×10^{-6} cases/yr per WLM is adopted in the NCRP 1984 report, this is the average risk coefficient derived from epidemiology studies of underground miners. P_t/P_{t_0} = lifetable correction to account for death from other causes, P_{t_0} = probability that an individual will be alive at age t_0, P_t = probability that an individual will be alive at age t, and λ = decrease in rate of risk expression due to repair, cell death or unspecified mechanisms. ($\lambda = \ln 2/20 \times yr^{-1}$) The lifetime risk, LR (t_0), from the single annual exposure at t_0 is calculated by summing the annual attributable rate over the ages of tumor appearance. $LR(t_0) = \sum_{t}^{85} A(t\|t_0)$ The lifetime risk for multiple annual exposures, LR is obtained by summing the lifetime risk from each single exposure, $\Sigma LR(t_0)$.
C	Pollutant Concentration	NCRP report presents a review of the literature on exposure concentrations to radon and radon decay products. The average exposure in the U.S. expressed as WLM/year is stated as 0.2. This was converted to WL using the following equation: 0.2 WLM/yr x 170 WLhr/1 WLM x 1 day/24 hr x 1 yr/365 days = 0.004 WL.

Figure 6. Radon: National Council on Radiation Protection and Measurements (1984)
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Key Assumptions and Comments on Data

Column	Data	Comments
D	Exposure Duration/ Setting	Assumed average lifespan of 70 years for the framework, with exposure over entire lifespan and no adjustment for time spent indoors. The authors do not quantify exposure duration. However, data presented in Table 10-2 of the NCRP report state that the estimates are developed for a population with age characteristics equal to that in the whole United States in 1975.
F	Dosimetry Factors	No adjustment for difference in breathing rate between miners and average person.
G	Response Factor	Using the equation described under comments for Column A, risk estimates were developed for various ages based on duration of exposure (expressed in terms of cases per year per WLM of exposure). For a lifetime exposure beginning at age 1, the lifetime lung cancer risk under environmental conditions was calculated to be 9.1×10^{-3} cases assuming exposure to 1 WLM each year of life. Assuming a 70 year lifetime of exposure, this would convert to a unit risk coefficient of 1.3×10^{-4} per WLM as shown in Column H.
I	Lifetime Individual Risk	Lifetime individual risk at an average exposure concentration of 0.004 WL is calculated to be 1.8×10^{-3} by multiplying Columns G and H.

Figure 6. Radon: National Council on Radiation Protection and Measurements (1984)
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RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	Tobacco smoking	Env. Tobacco smoke		Long duration Lifetime: 70 years Indoors, home Indoors, work				15 mg tar/cigarette 80% of tar retained Average body weight: 70 kg		Calculated $\frac{mg}{kg \times day}$		Calculated from author's data Carcinogenic potency $\frac{kg \times day}{mg}$		Endpoint: lung cancer mortality		Adults U.S. nonsmokers in 1985, age 45 or older (approximate calculation)		Annual lung cancer deaths $\frac{\text{Columns (I)x(J)}}{70}$
						0.1 - 0.9 cigarettes/day				2.0 E-2 to 1.6 E-1		2.0 E-1 to 6.2 E-2 Females		4.0 - 9.9 E-3 Females		23 million Females		1768 to 3220 Females
												2.0 E-1 to 6.2 E-2 Males		4.0 - 9.9 E-3 Males		13 million Males		721 to 1942 Males
																		2489 to 5162 Total
Qualitative Information	← Hazard Identification →																	
Quantitative Information	← Exposure Assessment →									← Dose-Response →				← Risk Characterization →				
	Estimates of the range of exposure expressed as carcinogen-equivalent number of actively smoked cigarettes inhaled daily are from Table D-3 of referenced article. This corresponds to the dose received by an average adult nonsmoker with a nonsmoking spouse.									Dose was calculated based on assumptions in Column (F). Response factors calculated from columns (G) and (I).				Columns (I) and (K) are taken from referenced article. Affected population was calculated using upper ranges from columns (I) and (K).				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: Robins, J., (1986), "Risk Assessment - Exposure to Environmental Tobacco Smoke and Lung Cancer." In National Research Council, (1986), Environmental Tobacco Smoke: Measuring Exposure and Assessing Health Effects. National Academy Press. Washington, DC.

Figure 7. ETS: Robins (1986)
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Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation Used by Robins (see further explanation in following text)	The equation used by Robins to calculate the total number of lung cancer deaths (AN) among nonsmoking women attributable to ETS in 1985 is: $AN = \sum_{t} AF(t) \times I_0(t) \times N(t)$ where AF(t) = the age-specific fraction of lung cancer deaths due to ETS exposure in nonsmoking women $I_0(t)$ = the age-specific lung cancer death rate among nonsmoking women in 1985 N(t) = the number of nonsmoking women at risk at age t in 1985 Data on these variables are given in Garfinkel (1981) and from the National Health Interview Survey Data available from the National Center for Health Statistics. In order to estimate the age-specific relative risk among nonsmoking women, Robins was required to use the age-specific estimates of probability of being married to a smoker and of the true relative risk in "exposed" and "unexposed" subjects. Robins obtained age-specific estimates of the probability of being exposed from the Garfinkel et al. (1985) control population. As seen in the equation above, risk to the exposed population is calculated without using lifetime individual risk estimates (Column I). A similar methodology was employed for calculating annual lung cancer deaths for males in 1985.
C	Pollutant Concentration	Robins' analysis does not address pollutant concentrations. The analysis begins with exposure estimates calculated from epidemiological studies or biomarkers. Since ETS is a complex mixture, back calculations from these exposure estimates to pollutant concentrations could not be done.
D	Exposure Duration/Setting	Thirty hypothetical exposure histories were developed to describe age-dependent ETS exposure levels under different activity assumptions. These histories cover a 70 year lifetime and various levels of exposure.
E	Exposure	The estimates in Column E are ranges of cigarette equivalents per day for the 30 exposure histories. These are the number of cigarettes that would have to be actively smoked to deliver to the lung a dose of active carcinogen equal to that attributable to the ETS exposure of an average adult nonsmoker with a nonsmoking spouse. The estimates are dependent on the relative risk ratio (1.3) and the estimates of coefficients of the dose-response model.
F	Dosimetry Factors	To convert exposure to dose, we assume 15 mg of tar per cigarette; 80% retention of the tar when inhaled and a 70 kg average adult body weight. This converts exposure expressed in cigarettes/day to mg/kg-day of tar inhaled.

Figure 7. ETS: Robins (1986)
Page 2 of 3

Key Assumptions and Comments on Data

Column	Data	Comments
G	Dose	Two estimates of dose were calculated based on the low and high estimates of exposure shown in Column E.
H	Response Factor	Robins assumes that cigarette smoke and ETS influence the rates of the first and fourth stages of a five-stage cancer process. The ratio of these two parameters is estimated by fitting data from studies of lung cancer in active smokers to the five-stage model. The estimates for the response factors shown in Column H were calculated by dividing Column I by Column G. These estimates are very close to the low and high estimates for B_4 (the coefficient on the fourth stage of the model) presented by the author.
I	Lifetime Individual Risk	Robins estimates lifetime individual risk as the fraction of all deaths subsequent to age 45 attributable to ETS-exposure-induced lung cancer. The maximum and minimum of these estimates across all exposure histories and dose-response coefficients are shown in Column I. These estimates assume a true relative risk ratio of 1.3.
J	Exposed Population	Robins estimates the age-specific fraction of total lung cancer deaths due to ETS exposure in nonsmoking women based on data from the Garfinkel et al. (1985) control population. These data are then combined with data on the number of nonsmoking women at risk at age t in 1985 and the age-specific lung cancer death rate among nonsmoking women in 1985. These data are given in Garfinkel (1981) and from the National Health Interview Survey available from the National Center for Health Statistics. The same data sources were used to estimate the number of lung cancers attributable to ETS in 1985 in nonsmoking males. Although it is difficult to calculate the exposed population from a lifetable model, a rough estimate of the exposed population can be calculated by dividing Column K by Column I and multiplying by 70 years. These estimates for females and males are shown in Column J using the maximum estimates of risk.
K	Risk to Exposed Population	Using the equation described in comment A above, Robins calculates the annual lung cancer death attributable to ETS exposure. The estimates shown in Column K are those calculated from epidemiological data, assuming a relative risk ratio of 1.3.

Figure 7. ETS: Robins (1986)
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RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	Tobacco smoking	Env. tobacco smoke Not presented		Long duration Indoors, work Indoors, home Adult lifetime assumed to be 50 years		Typical nonsmoker with 1.4 mg/day of tobacco tar exposure (0-14 mg/day estimated range)		Absorption rate: 100% Average body weight: 70 kg		Calculated from authors' data $\frac{mg}{kg \times day}$ 2.0 E-2		Calculated from authors' data $\frac{kg \times day}{mg}$ 1.8 E-1		Calculated from authors' data Incidence: lung cancer 3.6 E-3		Adults Male Female Nonsmoker 62.4 million U.S. nonsmokers over 35 years old at risk from ETS		Lung cancer deaths among U.S. nonsmokers attributable to ETS exposure $\frac{Columns\ (I) \times (J)}{50}$ 4700 per year (4891 per year)
Qualitative Information	← Hazard Identification →																	
Quantitative Information	← Exposure Assessment →					Estimated exposure to the particulate phase of ambient tobacco smoke determined from average concentrations of ETS for model workplace and home environments.				← Dose-Response →				← Risk Characterization →				
										Response factor calculated from columns (G) and (I). Column (G) was calculated by dividing column (E) by 70 kg.				The authors focus on estimating annual lung cancer deaths (column K) from epidemiologic data. Column (I) was back calculated. Estimates in parentheses in Column (K) are from Repace and Lowrey (1986).				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: Repace, J. L. and A. H. Lowrey (1985), "A Quantitative Estimate of Nonsmokers' Lung Cancer Risk from Passive Smoking," Environment International, 11:3-22.
2. Additional comments: See pages 2 and 3.

Figure 8. ETS: Repace and Lowrey (1985)
Page 1 of 3

Key Assumptions and Comments on Data

Column	Data	Comments
C	Pollutant Concentration	An equilibrium model is used to estimate pollutant concentration in various microenvironments. The parameters of the model include building ventilation rate, the occupancy of the building and number of smokers. Pollutant concentrations are presented for various occupancy-ventilation scenarios, however, these estimates are not used directly to calculate exposure.
D	Exposure Duration/ Setting	The estimated exposures to the particulate phase of ambient tobacco smoke for U.S. adults of working age, at work and at home were determined from average concentrations of tobacco smoke calculated for model workplace and home microenvironments, weighted for average occupancy. The authors assume a probability of being exposed at work of 63%; probability of not being exposed at work, 37%; probability of being exposed at home, 62%; probability of not being exposed at home, 38%. Probabilities of exposure at work and home were assumed to be independent. The authors assume that 50 years is the length of adult exposure. Childhood exposure is not considered in this analysis.
E	Exposure	Table 1 of the referenced article presents estimates of modeled daily average and daily probability weighted average exposures for workplace and home microenvironments. The average workplace exposure was estimated to be 1.82 mg/day. This assumes one third of the occupants at work are smokers, that they smoke average tar cigarettes at a rate of 32 per 16-h day, that individuals breathe at a rate of 1 m^3/hr, that 38% of the workforce was female (5.2 work hours per day) and 62% was male (6.7 work hours per day). Similar assumption for ventilation rate and number of cigarettes smoked per day are used to estimate an exposure in the home of 0.45 mg/day. This also assumes an occupancy-weighted average number of cigarettes equal to 22 cigarettes per day and that occupancy of the home by smokers and nonsmokers is coincident. Based on the assumption given for Column D, the daily probability weighted exposure was estimated at 1.43 mg/day.
F	Dosimetry Factors	Exposure is used in this analysis to mean the inhaled dose of ETS. A breathing rate of 1 m^3/hr is assumed in the calculation of exposure. We divide exposure by 70 kg to show dose in units of mg/kg-day.

Figure 8. ETS: Repace and Lowrey (1985)
Page 2 of 3

Key Assumptions and Comments on Data

Column	Data	Comments
G	Dose	Dose is calculated by dividing Column E by 70 kg.
H	Response Factor	The response factor was calculated by dividing Column I by Column G. The authors calculate an annual exposure-response relationship of 5×10^{-5} risk per 1 mg/day nominal exposure. Multiplying this annual response factor by 50 years also yields the estimate shown in Column H.
I	Lifetime Individual Risk	The author presents information for the exposed population (Column J) and the number of lung cancer deaths among nonsmokers attributable to ETS (Column K). Individual lifetime (Column I) risk was calculated by dividing Column K by Column J and multiplying by 50 years (50 years is length of adult exposure).
J	Exposed Population	Repace and Lowrey reference the U.S. Surgeon General's 1979 report as the basis for 62.4 million U.S. nonsmokers aged 35 years and older who are at risk from exposure to Environmental Tobacco Smoke.
K	Risk to Exposed Population	A calculation based on the age standardized differences in lung cancer mortality rates between Seventh Day Adventists who never smoked and demographically comparable non-Seventh Day Adventists who never smoked (age 35 to 85+). Based on studies of Phillips et al. (1980). Repace and Lowrey (1986) provide a refinement to this estimate (shown in parentheses).

Figure 8. ETS: Repace and Lowrey (1985)
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RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	Tobacco smoking	Env. tobacco smoke		Long duration Indoors, home Indoors, work								Calculated from authors' data Urinary nicotine		Calculated from authors' data Premature death		All U.S. adults Male Female Nonsmoker		Deaths/year due to ETS (includes heart disease, bronchitis, emphysema, as well as lung cancer) Columns (I)x(J) / 50
				50 years of exposure		Exposure of nonsmokers: 0.7% of active smokers		Not presented		Nonsmoker average 1.1 E+1 ng/ml urinary nicotine		1.3 E-4 ml/ng		1.4 E-3		147 E+6		>4000
Qualitative Information	← Hazard Identification →																	
Quantitative Information	← Exposure Assessment → The authors' exposure assessment is based on urinary nicotine concentrations taken from four studies of nonsmokers and four studies of smokers. The average of 188 nonsmokers was 10.8 ng/ml or 0.7% that of smokers. Exposure is estimated at 0.7% that of smokers.									← Dose-Response → Response factor is calculated by dividing column (I) by column (G). This assumes a linear dose-response function based on urinary nicotine concentrations.				← Risk Characterization → Column (I) is calculated by dividing column (K) by column (J) x 50 years. Column (J) is estimated at 65% of the U.S. population. Column (K) is calculated by the authors.				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: Russel, M. A. H., M. J. Jarvis, and R. J. West, (1986), "Use of Urinary Nicotine Concentrations to Estimate Exposure and Mortality from Passive Smoking in Nonsmokers," British Journal of Addiction (1986) 81:275-281.
2. Additional comments: See pages 2 and 3.

Figure 9. ETS: Russell et al. (1986)
Page 1 of 3

Key Assumptions and Comments on Data

Column	Data	Comments
C	Pollutant Concentration	The authors do not address pollutant concentration in their analysis.
D	Exposure Duration/ Setting	Exposure is assumed to occur indoors at home and at work. Since the analysis is based on an analogy with smokers, the duration of exposure is most appropriately matched with the length of time individuals smoke. The authors state that individuals usually start smoking in their late teens. Thus, a 50 year exposure duration is assumed to be consistent with the analysis presented by Repace and Lowrey (1985). The authors recognize that this exposure duration may be an underestimate since children can be exposed to ETS from birth.
E	Exposure	Since the authors' analysis is based on a biomarker, we consider urinary nicotine concentrations to be indicators of dose. Relative exposure of nonsmokers, however, is calculated to be 0.7% that of active smokers (10.8 ng/ml in nonsmokers divided by 1491 in smokers).
G	Dose	The authors used measures of urinary nicotine concentrations of smokers and nonsmokers from four studies, respectively, as an indicator of exposure to ETS. The average urinary nicotine concentration in 188 nonsmokers was 10.8 ng/ml, which amounts to 0.7% of the average urinary nicotine concentration in 229 smokers (1471 ng/ml). The authors point out that because nicotine has a tendency to evaporate from particles of aging smoke, the measurement of urinary nicotine in nonsmokers may underestimate the intake of tar from ETS. The authors suggest that tar intake from ETS relative to tar intake from active smoking may actually be higher than the estimate based on urinary nicotine measurements.

Figure 9. ETS: Russell et al. (1986)
Page 2 of 3

Key Assumptions and Comments on Data

Column	Data	Comments
I	Lifetime Individual Risk	Calculated from authors' data. Deaths/year from Column K divided by exposed population in Column J multiplied by a 50 year lifetime of exposure.
J	Exposed Population	Russel et al. state the exposed population of nonsmokers to be 65% of the U.S. population: 0.65 x 226,000,000 = 147,000,000.
K	Risk to Exposed Population	Russel et al. calculate this value based on their observation that the 65% of the U.S. population that are nonsmokers are exposed to the carcinogens in tobacco smoke at a rate 0.7% that of active smokers. Deaths attributed to active smoking in the U.S. are reported to be 320,000 deaths per year among the 35% of the population who smoke. Deaths due to ETS are calculated to be (320,000 deaths/year) x (65/35) x 0.007 = 4160 deaths/year. This estimate includes deaths due to coronary heart disease, bronchitis, and emphysema as well as lung cancer.

Figure 9. ETS: Russell et al. (1986)
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RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	Tobacco smoking	Env. tobacco smoke		Long duration Indoors, home Indoors, work Average adult lifetime: 50 yrs				Absorption rate: 100% Average body weight: 70 kg		Calculated from authors' data mg / kg x day		Calculated from authors' data kg x day / mg		Calculated from authors' data Endpoint: lung cancer deaths		Adult U.S. nonsmokers (from Repace and Lowrey, 1985)		Annual lung cancer deaths attributable to ETS Columns (I)x(J) / 50
						1.4 mg/day				2.0 E-2		1.3 E-1		2.6 E-3		62.4 E+6		3300
Qualitative Information	← Hazard Identification →																	
Quantitative Information	← Exposure Assessment →					The authors cite the National Research Council (1986) estimate of daily exposure to tobacco smoke particulate matter of 1.4 mg/day.				← Dose-Response →				← Risk Characterization →				
										Calculated response factor by dividing column (I) by column (G). Column (G) was calculated by dividing column (E) by 70 kg.				Column (J) is taken from Repace and Lowrey (1985). Column (I) is calculated by dividing column (K) by column (J) and multiplying by 50 years.				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: Wigle, D. T., N. E. Collishaw, J. Kirkbride, & Y. Mao, (1987). "Deaths in Canada from Lung Cancer due to Involuntary Smoking." Canadian Medical Association Journal, 1987; 136:945-951.

Figure 10. ETS: Wigle et al. (1987)
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Key Assumptions and Comments on Data

Column	Data	Comments
C	Pollutant Concentration	No information is presented on pollutant concentration.
D	Exposure Duration/ Setting	The author reports that exposure of nonsmokers to ETS outside the home may approach or exceed those at home. Since the authors base their analysis on that of Repace and Lowrey (1985), we show the same exposure duration assumption.
E	Exposure	The authors cite the National Research Council's (1986) estimate of daily exposure to tobacco smoke particulate matter of 1.4 mg/day which is roughly equivalent to 1 to 2 cigarettes per day. Wigle notes that it may be misleading to measure tobacco smoke contaminants in nonsmokers and then express the results as cigarette equivalents because sidestream smoke is more carcinogenic than mainstream smoke. Since sidestream smoke is the main source of ETS, the cancer risk may be greater than the predicted equivalent of 1 to 2 cigarettes per day.
F	Dosimetry Factors	We assume an average body weight of 70 kg and that the pollutants are absorbed 100%.
G	Dose	Dose was calculated by dividing Column E by 70 kg. Dose is expressed in units of mg/kg-day.
H	Response Factor	The response factor was calculated by dividing Column I by Column G. Since both of these columns refer to lifetime exposures the response factor also is associated with lifetime exposure. The units are the inverse of exposure, kg-day/mg.

Figure 10. ETS: Wigle et al. (1987)
Page 2 of 3

Key Assumptions and Comments on Data

Column	Data	Comments
I	Lifetime Individual Risk	This estimate was calculated by dividing Column K by Column J and multiplying by 50 years.
J	Exposed Population	The authors do not give exposed population. Since the authors reference the Repace and Lowrey methodology we have used their estimates of the U. S. population of nonsmokers.
K	Risk to Exposed Population	Wigle et al. conclude that in Canada, ETS exposure in all environments may cause 330 deaths due to lung cancer. The total lung cancer deaths of 330 are considered an upper limit by the author since it is based on the difference in lung cancer risk found between nonsmoking Seventh Day Adventists and nonsmoking non-Seventh Day Adventists as reported by Repace and Lowrey. When the risk is extrapolated to the U.S. population which is ten times the population of Canada, 3300 lung cancer deaths are calculated.

Figure 10. ETS: Wigle et al. (1987)
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RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	Tobacco smoking	Env. tobacco smoke		Long duration Indoors, home 50 years of exposure				Absorption rate: 100% Average body weight: 70 kg		Calculated from author's data		Calculated from author's data		Endpoint: lung cancer death		Adults Male Female Nonsmoker		Annual lung cancer deaths among U.S. nonsmokers attributable to ETS exposure
						mg/day				$\frac{mg}{kg \times day}$		$\frac{kg \times day}{mg}$						$\frac{\text{Columns (I)x(J)}}{50}$
						0.64				8.6 E-3		7.4 E-1 Males 2.3 E-1 Females 4.3 E-1 Total		6.4 E-3 Males 2.0 E-3 Females 3.7 E-3 Total		19.4 million Males 30.6 million Females 50 million Total		2499 Males 1232 Females 3731 Total
Qualitative Information	← Hazard Identification →																	
Quantitative Information	← Exposure Assessment →									← Dose-Response →				← Risk Characterization →				
	Author's risk estimates are not based on exposure data. Smoke retention by passive smoker was cited at 0.64 mg/day (tar) (from U.S.S.G., 1986). This is converted to an averaged dose per kg of body weight.									Column (H) was calculated by dividing column (I) by column (G).				Risk to affected population was calculated separately for males and females using a constant relative risk of 2.1 and 1.44 respectively.				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: Wells, A. J. (1988), "An Estimate of Adult Mortality in the United States from Passive Smoking," Environment International, 14:249-265.
2. Additional comments: See pages 2 and 3.

Figure 11. ETS: Wells (1986)
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Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation Used by Wells	The excess death rate for never-smokers exposed to ETS (D_{px}) was calculated using the following equation: $D_{px} = D_{ns}(R-1)/(F_p(R-1)+1)$ D_{ns} = never-smoker death rate from all causes R = relative risk from exposure to ETS (computed from epidemiological studies) F_p = fraction of population exposed
C	Pollutant Concentration	No information was provided regarding pollutant concentrations.
D	Exposure Duration/ Setting	Most of the epidemiology studies referenced by the author focus on exposures to ETS in the home. Exposure to ETS in the work place is not a major consideration. Epidemiology data are primarily for adults exposed to ETS through spouses' smoking habits. Therefore, a 50 year exposure period is considered appropriate here.
E	Exposure	Although the author does not use exposure estimates in the risk calculations, he references a U.S. Surgeon General exposure estimate of 0.64 mg of tar per day for the passive smoker. This is derived by assuming that smoke retention by the passive smoker is 1/400 that retained by a direct smoker in a 16 h-day. Direct smokers are assumed to retain 240 mg of tar per 16 h-day.
F	Dosimetry Factors	This paper contains a discussion of the regional deposition in the respiratory tract of particulate matter associated with active and passive smoking. Particle size and point of entry are considered. The smaller particles associated with passive smoking are retained in the deep alveolar regions of the lungs at a higher rate than the larger particles associated with active smoking. The nasal point of entry for passive smoke may also account for the observed differences in cancer sites (other than the lung) between active and passive smokers.

Figure 11. ETS: Wells (1986).
Page 2 of 3

Key Assumptions and Comments on Data

Column	Data	Comments
H	Response Factor	The response factors shown in Column H were calculated by dividing Column I by Column G. The author does not present response factors.
I	Lifetime Individual Risk	The author presents information on exposed population (Column J) and total deaths per year attributable to ETS (Column K). Column I was calculated from this information by dividing Column K by Column J and multiplying by 50 years.
J	Exposed Population	The authors considered the exposed population to include 1) nonsmokers living with smokers (24% of nonsmoking U.S. males and 60% of nonsmoking U.S. females), 2) nonsmokers living with other nonsmokers but exposed to ETS at work or other locations (37% of nonsmoking U.S. males, 16% of nonsmoking U.S. females). Total nonsmokers exposed to ETS is 61% of U.S. nonsmoking males and 76% of U.S. nonsmoking females. The total exposed population was calculated for each sex and age interval. The sum for each sex and the total exposed population is shown in Column J.
K	Risk to Exposed Population	The risk assessments presented by Wells are based on a series of epidemiological studies on adult nonsmokers with smoking spouses with a small component of occupational exposure. The combined relative risk for lung cancer in males as a result of exposure to ETS is 2.1 with a 95% confidence limit of 1.3 to 3.2 and is based on nine studies with 144 total cases. The relative risk for lung cancer in females as a result of exposure to ETS is 1.4 with a 95% confidence limit of 1.3 to 1.7 and is based on 17 studies with 1,174 cases. Population risks were calculated based on separate estimates of relative risk for males and females, and estimates of the exposed population for eleven separate age categories.

Figure 11. ETS: Wells (1986)
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RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	Tobacco smoking	Env. tobacco smoke		Long duration Indoors, work Other: commuting				15 mg tar/cigarette 80% of tar retained Average body weight: 70 kg		$\frac{mg}{kg \times day}$		$\frac{kg \times day}{mg}$		Calculated from author's data Endpoint: lung cancer death				Annual lung cancer and emphysema deaths among U.S. nonsmokers attributable to ETS exposure $\frac{Columns\ (I) \times (J)}{50}$
				11 hours workplace 1 hour commuting 12 hours not exposed		Equivalent of 1.6 cigarettes per day (1/13 of the exposure of an active smoker)				2.7 E-1		1) 4.0 E-2 2) 8.0 E-3		1) 1.1 E-2 2) 2.2 E-3		220 million		1) Max. estimate: 50,000 2) Realistic estimate: 10,000
Qualitative Information	← Hazard Identification →																	
Quantitative Information		← Exposure Assessment →								← Dose-Response →				← Risk Characterization →				
		Exposure is assumed to occur at a low level for 11 hours at work and at a higher level for one hour in transit. No exposure at home for 12 hours. Exposures of nonsmokers are calculated at 1/13 of the exposure of active smokers. Active smokers are assumed to smoke 20 cigarettes per day.								Dose is calculated assuming each cigarette contains 15 mg of tar, 80% of tar is retained and average individual weighs 70 kg. Two response factors are calculated (See comments)				Population risk is calculated as a fraction of U.S. deaths attributed to active smoking (320,000 per year).				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: Fong, P., (1982) "The Hazard of Cigarette Smoke to Nonsmokers," Journal of Biological Physics, 1982, 10:65-73.
2. Additional comments: See page 2 and 3.

Figure 12. ETS: Fong (1982)
Page 1 of 3

Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation Used by Fong	The author uses the following equation to estimate risk: $R = r_1 \times r_2 \times r_3 \times r_4$ where: R = the ratio of exposure of a nonsmoker from ambient smoke to that of a smoker from primary smoke r_1 = the ratio of the densities of the secondary smoke to primary smoke in the lungs r_2 = the ratio of the times of exposure of the two cases r_3 = the ratio of toxicities of the two kinds of smoke r_4 = the ratio of the amount of ambient smoke Two approaches are employed to calculate health effects associated with exposure to ETS. First, the assumption is made that health effects are in direct proportion to exposure and the known effects on smokers are scaled according to the ratio R. Second, a nonlinear effect is assumed and a reducing factor n ($n<1$) is used such that the health hazard to nonsmokers would be nR times that of smokers.
C	Pollutant Concentration	No pollutant concentration data are presented in this analysis.
D	Exposure Duration/ Setting	Exposure of a nonsmoker to ETS is estimate based on 11 hours/day of low density smoke exposure in ventilated space (workplace) and 1 hour/day of high density smoke exposure in unventilated space (e.g., bus).
E	Exposure	The author calculates the ratio of exposure of an active smoker to that of a non-smoker as 1/13. This assumes 37% of the population smokes at a rate of 1.6 cigarettes/hour. Values for r_1, r_2, r_3 and r_4 in the above equation are calculated. Total daily exposure of a nonsmoker is estimated to be equivalent to smoking 1.56 cigarettes per day, or 1/13 the rate of exposure of an active smoker.

Figure 12. ETS: Fong (1982)
Page 2 of 3

Key Assumptions and Comments on Data

Column	Data	Comments
F	Dosimetry Factors	In order to calculate dose as mg/kg-day we assume that an average cigarette contains 15 mg of tar, that 80% of the inhaled smoke is retained and that the average individual weighs 70 kg.
G	Dose	Dose was calculated as: 1.56 cigarettes/day x 15 mg/cigarette x 0.8 x (1/70 kg).
H	Response Factor	Two response factors were calculated. The first assumes that health effects are in direct proportion to exposure. This was calculated by dividing Column I by Column G. The second assumes a nonlinear relationship. The author calculates a reducing factor of 0.2 to account for this nonlinearity. Thus, the second response factor is 0.2 times the first (0.2 x 4.0 E-2 = 8.0 E-3).
I	Lifetime Individual Risk	Calculated from author's data. Population risk in Column K divided by exposed population in Column J multiplied by 50 year lifetime of exposure.
J	Exposed Population	Fong considers the entire U.S. population (220,000,000) to be exposed to ETS according to his scenario. If the same value for population at risk given by Repace and Lowrey were used here (62,400,000), the population risk would be 2600 deaths per year.
K	Risk to Exposed Population	The number of excess deaths in the U.S. attributed to active smoking is 350,000 per year. This number includes deaths due to coronary heart disease and cancers at sites other than the lung in addition to lung cancer and emphysema. Since non-smokers are estimated to be exposed 1/13 as much as active smokers and 37% of the population smokes, the maximum population risk of death due to ETS is calculated as: (350,000 deaths/year) x (1/13) x (63/37) = 50, 000 deaths/year (rounded). However, Fong cites the Japanese study by Hirayama (1981) as evidence that ETS does not present the same risk of heart disease and other cancers as active smoking, so these effects are not considered in the estimation of population risk. Lung cancer and emphysema account for 20% of the total deaths due to active smoking. Thus, a more "realistic" estimate of population risk is 50,000 x 0.20 = 10,000 deaths/year.

Figure 12. ETS: Fong (1982)
Page 3 of 3

RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	All sources	Organics		Long duration Lifetime: 70 yrs Indoors, home		Calculated		Contact rate: 20cu.m./day Absorption rate: 100% Average body weight: 70kg 1 mg = 1000 μg Conversion factor: 3E-4 cu.m. x mg / day x μg x kg		Dose not presented by author. Computed from (E)x(F). Continuous dose over a lifetime mg x lifetime / kg x day		Carcinogenic potency kg x day / mg		Incidence: cancer		Los Angeles, CA residents		
	Pollutants	μg/cu.m.		lifet.me		μg/cu.m. x lifetime												
	1) benzene	1) 13.6		1.0		1) 13.6				1) 4.1 E-3		1) 1.0 E-3		1) 3.9 E-6				
	2) ethylbenzene	2) 7.0				2) 7.0				2) 2.1 E-3		2) 3.4 E-4		2) 6.8 E-7				
	3) o-xylene	3) 8.9				3) 8.9				3) 2.7 E-3		3) 9.9 E-4		3) 2.5 E-6				
	4) m & p xylene	4) 20.3				4) 20.3				4) 6.1 E-3		4) 9.9 E-4		4) 5.7 E-6				
	5) trichloro-ethylene	5) 1.2				5) 1.2				5) 3.6 E-4		5) 1.7 E-3		5) 5.7 E-7				
	6) tetrachloro-ethylene	6) 8.9				6) 8.9				6) 2.7 E-3		6) 9.2 E-3		6) 2.4 E-5				
	7) chloroform	7) 1.5				7) 1.5				7) 4.5 E-4		7) 1.4 E-2		7) 6.0 E-6				
	8) 1,1,1-tri-chloroethane	8) 26.6				8) 26.6				8) 8.0 E-3		8) 1.7 E-5		8) 1.3 E-7				
	9) styrene	9) 2.4				9) 2.4				9) 7.2 E-4		9) 1.9 E-3		9) 1.3 E-6				
Qualitative Information	← Hazard Identification → The pollutants shown in column (B) are those which EPA considers probable human carcinogens (A or B Classification). The authors present risk estimates for a total of 52 chemicals.																	
Quantitative Information	← Exposure Assessment → The values in columns (C) and (E) are taken from Table 3 of the referenced article labeled as geometric mean concentrations derived from measured data presented in the Los Angeles, CA. TEAM study (Pellizzari et al. (1986)).									← Dose-Response → Median estimates of carcinogenic potency (Table 1) are derived from animal bioassay data for all compounds shown except for benzene, which is derived from human data.				← Risk Characterization → Median estimates of lifetime risk of cancer are taken from Table 4b of the referenced article. Multiplication of columns (G)x(H) give slightly different estimates than those in (I). Differences are attributed to rounding error.				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: Tancrede, M., R. Wilson, L. Zeise, and E. Crouch, (1987), "The Carcinogenic Risk of Some Organic Vapors Indoors: A Theoretical Survey." Atmospheric Environment, 21:10:2187-2205.
2. Additional comments: This table shows Tancrede's risk assessment methodology applied to indoor air concentrations of individual chemicals measured by the Los Angeles, CA. TEAM study. Median lifetime risk estimates are shown.
3. See key assumptions and comments on pages 2 and 8 of Figure 18.

Figure 13. VOC: Tancrede et al. (1987)
Los Angeles, CA: Median Estimates
Page 1 of 8

Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation Used by Tancrede et al. (1987) (see further explanation in following text)	Median estimates of lifetime risk (R) to an individual: $R = 3 \times 10^{-4} \beta d$ β = carcinogenic potency, $(mg/kg \text{ per day})^{-1}$ d = concentration, $\mu g/m^3$ 3×10^{-4} converts from μg to mg and accounts for an inhalation rate of $20 m^3$/day and an average body weight of 70 kg ($3 \times 10^{-4} = 20 \times \frac{1}{70} \times 1/1000$). Tancrede et al. assume a lognormal distribution for each factor in the equation and use a median potency factor and median concentration measurement in the equation.
B	Source Factor	Tancrede uses measured values of the various organic compounds, so all sources are included.
C	Pollutant Concentration	The compounds shown in this table are those measured by the TEAM study for which human or animal bioassay data were available to estimate the carcinogenic potency. The geometric mean pollutant concentration shown are taken from Table 3 of the referenced article as calculated from the Los Angeles, CA TEAM study.
D	Exposure Duration/Setting	Overnight personal air samples collected during the winter months are assumes to be representative of lifetime exposure to indoor air. It is also assumed that most people spend 80% to 90% of their time indoors, and are continuously exposed to the measured concentrations. Nighttime measured concentrations were assumed to be typical over a lifetime.

Figure 13. VOC: Tancrede et al. (1987)
Los Angeles, CA: Median Estimates
Page 2 of 8

Key Assumptions and Comments on Data

Column	Data	Comments
E	Exposure	Tancrede et al. do not explicitly calculate lifetime exposures. Data shown here as pollutant concentration x lifetime exposure duration.
F	Dosimetry Factors	Tancrede et al. use a dosimetry factor of 3×10^{-4}. The units on this dosimetry factor are $(m^3/day) \times (1/kg) \times (mg/\mu g)$. This dosimetry factor was developed assuming 20 cu.m./day of air breathed, absorption rate of 100% and average human body weight of 70 kg and a conversion of 1000 μg = 1 mg.
H	Response Factor	The authors use median estimates of carcinogenic potency for compounds for which human or animal bioassay data exist. They do not use the 95% upper confidence limit potency value recommended by EPA. Benzene is the only compound for which potency estimates are derived from human data. For chemicals from which potency is calculated using animal bioassay data, the following equation is used: $\beta = \log_e (2)/TD_{50}$ where TD_{50} = the dose leading to 50% lifetime tumor incidence in the animal population at a specific dose level. A standard deviation for the TD_{50} was obtained by using the reported upper and lower 99% confidence limit on TD_{50}. The median estimates shown in Column H are taken from Table 1 of the referenced article.
I	Lifetime Individual Risk	The median lifetime individual risk is estimated by the authors using the equation given in comments for Column A. The values shown in Column I are taken from Table 4b of the referenced article and are slightly different from values obtained by multiplying Columns G x H. Differences are attributed to rounding error.

Figure 13. VOC: Tancrede et al. (1987)
Los Angeles, CA: Median Estimates
Page 3 of 8

RISK CHARACTERIZATION FRAMEWORK

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	All sources	Organics		Long duration Lifetime: 70 yrs Indoors, home		Calculated		Contact rate: 20 cu.m./day Absorption rate: 100% Average body weight: 70 kg 1 mg = 1000 µg Conversion factor: 3E-4 cu.m. x mg / day x µg x kg		Dose not presented by author. Computed from (E)x(F). Continuous dose over a lifetime mg x lifetime / kg x day		Carcinogenic potency kg x day / mg $\exp(\sigma^2/2)$		Incidence: cancer		Los Angeles, CA residents		
	Pollutants	µg/cu.m.		lifetime		µg/cu.m. x lifetime (σ_z)												
	1) benzene	1) 13.6		1.0		1) 13.6 (1.0)				1) 4.1 E-3		1) 1.0 E-8 (22)		1) 9.0 E-5				
	2) ethyl benzene	2) 7.0				2) 7.0 (1.1)				2) 2.1 E-3		2) 3.4 E-4 (24)		2) 1.7 E-5				
	3) o-xylene	3) 8.9				3) 8.9 (1.0)				3) 2.7 E-3		3) 9.9 E-4 (19)		3) 5.0 E-5				
	4) m & p xylene	4) 20.3				4) 20.3 (0.8)				4) 6.1 E-3		4) 9.9 E-4 (16.3)		4) 9.8 E-5				
	5) trichloro-ethylene	5) 1.2				5) 1.2 (2.0)				5) 3.6 E-4		5) 1.7 E-3 (77)		5) 4.7 E-5				
	6) tetrachloro-ethylene	6) 8.9				6) 8.9 (0.9)				6) 2.7 E-3		6) 9.2 E-3 (16.4)		6) 4.1 E-4				
	7) chloroform	7) 1.5				7) 1.5 (1.0)				7) 4.5 E-4		7) 1.4 E-2 (18)		7) 1.1 E-4				
	8) 1,1,1-tri-chloroethane	8) 26.6				8) 26.6 (1.8)				8) 8.0 E-3		8) 1.7 E-5 (68.5)		8) 9.3 E-6				
	9) styrene	9) 2.4				9) 2.4 (1.8)				9) 7.2 E-4		9) 1.9 E-8 (28)		9) 8.9 E-				
Qualitative Information	← Hazard Identification → The pollutants shown in column B are those for which EPA considers probable human carcinogens (A or B classification). The authors present risk estimates for a total of 52 chemicals.																	
Quantitative Information	← Exposure Assessment → Pollutant concentration estimates are taken from Table 3 of the referenced article labeled as geometric mean concentrations derived from measured data presented in the Los Angeles, CA. TEAM study (Pellizzari et al. 1986).									← Dose-Response → Median estimates of carcinogenic potency in Table 1 of the article are derived from animal bioassay data for all compounds shown except for benzene, which is derived from human data.				← Risk Characterization → Mean estimates of individual lifetime risk of cancer are taken from Table 4a of the referenced article. The term $\exp(\sigma^2/2)$ is back calculated based on columns (G), (H) and (I).				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Additional comments: This table shows Tancrede's risk assessment methodology applied to indoor air concentrations of individual chemicals measured by the Los Angeles, CA. TEAM study. Mean lifetime risk estimates are shown.
2. See key assumptions and comments on pages 5 and 6 of Figure 13.

Figure 13. VOC: Tancrede et al. (1987)
Los Angeles, CA: Mean Estimates
Page 4 of 8

Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation used by Tancrede et al. (1987)	Mean or average estimate of lifetime risk to an individual: $R = 3 \times 10^{-4} \beta d \exp(\sigma^2/2)$ β = carcinogenic potency, $(mg/kg \text{ per day})^{-1}$ d = concentration, $\mu g/m^3$ σ^2 = uncertainty factor, including uncertainty in potency, interspecies extrapolation, route-to-route extrapolation, and variability in dose 3×10^{-4} converts from μg to mg and accounts for an inhalation rate of $20 m^3$/day and an average body weight of 70 kg Tancrede assumes a lognormal distribution for each factor in the equation, and thus the mean risk estimate is greater than the median risk estimate by the factor of $\exp(\sigma^2/2)$. Tancrede defines σ^2 by the following equation: $\sigma^2 = \sigma^2_x + \sigma^2_y + \sigma^2_t + \sigma^2_z$ where σ_x = the uncertainty in measuring potency in animal experiments, σ_y = the uncertainty in extrapolating from animal to humans assumed to be $\log_e(4.5) = 1.5$, σ_t = the uncertainty due to different routes of administration and assumed to be $\log_e(5) = 1.6$, and σ_z = the uncertainty (variability) in dose.
B	Source Factor	Same as Page 2.
C	Pollutant Concentration	The compounds shown in this table are those measured in the TEAM study for which human or animal bioassay data were available to estimate the carcinogenic potency. The geometric mean concentrations shown are taken from Table 3 of the referenced article as calculated from the Los Angeles, CA TEAM Study.
D	Exposure Duration/Setting	Same as Page 2.

Figure 13. VOC: Tancrede et al. (1987)
Los Angeles, CA: Mean Estimates
Page 5 of 8

Key Assumptions and Comments on Data

Column	Data	Comments
E	Exposure	Same as Page 3.
F	Dosimetry Factors	Same as Page 3.
H	Response Factor	The median estimates shown in Column H are taken from Table 1 of the referenced article. Included in Column H is the term $\exp(\sigma^2/2)$. The σ^2 term is defined above in the comment to Column A as the sum of four components: uncertainty in potency (σ_X); uncertainty in extrapolating from animals to humans (σ_Y); uncertainty due to different routes of administration (σ_t); and uncertainty in dose (σ_Z). We have calculated σ^2 for each of the compounds in Figure 13. Also shown in Column H is $\exp(\sigma^2/2)$.
I	Lifetime Individual Risk	The median lifetime individual risk is estimated by the authors using the equation given in comments for Column A. The values shown in Column I are taken from Table 4a of the reference article and are the same as the values obtained by multiplying Column G x Column H on page 4 of this figure.

Figure 13. VOC: Tancrede et al. (1987)
Los Angeles, CA: Mean Estimates
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RISK CHARACTERIZATION FRAMEWORK

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	All sources Pollutants	Organics µg/cu.m.		Long duration Lifetime: 70 yrs Indoors, home		Calculated µg/cu.m. x lifetime (σ_z)		Contact rate: 20 cu.m./day Absorption rate: 100% Average body weight: 70 kg		Dose not presented by author. Computed from (E)x(F). Continuous dose over a lifetime. mg x lifetime / kg x day		Carcinogenic potency kg x day / mg exp(2.1σ)		Incidence: cancer		Los Angeles, CA residents		
	1) benzene	1) 13.6				1) 13.6 (1.0)				1) 4.1 E-3		1) 1.0E-3(163)		1) 6.7 E-4				
	2) ethylbenzene	2) 7.0		365 days/yr x		2) 7.0 (1.1)		1 mg = 1000 µg		2) 2.1 E-3		2) 3.4E-4(170)		2) 1.2 E-4				
	3) o-xylene	3) 8.9		70 yrs/lifetime		3) 8.9 (1.0)				3) 2.7 E-3		3) 9.9E-4(142)		3) 3.8 E-4				
	4) m & p xylene	4) 20.3				4) 20.3 (0.8)		Conversion factor:		4) 6.1 E-3		4) 9.9E-4(125.8)		4) 7.6 E-4				
	5) trichloro-ethylene	5) 1.2				5) 1.2 (2.0)		3E-4 cu.m. x mg / day x µg x kg		5) 3.6 E-4		5) 1.7E-3(408.5)		5) 2.5 E-4				
	6) tetrachloro-ethylene	6) 8.9				6) 8.9 (0.9)				6) 2.7 E-3		6) 9.2E-3(128.8)		6) 3.2 E-3				
	7) chloroform	7) 1.5				7) 1.5 (1.0)				7) 4.5 E-4		7) 1.4E-2(139.7)		7) 8.8 E-4				
	8) 1,1,1-tri-chloroethane	8) 26.6				8) 26.6 (1.8)				8) 8.0 E-3		8) 1.7E-5(382)		8) 5.2 E-5				
	9) styrene	9) 2.4				9) 2.4 (1.3)				9) 7.2 E-4		9) 1.9E-3(197)		9) 2.7 E-4				
Qualitative Information	← Hazard Identification → The pollutants shown in column B are those which EPA considers probable human carcinogens (A or B classification). The authors present risk estimates for a total of 52 chemicals.																	
Quantitative Information	← Exposure Assessment → Pollutant concentration estimates are taken from Table 3 of the referenced article labeled as geometric mean concentrations derived from measured data presented in the Los Angeles, CA. TEAM study (Pellizzari et al. 1986).									← Dose-Response → Median estimates of carcinogenic potency (Table 1) derived from animal bioassay data for all compounds shown except benzene, which is derived from human data.				← Risk Characterization → The 98th percentile estimates of lifetime risk of cancer are taken from Table 4c of the referenced article. The term exp(2.1σ) is back calculated based on columns (G), (H) and (I).				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Additional comments: This table shows Tancrede's risk assessment methodology applied to indoor air concentrations of individual chemicals measured by the Los Angeles, CA. TEAM study.
2. See key assumptions and comments on page 8 of Figure 13.

Figure 13. VOC: Tancrede et al. (1987)
Los Angeles, CA: 98th Percentile Estimates
Page 7 of 8

Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation Used by Tancrede et al. (1987)	The 98th percentile estimate of lifetime risk to an individual: $R_{98} = 3 \times 10^{-4} \times \beta \times d \exp(2.0537\sigma)$ β = carcinogenic potency, (mg/kg per day)$^{-1}$ d = concentration, $\mu g/m^3$ σ^2 = uncertainty factor, including uncertainty in potency, interspecies extrapolation, route-to-route extrapolation, and variability in dose 3×10^{-4} converts from μg to mg and accounts for an inhalation rate of 20m^3/day and an average body weight of 70 kg Tancrede assumes a lognormal distribution for each factor in the equation, and thus the mean risk estimate is greater than the median risk estimate by the factor of $\exp(\sigma^2/2)$. Tancrede uses a median potency factor and median concentration measurement in the equation. The estimate of σ is critical to the equations and is defined on page 5 of this Figure.
B	Source Factor	Same as Page 2.
C	Pollutant Concentration	Same as Page 2.
D	Exposure Duration/Setting	Same as Page 2.
E	Exposure	Same as Page 3.
F	Dosimetry Factors	Same as Page 3.
H	Response Factor	The median estimates shown in Column H are taken from Table 1 of the referenced article. Included in Column H is the term $\exp(2.0537\sigma)$. The term σ^2 is defined above in the comment to Column A and Column H on page 6. Shown in Column H is $\exp(2.0537\sigma)$.
I	Lifetime Individual Risk	The 98th percentile lifetime individual risk is estimated by the authors using the equation given in comments for Column A. The values shown in Column I are taken from Table 4c of the referenced article and are the same as the values obtained by multiplying Column G x Column H on page 7 of this figure.

Figure 13. VOC: Tancrede et al. (1987)
Los Angeles, CA: 98th Percentile Estimates
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RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	All sources	Organics		Long duration Lifetime 70 yrs All settings		Integrated by direct measurement		Contact rate: 20 cu.m./day Absorption rate: 100% Average body weight: 70 kg Lifetime: 70 yrs mg = 1000 µg 1.12 E-8		Pollutant mass per body weight per time		Carcinogenic potency		Incidence: cancer		All U.S. metropolitan residents in 1983		Cancer cases per year Columns (I)x(J) / 70
	Pollutants	µg/cu.m.		365 x 70 days / lifetime = 25,550 days		µg x days / cu.m. lifetime				mg / kg x day		kg x day / mg						
	1) benzene	1) 20				1) 5.1 E+5				1) 5.7 E-3		1) 2.8 E-2		1) 1.6 E-4		178,000,000		1) 400
	2) p-dichloro-benzene	2) 35				2) 8.9 E+5				2) 1.0 E-2		2) 1.4 E-2		2) 1.4 E-4				2) 356
	3) chloroform	3) 3				3) 7.7 E+4				3) 8.6 E-4		3) 8.0 E-2		3) 6.9 E-5				3) 170
	4) carbon tetra-chloride	4) 3				4) 7.7 E+4				4) 8.6 E-4		4) 5.3 E-2		4) 4.5 E-5				4) 110
	5) tetrachloro-ethylene	5) 15				5) 3.8 E+5				5) 4.3 E-3		5) 2.0 E-3		5) 9.0 E-6				5) 23
	6) trichloro-ethylene	6) 7				6) 1.8 E+5				6) 2.0 E-3		6) 4.5 E-3		6) 9.0 E-6				6) 20
Qualitative Information	← Hazard Identification →																	
Quantitative Information	← Exposure Assessment → Arithmetic mean concentrations based on 24-hour average exposures of 640 persons in Bayonne-Elizabeth, NJ; Los Angeles, CA; and Antioch-Pittsburg, CA. TEAM studies.									← Dose-Response → The carcinogenic potency factors were calculated from the unit risk factors provided in Table III of the referenced article.				← Risk Characterization → Values in columns (I) and (K) are from Table III of the referenced article. Column (I) is based on arithmetic mean concentrations while column (K) is based on population-weighted arithmetic mean concentrations. The author assumes a U.S. metropolitan population of 178,000,000.				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: Wallace, L. A., (1985), "Cancer Risks from Organic Chemicals in the House." Proceedings of an APCA International Speciality Conference on Environmental Risk: Is Analysis Useful?
2. Additional comments: See page 2 and 3.

Figure 14. VOC: Wallace (1986)
Upperbound Estimates for Metropolitan Areas
Page 1 of 5

Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation used in Wallace (1985)	For individual risk: $y_i = ax_i$ y_i = risk of excess cancer incidence from lifetime exposure to carcinogen of interest (See Column I) a = potency of carcinogen expressed as a unit risk factor in units of excess cases per unit concentration normalized to a 70 kg male with a 70 year lifetime. x_i = mean lifetime exposure concentration of carcinogen For population risk: Y = Pawx Y = cases/year of cancer in a given population due to exposure to the chemical of concern (See Column K) P = target population (See Column J) a = potency factor (as described above) wx = population weighted arithmetic mean exposure concentration
B	Source Factors	The exposure data used by Wallace are for six organic chemicals measured in EPA's TEAM study, which used personal exposure monitors to measure the total exposure of individuals to organic chemicals from all sources in two consecutive 12-hour periods.
C	Concentration	The values in Column C are arithmetic mean concentrations based on 24-hour average exposures of about 540 persons in Bayonne-Elizabeth, NJ; Los Angeles, CA; and Antioch-Pittsburg, CA; for benzene, para-dichlorobenzene, chloroform, carbon tetrachloride, tetrachloroethylene, and trichloroethylene.

Figure 14. VOC: Wallace (1986)
Upperbound Estimates for Metropolitan Areas
Page 2 of 5

Key Assumptions and Comments on Data

Column	Data	Comments
F	Dosimetry Factors	A dosimetry factor of 1.12×10^{-8} was calculated by using the following equation: ($20 m^3$/day inhaled air)(100% absorbed)/70(kg)body weight) (1000 µg/mg)(25550 days/lifetime). Units of the dosimetry factor are (mg/kg per day) x $(\mu g/m^3)^{-1}$ x $(\text{days/lifetime})^{-1}$.
H	Carcinogenic Potency	Unit risk factors, were converted to potency factors by dividing by a contact rate of 20 m^3/day and multiplying by an average body weight of 70 kg. The unit risk factors used by Wallace are CAG upper bound estimates, except for benzene which is a best estimate based on human data, and para-dichlorobenzene, which is based on the NTP mouse potency of 1.5×10^{-3} $(\text{mg/kg per day})^{-1}$ multiplied by uncertainty factor of 10 for interspecies extrapolation.
I	Lifetime Individual Risk	The values shown in Column I are taken from Table III of the referenced article. They also can be calculated by multiplying across the columns of the framework starting with the arithmetic mean concentrations shown in Column C.
J	Exposed Population	Since the exposure data used from the TEAM Study were from metropolitan areas, Wallace assumed these data are generally representative of all U.S. metropolitan areas and thus, used the U. S. metropolitan population in 1983 of 178×10^6 people to calculate population risk.
K	Risk to Exposed Population	The values shown in Column K are taken from Table III of the referenced article. These estimates are based on population weighted arithmetic mean exposure concentrations. They also can be calculated by multiplying Column I x Column J and dividing the product by 70 years.

Figure 14. VOC: Wallace (1986)
Upperbound Estimates for Metropolitan Areas
Page 3 of 5

RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	All sources	Organics		Long duration Lifetime: 70 yrs All settings		Integrated by direct measurement		Contact rate: 20 cu.m./day Absorption rate: 100% Average body weight: 70 kg Lifetime: 70 yrs mg = 1000 µg		Pollutant mass per body weight per time		Carcinogenic potency		Incidence: cancer		All U.S. non-metropolitan residents in 1983		Cancer cases per year Columns (I)x(J) / 70
	Pollutants	µg/cu.m.				µg / cu.m. x days / lifetime				mg / kg x day		kg x day / mg						
	1) benzene	1) 10		365 x 70 days / lifetime = 25,550 days		1) 2.6 E+5		1.12 E-8		1) 2.9 E-3		1) 2.8 E-2		1) 8.0 E-5		56,000,000		1) 64
	2) p-dichloro-benzene	2) 12				2) 3.1 E+5				2) 3.4 E-3		2) 1.4 E-2		2) 4.8 E-5				2) 38
	3) chloroform	3) 2				3) 5.1 E+4				3) 5.7 E-4		3) 8.0 E-2		3) 4.6 E-5				3) 37
	4) carbon tetra-chloride	4) 1				4) 2.6 E+4				4) 2.9 E-4		4) 5.3 E-2		4) 1.5 E-5				4) 12
	5) tetrachloro-ethylene	5) 15				5) 3.8 E+5				5) 4.3 E-3		5) 2.0 E-3		5) 9.0 E-6				5) 7
	6) trichloro-ethylene	6) 4				6) 1.0 E+5				6) 1.1 E-3		6) 4.5 E-3		6) 5.0 E-6				6) 4
Qualitative Information	← Hazard Identification →																	
Quantitative Information	← Exposure Assessment → Arithmetic mean concentrations based on 24-hour average exposures of approximately 50 persons in Greensboro, NC and Devils Lake, ND.									← Dose-Response → The carcinogenic potency factors were calculated from the unit risk factors provided in Table III of the referenced article.				← Risk Characterization → Values in columns (I) and (K) are from Table III of the referenced article. Column (I) is based on arithmetic mean concentrations while column (K) is based on arithmetic mean concentrations. The author assumes a U.S. nonmetropolitan population of 56,000,000.					
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: Wallace, L. A., (1985), "Cancer Risks from Organic Chemicals in the House." Proceedings of an APCA International Speciality Conference on Environmental Risk: Is Analysis Useful?
2. Additional comments: See page 4.

Figure 14. VOC: Wallace (1986)
Upperbound Estimates for Nonmetropolitan Areas
Page 4 of 5

Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation used in Wallace (1985)	For individual risk: Same as Page 2. For population risk: Same as Page 2.
B	Source Factors	Same as Page 2.
C	Concentration	The values in Column C are arithmetic mean concentrations based on 24-hour average exposures of about 50 persons in Greensboro, NC and Devils Lake, ND for for benzene, para-dichlorobenzene, chloroform, carbon tetrachloride, tetra-chloroethylene, and trichloroethylene.
F	Dosimetry Factors	Same as Page 3.
H	Carcinogenic Potency	Same as Page 3.
I	Lifetime Individual Risk	The values shown in Column I are taken from Table III of the referenced article. They also can be calculated by multiplying across the columns of the framework starting with the arithmetic mean concentrations shown in Column C.
J	Exposed Population	Since the exposure data used from the TEAM Study were from nonmetropolitan areas, Wallace assumed these data are generally representative of all U.S. non-metropolitan areas and thus, used the U. S. nonmetropolitan population in 1983 of 56 x 10^6 people to calculate population risk.
K	Risk to Exposed Population	The values shown in Column K are taken from Table III of the referenced article. These estimates are based on population weighted arithmetic mean exposure concentrations. They also can be calculated by multiplying Column I x Column J, and dividing the product by 70 years.

Figure 14. VOC: Wallace (1986)
Upperbound Estimates for Nonmetropolitan Areas
Page 5 of 5

RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	All sources Pollutants 1) benzene 2) 1,2-dibromo-ethane 3) 1,2-dichloro-ethane 4) dichloro-methane 5) dimethyl nitrosoamine 6) tetrachloro-ethylene 7) trichloro-ethylene 8) vinylidene--chloride	Organics µg/cu.m. max. (mean): 1) 387 (13.9) 2) 0.1 (NR) 3) 69 (1.1) 4) 5000 (225) 5) 0.8 (NR) 6) 716 (4.5) 7) 178 (3.4) 8) 418 (2.8)		Long duration Lifetime: 70 yrs Indoors, home 365 x 70 days / lifetime = 25,550 days		Calculated µg/cu.m. x days max.(mean): 1) 9.9 E+6 (3.6 E+5) 2) 2.6 E+3 (NR) 3) 1.8 E+6 (2.8 E+4) 4) 1.3 E+8 (5.8 E+6) 5) 2.0 E+4 (NR) 6) 1.8 E+7 (1.2 E+5) 7) 4.6 E+6 (8.7 E+4) 8) 1.1 E+7 (7.2 E+4)		Contact rate: 20 cu.m./day Absorption rate: 100% Average body weight: 70 kg Lifetime: 70 yrs Conversion factor: 1.12 E-8 cu.m. x mg / dayxµgxkg		mg / kg x day max. (mean): 1) 1.1 E-1 (3.9 E-3) 2) 2.9 E-5 (NR) 3) 2.0 E-2 (3.2 E-4) 4) 1.4 (6.4 E-2) 5) 2.3 E-4 (NR) 6) 2.1 E-1 (1.3 E-3) 7) 5.1 E-2 (9.7 E-4) 8) 1.2 E-1 (8.0 E-4)		Maximum likelihood potency estimate (kg x day/mg) 1) 1.2 E-2 2) 4.9 E-1 3) 3.2 E-2 4) 1.7 E-3 5) 1.7 6) 6.3 E-3 7) 3.0 E-3 8) 9.0 E-2		Incidence: cancer max. (mean): (x10^{-5}) 1) 128 (4.6) 2) 1.4 (NR) 3) 62 (1.0) 4) 180 (8.1) 5) 38 (NR) 6) 130 (0.8) 7) 15 (0.3) 8) 1080 (7.3)			
Qualitative Information	← Hazard Identification → Of the 24 chemicals for which risk estimates are provided in the referenced article, eight are shown here. These eight were selected because EPA has estimated carcinogenic unit risk factors for them.																	
Quantitative Information	← Exposure Assessment → The authors compiled indoor air concentration measurement data for 24 chemicals, and recorded the maximum and mean values measured. Table 1 of the referenced article presents these data. Maxima shown in column (C) are from Table 1 and the mean (in parentheses) are the average of all means or medians reported in Table 1.									← Dose-Response → Maximum likelihood estimates of carcinogenic potency were estimated using the multistage model, assuming additivity. Tumor data are from Gold et al. (1984, 1986).				← Risk Characterization → Column (I) shows lifetime individual risk estimates based on both maximum and mean pollutant concentrations and the maximum likelihood estimates of carcinogenic potency. Estimates are from Table 3 of the referenced article.				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: McCann, J., L. Hora, J. Girman, A. Nero, (1986), "Potential Risks from Exposure to Organic Carcinogens in Indoor Air," Presented at EPA Symposium on the Application of Short-term Bioassays in the Analysis of Complex Environmental Mixtures, Durham, NC.
2. Additional comments: See page 2.

Figure 15. VOC: McCann et al. (1986)
Maximum Estimates of Potency
Page 1 of 4

Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation used by McCann et al. (1986)	Concentration ($\mu g/m^3$) x unit risk factor ($m3/\mu g$) = lifetime individual risk.
B	Source Factors	McCann et al. reviewed the literature for indoor air concentrations of 140 compounds. All concentration data are direct field measurements in homes and public buildings, and thus represent multiple sources of the pollutant. The authors selected 24 carcinogens for which dose-response factors could be estimated for preliminary assessment of risks. The eight compounds shown in the table are those for which EPA unit risk factors are available. (Formaldehyde is shown in a separate table).
C	Pollutant Concentration	The authors report both maximum and mean or median concentrations from the literature. They then selected the highest of the maximum values reported and calculated an average of the mean values reported for use in their risk assessment. Both maximum and mean (in parentheses) concentrations are shown in Column C.
F	Dosimetry Factors	A dosimetry factor of 1.12×10^{-8} was calculated using the following equation ($20m^3$/day inhaled air)(100% absorbed)/[(70 kg body weight)(1000 $\mu g/mg$) (25550 days/lifetime)]. Units of the dosimetry factor are (mg/kg per day) x $(\mu g/m^3)^{-1}$ x $(days/lifetime)^{-1}$.
H	Response Factor	McCann et al., in general, do not provide the potency or unit risk estimates that they used except for several of the EPA unit risk numbers. The maximum likelihood estimates (MLE) of carcinogenic potency were back calculated by dividing the MLE of lifetime individual risk estimated from maximum concentrations and mean concentrations (Column I) by the corresponding maximum or mean dose (Column G).
I	Lifetime Individual Risk	McCann et al. estimate risk based on maximum likelihood estimates of carcinogenic potency using the multistage model and maximum or mean concentrations assuming lifetime exposures. Shown in Column I are the MLE risk estimates for eight carcinogens calculated from maximum concentrations and mean concentrations (in parentheses) taken from Table 3 of the referenced article.

Figure 15. VOC: McCann et al. (1986)
Maximum Estimates of Potency
Page 2 of 4

RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	All sources Pollutants 1) benzene 2) 1,2-dibromo-ethane 3) 1,2-dichloro-ethane 4) dichloro-methane 5) dimethyl nitrosoamine 6) tetrachloro-ethylene 7) trichloro-ethylene 8) vinylidene-chloride	Organics µg/cu.m. max. (mean): 1) 387 (13.9) 2) 0.1 (NR) 3) 69 (1.1) 4) 5000 (225) 5) 0.8 (NR) 6) 716 (4.5) 7) 178 (3.4) 8) 418 (2.8)		Long duration Lifetime: 70 yrs Indoors, home 365 x 70 days lifetime = 25,550 days		Calculated µg/cu.m. x days max.(mean): 1) 9.9 E+6 (3.6 E+5) 2) 2.6 E+3 (NR) 3) 1.8 E+6 (2.8 E+4) 4) 1.3 E+8 (5.8 E+6) 5) 2.0 E+4 (NR) 6) 1.8 E+7 (1.2 E+5) 7) 4.6 E+6 (8.7 E+4) 8) 1.1 E+7 (7.2 E+4)		Contact rate: 20 cu.m./day Absorption rate: 100% Average body weight: 70 kg Lifetime: 70 yrs Conversion factor: 1.12 E-8 cu.m. x mg day x µg x kg		mg kg x day max. (mean): 1) 1.1 E-1 (3.9 E-3) 2) 2.9 E-5 (NR) 3) 2.0 E-2 (3.2 E-4) 4) 1.4 (6.4 E-2) 5) 2.3 E-4 (NR) 6) 2.1 E-1 (1.3 E-3) 7) 5.1 E-2 (9.7 E-4) 8) 1.2 E-1 (8.0 E-4)		Maximum likeli-hood potency estimate (kg x day/mg) 1) 1.2 E-2 2) 4.9 E-1 3) 3.2 E-2 4) 1.7 E-3 5) 1.7 6) 6.3 E-3 7) 3.0 E-3 8) 9.0 E-2		Incidence: cancer max. (mean): ($x10^{-5}$) 1) 128 (4.6) 2) 1.4 (NR) 3) 62 (1.0) 4) 180 (8.1) 5) 38 (NR) 6) 130 (0.8) 7) 15 (0.3) 8) 1080 (7.3)			
Qualitative Information	Hazard Identification Of the 24 chemicals for which risk estimates are provided in the referenced article, eight are shown here. These eight were selected because EPA has estimated carcinogenic unit risk factors for them.																	
Quantitative Information	Exposure Assessment The authors compiled indoor air concentration measurement data for 24 chemicals, and recorded the maximum and mean values measured. Table 1 of the referenced article presents these data. Maxima shown in column (C) are from Table 1 and the mean (in parentheses) are the average of all means or medians reported in Table 1.									Dose-Response Maximum likelihood estimates of carcinogenic potency were estimated using the multistage model, assuming additivity. Tumor data are from Gold et al. (1984, 1986).				Risk Characterization Column (I) shows lifetime individual risk estimates based on both maximum and mean pollutant concentrations and the maximum likelihood estimates of carcinogenic potency. Estimates are from Table 3 of the referenced article.				
Qualitative or Quantitative Analysis	Risk Assessment																	

Footnotes: 1. Reference: McCann, J., L. Horn, J. Girman, A. Nero, (1986), "Potential Risks from Exposure to Organic Carcinogens in Indoor Air," Presented at EPA Symposium on the Application of Short-term Bioassays in the Analysis of Complex Environmental Mixtures, Durham, NC.
2. Additional comments: See page 2.

Figure 15. VOC: McCann et al. (1986)
Upper 95% Estimates of Potency
Page 3 of 4

Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation used by McCann et al. (1986)	Same as Page 2.
B	Source Factors	Same as Page 2.
C	Pollutant Concentration	Same as Page 2.
F	Dosimetry Factors	Same as Page 2.
H	Response Factor	McCann et al., in general, do not provide the potency or unit risk estimates that they used except for several of the EPA unit risk numbers. The 95% upper confidence limit estimates (UCL) of carcinogenic potency were backcalculated by dividing the UCL lifetime individual risk estimated from maximum concentrations and mean concentrations (Column I) by the corresponding maximum or mean dose (Column G).
I	Lifetime Individual Risk	McCann et al. estimate risk based on the 95% upper confidence limit of carcinogenic potency using the multistage model and maximum or mean concentrations assuming lifetime exposures. Shown in Column I are the UCL risk estimates for eight carcinogens calculated from maximum concentrations and mean concentrations (in parentheses) taken from Table 3 of the referenced article.

Figure 15. VOC: McCann et al. (1986)
Upper 95% Estimates of Potency
Page 4 of 4

RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	Building materials Insulation	Organics: Formaldehyde		Long duration lifetime Indoors, home		Calculated from columns (C)x(D) mg/cu.m. x yr		Inhalation rate: 20 cu.m./day Average body weight: 70 kg Average lifetime: 70 yrs 4.08 E-3 cu.m. / day x kg x yr		Calculated from columns (E)x(F) mg / kg x day		Carcinogenic potency		Incidence: lung cancer				Cases per year Calculated Columns (I)x(J) / 70 years
	1) Particle board and plywood in mobile homes	1) <0.01-2.54ppm		100-150 hrs/wk for max.15 yrs		1) <0.16 to 41.5				1) 6.5 E-4 to 0.17		Linear range 1.65 E-4 for column (C) < 1 ppm		1) 2.0 E-5 to 1.0 E-2		1) 2,200,000		1) 0.6 to 314
	2) Particle board and plywood in conventional homes	2) 2.24 ppm worst case		100-150 hrs/wk for max. 15 yrs		2) 36.6				2) 0.149		Nonlinear range See comments on page 2, Column (I) below for Column (C) >1 ppm		2) 9.0 E-3		2) Unknown		2) Unknown
	3) Urea-formaldehyde foam	3) 3.40 ppm worst case		100-150 hrs/wk for max. 15 yrs		3) 55.7				3) 0.227				3) 7.0 E-3		3) 1,330,000 to 1,560,000		3) 133 to 156
Qualitative Information	Hazard Identification CIIT study of Fischer 344 rats																	
Quantitative Information	Exposure Assessment The range of pollutant concentrations are provided for three sources of formaldehyde vapors, indoor at home. These data are taken from numerous sources reviewed by the authors.									Dose-Response The author used a linear dose-response equation for exposure concentrations below 1 ppm and a nonlinear equation for exposure concentrations above 1 ppm.				Risk Characterization The authors calculate lifetime individual risk for the range of concentrations for each of the three sources. The appropriate equations are provided in the comments below.				
Qualitative or Quantitative Analysis	Risk Assessment																	

Footnotes: 1. Reference: Interagency Regulatory Liaison Group, Task Group on Formaldehyde, (1981), Integrated Risk Assessment: Formaldehyde, Washington, DC, USGPO.
2. Additional comments: This report is a summary of many studies on exposure and resulting health effects, environmental effects, and health risks attributable to formaldehyde.
3. See key assumptions and comments on page 2 of Figure 16.

Figure 16. Formaldehyde: Interagency Regulatory Liaison Group (1981)
Page 1 of 2

Key Assumptions and Comments on Data

Column	Data	Comments
C	Pollutant Concentration	IRLG (1981) presents extensive summary tables of subpopulation data on both occupational and nonoccupational exposure to formaldehyde, including the mean, range, and worst case exposure concentrations.
E	Exposure	Parts per million of formaldehyde were converted to mg/m^3 using the formula mg/m^3 = ppm x molecular weight/24.5. Molecular weight of formaldehyde is 30.03. Multiplying the concentration by (150 hours/week) x (52 weeks/year) x (1 year/760 hrs.) x (years of exposure) gives exposure in (mg/m^3) x years. The authors assume that people spend approximately 90 percent of their time indoors, which is equivalent to 150 hours per week.
F	Dosimetry Factors	A dosimetry factor was calculated from the author's data assuming an inhalation rate of 20 m^3/day and an average body weight of 70 kg for an average lifetime of 70 years. $4.08 \times 10^{-3} = 20 m^3/day \times (70\ kg)^{-1} \times (70\ years)^{-1}$
G	Dose	Dose was calculated as Column E x Column F.
H	Response Factor	Data from the CIIT study of Fischer 344 rats was used. Evidence indicates that the dose-response curve for formaldehyde is nonlinear at high doses. However, it is hypothesized that the effects of formaldehyde may be additive to the effects of other carcinogenic processes and other carcinogens in the environment. This hypothesis implies that at low doses the dose-response curve will be linear, so a linear multi-stage model (Global 79) was used to calculate risk. The estimated risk is adjusted for the proportion of lifetime exposed, but is not adjusted for interspecies extrapolation or uncertainty. The author uses two different carcinogenic potency factors (shown below) depending on dose due to the non-linearity of the dose-response curve.
I	Lifetime Individual Risk	Lifetime individual risks were calculated by the author using the following equation: For exposure concentration < 1ppm $P(d) = 0.000165d$ For exposure concentration > 1ppm $P(d) = 1-e^{-0.00164d^3}$ where: d = dosage, e = 2.7183
K	Risk to Exposed Population	Based on the author's estimate of lifetime individual risk (Column I) and the estimated exposed population (Column J), the risk to the exposed population in terms of cases per year was calculated (Column J x Column I ÷ 70).

Figure 16. Formaldehyde: Interagency Regulatory Liaison Group (1981)
Page 2 of 2

RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	All indoor sources	Organics: Formaldehyde Median concentration 6.1 E+1 μg/cu.m. (5.0 E-2 ppm)		Long duration Indoors, home 1 lifetime		Calculated from columns (C)x(D) μg/cu.m. x lifetime 6.1 E+1		Contact rate 20 cu.m./day Absorption rate: 100% Average body weight: 70 kg 1 mg = 1000 μg Conversion factor: 3E-4 cu.m. x mg / day x μg x kg		Pollutant mass per body weight per time mg / kg x day 2.0 E-2		Carcinogenic potency kg x day / mg Potency: 98th percentile exp(2.1σ) 1.1 E-1 (118) mean exp(σ²/2) 1.1 E-1 (15.5) median 1.1 E-1		Endpoint: cancer deaths 98th percentile 2.6 E-1 mean 3.4 E-2 median 2.0 E-3				Not addressed by the authors
Qualitative Information	← Hazard Identification →																	
Quantitative Information	← Exposure Assessment →									← Dose-Response →				← Risk Characterization →				
	The authors assumed pollutant concentration and exposure values consisted with those reported in several studies.									Potency factor from inhalation bioassay studies in rats. Presented in Table 1.				Mean, median and 98th percentile estimates of risk of cancer are taken from Table 5 of the referenced article.				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: Tancrede, M., R. Wilson, L. Zeise, E. Crouch, (1987), "The Carcinogenic Risk of Some Organic Vapors Indoors: A Theoretical Survey," Atmospheric Environment, 21:10:2187-2205.
2. Additional comments: Existing literature was reviewed for exposure (NAS, 1981; IARC, 1982; Hawthorne et al., 1984; Dalley et al., 1981) and dose-response (Zeise et al., 1984).

Figure 17. Formaldehyde: Tancrede et al. (1987)
Page 1 of 2

Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation used by Tancrede (see further explanation in following text)	The set of risk equations used by Tancrede et al. for estimating the mean median and 98 percentile lifetime individual risk were: $R = 3 \times 10^{-4}\ \beta d$ (median) $R = 3 \times 10^{-4}\ \beta d\ (\exp \sigma^2/2)$ (mean) $R = 3 \times 10^{-4}\ \beta d\ (\exp 2.0537\sigma)$ (98th percentile estimate) β = Medium potency factor $(mg/kg\ per\ day)^{-1}$ d = Medium estimate of dose $(\mu g/m^3)$ σ^2 = logarithmic standard deviation This equation holds for small doses, assuming 70 kg man breathing 20 m^3/day with 100% absorption
B, C	Source Factors and Concentration	Lognormal distribution assumed, median value 0.05 ppm and logarithmic standard deviation of 1.8. Estimate is consistent with several studies for all U.S. houses. NAS (1981), IARC (1982), Hawthorne et al. (1984), Dally et al. (1981). As the assumed value of 0.05 ppm is based somewhat on studies of U.S. houses, all indoor sources of formaldehyde and thus represent all sources of the pollutant. For the average U.S. home, the authors assume uncertainty in concentration is negligible.
G	Dose	The author reported the dose as 2.0 E-2 mg-kg/day. However, conversion of 0.05 ppm yields an estimate of 1.8 E-2 mg/kg-day.
H	Response Factor	Zeise et al. (1984) derived potency factor from the inhalation study by Albert et al. (1982) of Sprague-Dawley rats. The dose-response curve for nasal cancers in rats exhibits a strong upward curvature (IARC, 1982) so that risks at low doses may be overestimated. The values for σ for the mean and 98th percentile estimates were back calculated from reported risk values in Table 5 of the referenced article. The back calculated values for the standard deviation, σ, were 1.01 and 1.54 for the 98th percentile and the mean estimates, respectively. This corresponds to $\exp(2.0537\sigma) = 118$; and $\exp(\sigma^2/2) = 15.5$. The authors presented a value for $\sigma = 1.5$.
I	Lifetime Individual Risk	Values shown in Column I are taken from Table 5 of the referenced article. These values can also be calculated by multiplying Column G x Column H, including the uncertainty terms for the 98th percentile and mean estimates.

Figure 17. Formaldehyde: Tancrede et al. (1987)
Page 2 of 2

RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	All sources	Maximum		Long duration Lifetime: 70 yrs Indoors, home				Contact rate: 20 cu.m./day Absorption rate: 100% Average body weight: 70 kg 1 year = 365 days Lifetime: 70 yrs						Incidence: cancer		All U.S. population		Percent of population with) 10 E-3
		$\frac{\mu g}{cu.m.}$		$\frac{365 days \times 70 yrs}{lifetime}$		$\frac{\mu g}{cu.m.} \times days$		$\frac{cu.m. \ year}{day \times kg \times yr \times day}$		$\frac{\mu g}{kg \times day}$		$\frac{kg \times day}{\mu g}$						Individual lifetime risk:
	Formaldehyde	500		= 25,550 days		1.3 E+7		1.1 E-5		1.4 E+2		2.2 E-5 (MLE) 6.5 E-5 (UCL) 4.6 E-5 (EPA) 6.3 E-4 (TD_{50})		3.1 E-3 (MLE) 9.3 E-3 (UCL) 6.5 E-3 (EPA) 9.0 E-2 (TD_{50})		226,545,805		(0.01 (MLE) 1.2 (UCL) NA[2] (EPA)) 99.0 (TD_{50})
Qualitative Information	← Hazard Identification →																	
Quantitative Information	← Exposure Assessment → The authors compiled indoor air concentration data from several studies, and recorded the maximum and mean values measured, presented in Table 1 of the referenced article. As these are direct field measurements, they represent all sources of the pollutant.									← Dose-Response → Estimates of unit risk values used were: Maximum likelihood estimate (MLE), upper 95% confidence level (UCL) from multistage model, EPA estimates from unit risk factors, and TD_{50} derived potency factors.				← Risk Characterization → Column (I) shows the individual risk for each of the four methods. Estimates are from Table 3 of the referenced article.				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: McCann, J., L. Horn, J. Girman, A. Nero, (1986), "Potential Risks from Exposure to Organic Carcinogens in Indoor Air," presented at EPA Symposium on the Application of Short-term Bioassays in the Analysis of Complex Environmental Mixtures, Durham, NC.
2. NA: Not analyzed
3. See key assumptions and comments on pages 2 and 3 of Figure 18.

Figure 18. Formaldehyde: McCann et al. (1986)
Maximum Measured Concentration
Page 1 of 5

(Maximum)
Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation Use by McCann et al. (see further explanation in following text	Concentration ($\mu g/m^3$) x unit risk factor ($m^3/\mu g$) = lifetime individual risk
B, C	Source Factors and Pollutant Concentration	McCann et al. reviewed the literature for indoor air concentrations. All concentration data are direct field measurements in homes and public buildings and thus represent all sources of the pollutant. Both maximum and mean concentrations were reported for each study reviewed. The actual maximum observed concentration was 3720 $\mu g/m^3$. However, at this concentration, considerable irritation would be expected. Therefore the HUD standard for mobile homes, 500 $\mu g/m^3$ was used to represent the reasonable expected maximum lifetime exposure.
F	Dosimetry Factors	Since McCann et al. used unit risk factors for their calculations of EPA values (rather than potency factors) contact rate, absorption, and average body weight are included in the unit risk factor. McCann uses CAG unit risk factors, which assume an inhalation rate of $20 m^3$/day, 100% absorption, 70 kg body weight and 70 year lifetime. A conversion of years to days is also required. These assumptions were also used for the other estimates as well. The following calculation was used: (20 cu.m./day) x $(70 kg)^{-1}$ $(70 yr)^{-1}$ x (1 year/365 days) = 1.1 E-5.
H	Response Factor	The Maximum Likelihood Estimate (MLE) and the 95% Upper Confidence Limit (UCL) estimate for carcinogenic potency for formaldehyde were directly estimated from experimental results (i.e. tumor data) used by Gold et al. (1984, 1986) to calculate the most potent TD_{50}. Note that the potency factors, MLE (Page 3) and UCL (Page 1), differ for mean and maximum concentrations. This is due to the high order of nonlinearity in the dose-response curve. McCann et al. state that the "EPA estimates are presented as 95% UCL risks and were calculated from the unit (Continued)

Figure 18. Formaldehyde: McCann et al. (1986)
Maximum Measured Concentration
Page 2 of 5

(Maximum)
Key Assumptions and Comments on Data

Column	Data	Comments
H	Response Factor (continued)	risk values for exposure to a lifetime airborne concentration of 1 $\mu g/m^3$ (Anderson, et al, 1983; EPA, 1988 a-f; EPA, 1986), assuming linearity." The unit risk factor for formaldehyde of 1.3×10^{-5} was used and was converted to cancer potency value. The TD_{50} is assumed to be a value on a linear dose-response curve and the potency factor is estimated by dividing 0.5 by the most potent TD_{50} (798 $\mu g/kg$-day). All of these unit risk values were converted to potency values using the following equation: potency = unit risk ($\mu g/m^3$) x (20 m^3/day) x (20 kg)$^{-1}$.
I	Lifetime Individual Risk	Lifetime individual risk are presented for the maximum exposure concentration combined with four different estimates of the response factor. These values are taken from Table 3 of the referenced article.
K	Risk to Exposed Population	While the authors did not calculate excess cases, they did estimate the percentage of the population at "high risk", greater than 10^{-3}. These estimates were based on the geometric mean and geometric standard deviation from DeBortoli et al. (1985), and are reported in Table 4. Estimates ranged from less than 0.01 percent for MLE to greater than 99 percent based on TD_{50}.

Figure 18. Formaldehyde: McCann et al. (1986)
Maximum Measured Concentration
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RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	All sources	Mean		Long duration Lifetime: 70 yrs Indoors, home				Contact rate: 20 cu.m./day Absorption rate: 100% Average body weight: 70 kg						Incidence: cancer				
		$\frac{\mu g}{cu.m.}$		$\frac{365 \times 70}{lifetime}$		$\frac{\mu g}{cu.m.} \times days$		Lifetime: 70 yrs		$\frac{\mu g}{kg \times day}$		$\frac{kg \times day}{\mu g}$						
		53.1		= 25,550 days		1.3 E+7		1.1 E-5		1.5 E+1		2.4 E-7 (MLE) 4.4 E-5 (UCL) 4.6 E-5 (EPA) 6.3 E-4 (TD_{50})		3.7 E-6 (MLE) 6.7 E-4 (UCL) 6.9 E-4 (EPA) 9.5 E-3 (TD_{50})				
Qualitative Information	← Hazard Identification →																	
Quantitative Information	← Exposure Assessment → The authors compiled indoor air concentration data from several studies, and recorded the maximum and mean values measured, presented in Table 1 of the referenced article. As these are direct field measurements, they represent all sources of the pollutant.									← Dose-Response → Estimates of unit risk values used were: Maximum likelihood estimate (MLE), upper 95% confidence level (UCL) from multistage model, EPA estimates from unit risk factors, and TD_{50} derived potency factors.				← Risk Characterization → Column (I) shows the individual risk for each of the four methods. Estimates are from Table 3 of the referenced article.				
Qualitative or Quantitative Analysis	← Risk Assessment →																	

Footnotes: 1. Reference: McCann, J., L. Horn, J. Girman, A. Nero, (1986), "Potential Risks from Exposure to Organic Carcinogens in Indoor Air," presented at EPA Symposium on the Application of Short-term Bioassays in the Analysis of Complex Environmental Mixtures, Durham, NC.

Figure 18. Formaldehyde: McCann et al. (1986)
Mean Concentration
Page 4 of 5

(Maximum)
Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equation Used by McCann et al.	Same as Page 2.
B, C	Source Factors and Pollutant Concentration	McCann et al. reviewed the literature for indoor air concentrations. All concentration data are direct field measurements in homes and public buildings and thus represent all sources of the pollutant. Both maximum and mean concentrations were reported for each study reviewed. The mean concentration of 53.1 $\mu g/m^3$ used by the authors is the average of all of the mean values reported in the reviewed literature.
F	Dosimetry Factors	Same as Page 2.
H	Response Factor	The Maximum Likelihood Estimate (MLE) and the 95% Upper Confidence Limit (UCL) estimate for carcinogenic potency for formaldehyde were directly estimated from experimental results (i.e. tumor data) used by Gold et al. (1984, 1986) to calculate the most potent TD_{50}. Note that the potency factors, MLE (Page 3) and UCL (Page 1), differ for mean and maximum concentrations. This is due to the high order of nonlinearity in the dose-response curve. McCann et al. state that the "EPA estimates are presented as 95% UCL risks and were calculated from the unit risk values for exposure to a lifetime airborne concentration of 1 $\mu g/m^3$ (Anderson, et al, 1983; EPA, 1988 a-f; EPA, 1986), assuming linearity." The unit risk factor for formaldehyde of 1.3×10^{-5} was used and was converted to cancer potency value. The TD_{50} is assumed to be a value on a linear dose-response curve and the potency factor is estimated by dividing 0.5 by the most potent TD_{50} (798 $\mu g/kg$-day). All of these unit risk values were converted to potency values using the following equation: potency = unit risk ($\mu g/m^3$) x (20 m^3/day) x (20 kg)$^{-1}$.
I	Lifetime Individual Risk	Lifetime individual risk are presented for the mean exposure concentration combined with four different estimates of the response factor. These values are taken from Table 3 of the referenced article.

Figure 18. Formaldehyde: McCann et al. (1986)
Mean Concentration
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RISK CHARACTERIZATION FRAMEWORK
(Data from Reference in Footnote 1)

Predictive Risk Equation (A)	Source Factors (B)	Pollutant Concentration (C)	X	Exposure Duration/Setting (D)	=	Exposure (E)	X	Dosimetry Factors (F)	=	Dose (G)	X	Response Factor (H)	=	Lifetime Individual Risk (I)	X	Exposed Population (J)	=	Risk to Exposed Population (K)
Elements of risk equation	Building materials Household products 9 Categories 1) Friction products 2) A/C pipe 3) Coatings and sealants 4) Paper products 5) V/A floor tile 6) Gaskets/ packing 7) Textiles 8) A/C Sheets 9) Plastics	Asbestos		Long duration Indoors, home Indoors, work Outdoors 5 occupational and 4 non-occupational exposure categories Durations ranged between 1 and 30 years		Weighted lifetime average from all sources Occupational: 1.4 E+7 fibers per year Nonoccupational: 1.9 E+5 fibers per year		40 hrs/wk 50 wks/yr Breathing rate = 1 m^3/hr 1 fiber/ml = 2.0 E+9 fibers per year or 1 fiber/yr = 5.0 E-10 fibers per ml		Yearly lifetime average Occupational: 7.0 E-3 f/ml Nonoccupational: 9.5 E-5 f/ml		Carcinogenic potency ml/fiber Lung cancer Occupational: 5.4 E-2 Nonoccupational: 2.8 E-2 Mesothelioma Occupational: 3.1 E-2 Nonoccupational: 4.2 E-2		Incidence Lung cancer Occupational exposure: 3.8 E-4 Nonoccupational exposure: 2.7 E-6 Mesothelioma Occupational exposure: 2.2 E-4 Nonoccupational exposure: 4.0 E-6		Children/home Adults Worker Homemaker 0.9 million occupationally exposed 150 million nonoccupationally exposed		Total excess cases Lung cancer Occupational exposure: 339 cases Nonoccupational exposure: 406 cases Mesothelioma Occupational exposure: 197 cases Nonoccupational exposure: 599 cases
Qualitative Information	← Hazard Identification →																	
Quantitative Information	← Exposure Assessment → Exposure estimates, when possible, were developed based on data collected for each stage in the product life-cycle for nine product types. These data were collected from several studies and were assumed to correspond to 1983 levels. Future exposures were based on relative changes in future production levels.									← Dose-Response → Used a relative risk model to predict lung cancer. Used an absolute risk model for mesothelioma. Occupational data assumed apply for nonoccupational exposures due to linear dose-response relationship.				← Risk Characterization → Column (K) shows the author's estimates of the total number of excess cases of lung cancer and mesothelioma that will occur over a 100 year period. The number of cases are not equal for each year of this period.				
Qualitative or Quantitative Analysis	← Risk Assessment → The author applies a life-table model approach taking into consideration time and duration of exposure, latency period, survival, other causes of death, and demographic characteristics.																	

Footnotes: 1. Reference: Mauskopf, J. A., (1987), "Projections of Cancer Risks Attributable to Future Exposure to Asbestos," Risk Analysis, 7:4:477-486.
2. Additional comments: The numbers presented in columns (E), (F), (G), and (H) have been calculated from the authors results to fit into this linear characterization framework and are not those directly reported in the article.

Figure 19. Asbestos: Mauskopf (1987)
Page 1 of 5

Key Assumptions and Comments on Data

Column	Data	Comments
A	Risk Equations Used by Mauskopf (see further explanation in following text)	Linear, no threshold dose-response relationships proposed by Nicholson (1983) were used to convert information on asbestos exposure into excess lung cancer and mesothelioma incidence rates. For lung cancer, Nicholson postulated a relative risk model that includes a 10-year latency period between onset of exposure and increased risk: $I_E = I_L K_L f d(t-10)$ where: I_E = annual excess risk of lung cancer I_L = age specific lung cancer incidence rate without exposure to asbestos K_L = the dose-response factor (from Selikoff et al. 1979) f = level of exposure, fibers per milliliter (Column G above) $d(t-10)$ = duration of exposure from onset until 10 years before the current age (t) For mesothelioma, Nicholson postulated an absolute risk model: $I_M = K_M f[(t-10)^3 - (t-10-d)^3]$, $t > 10+d$ $= K_M f(t-10)^3$, $10+d > t > 10$ $= 0$ $10 > t$ where: I_M = annual excess risk of mesothelioma K_M = dose-response factor (from Selikoff et al. 1979) f = level of exposure, fibers/milliliter (Column G above) t = time since first exposure d = total duration of exposure (Column D above)
B	Sources of Exposure	Risk was calculated for each of nine categories of asbestos products manufactured between 1985 and 2000 on a product-by-product basis. Projections of the annual growth rates for these nine products were made and with the exception of friction materials all growth rates were predicted to be negative.

Figure 19. Asbestos: Mauskopf (1987)
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Key Assumptions and Comments on Data

Column	Data	Comments
C	Pollutant Concentration	The author used data on exposure levels, which are based on available data. While the author cites several studies on which the exposure assessment was based. No specific pollutant concentration values were presented in the article.
D	Exposure Duration/ Setting	The total number of person-years of exposure for each of the nine product categories were estimated using exposure data for 1983, the production indices, and the mean duration of exposure for each year of production. Five occupational and four nonoccupational exposure categories are included where relevant for each of the nine product categories. Mean levels of exposure were assumed to remain constant at the 1983 levels during the years 1985-2000 except for the consumer use category where they were assumed to vary with with production levels. For each product, the population at risk was subdivided into exposure categories according to when in the product life-cycle exposure takes place and whether they were exposed occupationally, in the ambient air, or in the use of the product. Each exposure category was also subdivided into 10-year age groups. Durations within exposure categories ranged from one to 30 years. Exposures prior to 1985 and after the lifetime of products produced within 1985-2000 were not included in this analysis.
E	Exposure	Exposure levels expressed as fibers per year were presented for each exposure category for each of the nine product categories which occur during: primary manufacture, secondary manufacture (for occupational only), installation, use and repair/disposal. The exposure levels reported are assumed to correspond to 1983 levels. The exposure levels reported range from 30 to 1560 million fibers per year from occupational exposures, and 3×10^{-5} to 2.79 million fibers per year for nonoccupational exposures. Average lifetime exposures expressed in million fibers per year were calculated as weighted averages over all product and exposure categories. Exposure levels, durations and populations were estimated based on 1983 levels presented in Table 1 and the methodology outlined in RTI (1985). This exposure level is a weighted average across exposure categories and time. The authors' analysis does not show exposure as constant across either variable. Therefore, these weighted averages cannot be used to back calculate either duratin or pollutant concentration.

Figure 19. Asbestos: Mauskopf (1987)
Page 3 of 5

Key Assumptions and Comments on Data

Column	Data	Comments
F	Dosimetry Factors	The unit of measure for dose in the risk model is fibers/milliliter of air inhaled. The risk models used were developed from occupational studies with typical exposures of 40 hours/week, 50 weeks/year and a breathing rate of 1 m^3/hour. Since data from the occupational exposure studies indicate that excess mortality from lung cancer and mesothelioma is proportional to both the level and duration of exposure to asbestos fibers, it is reasonable to use these dosimetry factors to normalize exposure to indoor air. 1 fiber/ml x 40 hr/wk x 50 wk/yr x 1 m^3/hr x 10^6 ml/m^3 = 2 x 10^9 fibers/yr.
G	Dose	This value represents the yearly lifetime average dose normalized to an occupational exposure setting. The total exposure in fibers per year is divided by the dosimetry factor of 2 x 10^9 ml/yr to obtain the normalized concentration, as if that exposure occurred during a typical work year of 40 hours per week, 50 weeks per year and a breathing rate of 1 m^3 per hour.
H	Response Factor	These response factors represent the average lifetime risks from asbestos exposures of an occupationally normalized exposure of 1 f/ml. These response factors were back calculated by dividing lifetime individual risk (Column I) by dose estimates Column G. For both lung cancer and mesothelioma, the occupational and non-occupational values were within the same order of magnitude. Differences exist between the occupational and nonoccupational values. For lung cancer, the risk factors are sensitive to exposure duration and concentration with nonoccupational nonoccupational exposures tending to be of longer duration and lower concentration, supporting a lower risk factor than the occupational setting. Mesothelioma is highly sensitive to age at exposure. The higher risk factor for the nonoccupational exposures is supported by the fact exposures would likely occur earlier in life on the average than for occupational exposures.

Figure 19. Asbestos: Mauskopf (1987)
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Key Assumptions and Comments on Data

Column	Data	Comments
I	Lifetime Individual Risk	Lifetime probability of lung cancer or mesothelioma from exposure to asbestos products manufactured between 1985 and 2000. Lifetime individual risks presented were those back calculated from excess cases and exposed population. The author reported lifetime individual risks in Table VI. There is an unexplained discrepancy between the values shown here and those reported by the author for occupational exposures. Values of 3.1×10^{-4} for lung cancer and 2.1×10^{-4} for mesothelioma were reported by the author. Values for nonoccupational were in agreement.
J	Exposed Population	Lung cancer rates are assumed to be age dependent and also to vary according to smoking habits, sex, race, and exposure duration and intensity, while mesothelioma incidence rates are assumed to be independent of age, sex, race, and smoking habits, but dependent on exposure duration and intensity and age at onset of exposure. 150 million for nonoccupational exposures represents the total exposed population. Individuals within this group may have been exposed to multiple sources.
K	Risk to Exposed Population	The total number of cases of lung cancer and mesothelioma, 1541, are predicted to be distributed over the next 100 years. The author presents projections of excess cancer and mesothelioma for each decade from 1995-2095 attributable to asbestos products manufactured between 1985 and 2000 for each exposure type.

Figure 19. Asbestos: Mauskopf (1987)
Page 5 of 5

population risks were reported in column K of Figures 4 through 19. All assumptions and caveats associated with these population risks are stated in the footnotes and comments of the referenced figures (Figures 4 through 19).

Figures 2 and 3 should be interpreted by considering both their uses and their limitations as follows:

- Lifetime risks to individuals as shown in Figure 2 are highly dependent on local exposure conditions and activity patterns. The results of broadly focused studies by others are intended for comparison purposes and do not cover the full range of all local exposure conditions. Despite efforts to equally compare the indoor pollutants, differences are bound to exist based on varying assumptions by different researchers.

- Data on nationwide risks as presented in Figure 3 are important for broad policy considerations but are not widely reported by researchers. Additional work needs to be done to expand beyond the relatively few reported studies and to perform sensitivity analyses to look at the range of outputs given various input assumptions.

- Figures 2 and 3 should be used in combination to draw conclusions. For example, the 5,000 to 20,000 estimated deaths due to radon (Figure 3) are greater than reported estimates for other pollutants, yet individual lifetime risks due to environmental tobacco smoke and some organic compounds can be very high as shown in Figure 2.

- For comparisons of the assumptions implicit in the summary data shown, readers are encouraged to follow the pathway leading to greater and greater detail. Summary data in Figures 2 and 3 come from column I and column K of Figures 4 through 19. These figures clearly show key measurements or assumptions in columns A through K. They are further described in comments below each risk characterization figure. When additional explanation is required, it is provided in the text near each figure. Finally, since no figure and brief analysis can cover all aspects of the complex studies analyzed, readers are encouraged to refer to the original references cited and listed at the end of this report.

REVIEW AND ANALYSIS OF STUDIES

The following sections present a detailed review of risk characterization studies for radon, ETS, volatile organics, formaldehyde, and asbestos. Each of the studies reviewed has a corresponding figure which presents the components of the risk characterizations. The discussion is organized by pollutant category.

RADON

There are numerous studies in the literature that report risk estimates associated with exposure to radon and radon decay products. Most of the risk estimates are based on epidemiological studies of underground miners. Miners are typically healthy males, working in an occupation that requires a much-higher-than-average amount of physical activity and breathing air containing a pollutant mix (airborne particulates, chemical vapors, and a relatively high concentration of radon and radon decay products) that is quite different from most work or home environments. As a result, the uncertainty associated with extrapolation from the dose-response factors typically derived for miners to a dose-response factor for the general population is high (Ellett and Nelson, 1985; Nazaroff and Nero, 1988).

The information presented in this section is centered around an attempt to compare risk estimates from three recent studies: the U.S. Environmental Protection Agency's *Radon Reference Manual* (1987c), the most recent report from the National Academy of Sciences' Committee on the Biological Effects of Ionizing Radiations (National Research Council, 1988), and the National Council on Radiation Protection and Measurement's Report 78 (NCRP, 1984). Although other risk estimates have been reviewed, these are presented as representative of the best available risk estimates. Some of the terms used in this discussion are defined in Appendix A.

U.S. Environmental Protection Agency (1987c)

Figure 4 presents a review of the characterization of risk attributable to exposure to radon and radon decay products. The derivation of EPA's risk estimates involves assumptions relating to four steps in the estimation process: (1) determination of radon decay product concentrations from radon concentrations; (2) estimation of cumulative radon decay

product exposure; (3) conversion of individual cumulative exposure to lifetime risk; and (4) projection of individual lifetime risks to the entire population. With regard to the relationship between radon and radon decay product concentration, it has been found that the equilibrium fraction (defined in Appendix A) ranges from 0.3 to 0.7, with an average of approximately 0.5. Using the average equilibrium fraction of 0.5, a ratio of 200 picocuries per liter of radon to 1 working level (WL) of decay products is considered to be fairly typical for residential environments. EPA used this ratio to convert radon concentrations to the average working level of 0.004 shown in column C of Figure 4.

The estimation of average lifetime dose of radon and radon decay products requires several assumptions in order to convert cumulative residential exposures to working level months (WLMs), defined as the integrated exposure a miner receives during 170 hours in a one-working-level (WL) environment. First, an adjustment factor for exposure duration is needed. EPA assumed that a resident is exposed to a given radon level 75 percent of the time (shown in Figure 4, column D), as opposed to 170 hours per month. Second, an adjustment factor for inhalation rate is required since, on average, the inhalation rate of a miner is considerable higher than the rate for the general population as a result of the miner's increased physical activity. EPA assumed the average breathing rate of an adult is 15.3 liters per minute while the rate for a miner is about 30 liters per minute. This ratio (15.3 to 30) was used by EPA to correct the estimate of cumulative residential exposure expressed in WLM (shown in Figure 4, column F). EPA's estimates of cumulative exposure do not account for potential variations in lung cell sensitivity or differences in sensitivity of different individuals.

Estimates of the risk of lung cancer associated with exposure to radon and radon decay products are obtained primarily from epidemiological studies of underground miners. Since miners in the study are still living, the risk observed during part of a lifetime must be extrapolated to a whole lifetime. EPA has adopted a relative risk model to estimate an increase in risk per WLM of one percent to four percent. EPA's calculations are based on an assumed linear dose-response relationship and a minimum latent period of 10 years.

EPA has projected lifetime risk to the entire population resulting in predictions of 5,000 to 20,000 deaths per year from radon exposures. The following equation was used:

$$\text{Total LCDs from Indoor Radon} = C_R \times T \times F_{WLM} \times R_{RRN} \times TCR \times POP$$

where:

C_R = average (mean) lifetime indoor radon decay product concentration
= 0.004 WL-life

T = average interval of lifetime exposure in hours, following a 10 year minimum induction period during which no lung cancer will be observed, assuming 75% occupancy and 73.88 year life span (1980 vital statistics)
= .75 × (73.88-10) × 365 × 24 = 419,691.6 hours/life

F_{WLM} = factor converting average cumulative indoor exposure in WL hours to working level months (WLM) for a miner (since risk estimates are based on miner data), accounting for 170 hours per month exposure period per WLM (by definition), and the difference in breathing rate between the average adult (15.3 liters per minute) and a miner (30 liters per minute)
= 1/170 × 15.3/30 = 0.003 WLM per hour

R_{RRM} = relative lung cancer risk for lifetime exposure to radon, per WLM, using relative risk model
= 1% to 4% per WLM

TCR = underlying annual average of U.S. lifetime lung cancer risk (1980 vital statistics)
= 4.584×10^{-4} per person.

POP = 1980 U.S. population
= 226,545,805

National Research Council (1988)

A relative-risk, time-since-exposure model is used by the National Research Council's Committee on the Effects of Ionizing Radiation (National Research Council, 1988) for computing an age-specific lung cancer mortality rate attributable to exposure to radon and radon decay products. Components, with appropriate conversion of units to fit the generic Risk Characterization Framework, are shown in Figure 5.

The National Research Council (1988) report uses the time-since-exposure (TSE) model to generate tables of gender-specific lifetime risks of lung cancer mortality by age exposure

started and age exposure ended and at annual exposures ranging from 0.10 WLM/year to 20.00 WLM/year.

The mathematical form of the TSE model is:

$$r(a) = r_o(a)\,[1+\beta\gamma(a)\,(W_1 + 0.5W_2)]$$

where:

$r(a)$ = age-specific lung cancer mortality rate from all causes

$r_o(a)$ = age-specific background lung cancer mortality rate from all causes other than radon

β = the slope of the dose-response relation

(a) = effect of age on risk (1.2 for ages <55, 1.0 for ages 55 to 64, and 0.4 for ages ≥ 65 years)

W_1 = WLM of exposure incurred between 5 and 15 years before current age

The steps used in applying the TSE model are:

1. Each year of the period of interest is placed in the appropriate interval:
   a. 5-15 years before current age,
   b. >15 years before current age;

2. The total annual risk for the person's age is calculated using the TSE risk equation shown above (using an appropriate background age-specific risk $r_o(a)$);

3. The calculated value of r(a) is multiplied by the chance of surviving all causes of death to that age (including the increased risk due to exposure).

The choice of an appropriate age-specific background rate is dependent on proper treatment of smoking as well as gender and calendar time.

National Research Council (1988) estimates the unit lifetime risk (response factor) of lung cancer mortality due to a lifetime exposure to radon progeny for males at 5.06×10^{-4} per WLM of exposure and for females at 1.86×10^{-4} per WLM of exposure. This estimate is based on a lifetime (69.7 years for males and 76.4 years for females) exposure to 0.1 WLM/year of radon decay products. Conversion of unit lifetime risk to average lifetime individual risk is shown in Figure 5, column I.

National Council on Radiation Protection and Measurements (1984)

The NCRP risk model relies on the Harley and Pasternack (1981) Model B of lung cancer excess due to radon progeny. Components, with appropriate conversion of units to fit the generic Risk Characterization Framework, are shown in Figure 6.

The mathematical form of the model is:

$$LR(t_o) = \Sigma_{t}^{85} A(t|t_o)$$

where:

$LR(t_o)$ = the lifetime risk from a single annual exposure at time t_o

$A(t|t_o)$ = attributable annual tumor rate at age t ($t \geq 40$) due to a single annual exposure at t_o. If exposure occurs after age 35, risk commences at t_o + 5 years, if exposure occurs before age 35, risk commences at age 40.

The equation used to calculate $A(t|t_o)$ is the following:

$$A(t|t_o) = RC\ (P_t/P_{t_o})\ e^{-\lambda(t-t_o)}$$

where:

RC = risk coefficient; assumed 10×10^{-4} cases/yr/WLM, which is an average value of all epidemiology studies reviewed

P_t/P_{t_o} = life-table correction to account for death from other causes

P_{t_o} = probability that an individual will be alive at age t_o.

P_t = probability that an individual will be alive at age t.

λ = decrease in rate of risk expression due to repair, cell death, or unspecified mechanisms.

The model includes the following assumptions:

1. Based on a review of epidemiologic data on underground miners, estimates of attributable risk range from 1.5 to 45 $\times$ 10^{-6} with a rounded average value of 10 $\times$ 10^{-6} cases per year per WLM of exposure. This average value was used in the calculations in the National Council on Radiation Protection and Measurements (1984) report.

2. Following a latent period, the tumor rate is an exponentially decreasing function of the time since exposure.

3. Disease rate excess associated with a single exposure increases with age at exposure.

4. Lung cancer is rare before the age of 40 years.

5. Median age of lung cancer among miners is about 60 years in nonsmokers and 50 years or older in smokers.

6. The minimal time for tumor growth, from initial cell transformation to clinical detection, is 5 years.

The model specifies a 5-year latency period for persons first exposed at age 35 or older and a (40 - u)-year latency period for persons under the age of 35 years. To obtain lifetime risk due to a single exposure at age u, the equation is integrated over t from age 40 to maximal assumed life (age 85 years). To obtain the excess risk at t due to all previous exposure, the equation is integrated over years of exposure, $u_1,\ldots,u_n$. To obtain lifetime excess risk from all exposures, the equation is integrated over t and u.

The NCRP model predicts 130 lung-cancer deaths per 10^6 persons per WLM of exposure. The estimate is based on an average lifespan of 70 years and a lifetime risk for lifetime exposure starting at age 1 of 9.1 $\times$ 10^{-3}. Conversions to average lifetime individual risk estimates are shown in Figure 6, column I.

ENVIRONMENTAL TOBACCO SMOKE (ETS)

A number of studies have been completed which estimate the annual number of deaths attributable to exposure to environmental tobacco smoke (ETS) and the lifetime individual risks attributed to such exposures. Six of these studies are reviewed using the Risk

Characterization Framework. These reviews are presented in Figures 7 through 12. Although different methodologies and source data are employed by each of the authors, most of the estimates for both lifetime individual risk and annual death attributable to exposure to ETS are similar. The predominant health effect that has been assessed is lung cancer. However, Fong (1982) includes lung cancer and emphysema together in his population risk estimates; Russell et al. (1986) include heart disease, bronchitis, emphysema, and lung cancer together in their risk estimates; and Wells (1988) provides separate risk estimates for other cancers and for heart disease.

Robins (1986)

The most mathematical analysis of risk attributable to exposure to ETS is Robins' (1986) appendix to the National Research Council's (1986) report entitled "Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects", which presents a sensitivity analysis approach to risk assessment that considers both the epidemiological data and some measures of exposure to the constituents of ETS. Study components are shown in the Risk Characterization Framework in Figure 7. Robins' estimates of the lifetime risk of lung cancer attributable to ETS in a nonsmoker with moderate ETS exposure are developed from an analysis that considers 30 different "exposure histories", three different estimates of the coefficients of the dose-response model, and two different estimates of the overall summary rate ratio (i.e., average relative risk) which is assumed to be the ratio of the "true" relative risk in exposed subjects to that in unexposed subjects. ("True" is used here to mean that this is, in fact, an assumed true value and not the mean of a sample.)

Two different average relative risk ratios are used in this analysis. A weighted average of the average relative risk ratios from 13 epidemiological studies presented in the National Research Council report is calculated to be roughly 1.3. In the analysis, the author assumes that a weighted average of 1.3 is causally related to differences in ETS exposure between "exposed" and "unexposed" individuals and not to bias. A second average relative risk ratio of 1.14 was calculated based on the subset of these epidemiological studies that were identified as U.S. studies. The assumption underlying the use of these two different average relative risk ratios is that the true average relative risk ratio is most likely 1.3 and at least 1.14.

In this risk assessment, the most important assumption was that the average relative risk was caused by ETS exposure. This assumption is basic to the equations used for estimating the true relative risk, the dose (expressed as the carcinogen-equivalent number of actively smoked cigarettes inhaled daily), and the subsequent estimates of lifetime risk of death from lung cancer. The following is a presentation of the general equations used by the author:

$$\gamma_{EXCESS}(t) = \gamma_0(t)\,[RR_{EXCESS}(t)]$$

where:

$_{EXCESS}(t)$ = the excess of lung cancer deaths at age t due to a specific exposure history

$\gamma_0(t)$ = the incidence of lung cancer death at age t in the absence of all exposure to ETS

$RR_{EXCESS}(t)$ = the excess relative risk for lung cancer due to a particular exposure history. Mathematically, it can be expressed in the following way:

$$RR_{EXCESS} = RR(t) - 1$$

where RR(t) is the true relative risk at age t for the exposed group compared to a completely unexposed group. The terms in this model are described mathematically by the author in his article (see National Research Council, 1986, pages 325-326).

One method the author employs in the analysis is to estimate the coefficients of a five-stage cancer process under the assumption that cigarette smoke influences the first and fourth stages of the process. The author further assumes that the ratio of the effect (on a multiplicative scale) on stage four to that of stage one is the same for ETS and mainstream smoke. Thus, an estimate of this ratio can be obtained by fitting a five-stage cancer model to data on the lung cancer experience of active smokers. The author calculates this ratio based on two different data sets and calculates a third ratio based on an adjustment to one of the data sets.

In addition to the two average relative risk ratios (i.e., 1.3 and 1.14) and the three different sets of values for the coefficients of the multistage model, the author developed

30 exposure histories for the exposed and unexposed individuals. The 30 exposure histories describe the percentage of time individuals in various age groups were exposed to various ETS dose rates (relative to the current ETS exposure of an adult nonsmoker without a smoking spouse). The author states that smaller differences postulated between the lifetime ETS exposure of "exposed" and "unexposed" individuals will be associated with larger estimates of the true relative risk. Thus, some exposure histories were selected that would modestly underestimate the true difference in exposure between the "exposed" and "unexposed" study subjects, and others were selected that would modestly overestimate this difference. Shown in Figure 7 are the maximum and minimum estimates from the range of lifetime risk estimates for males and females covering the 30 exposure histories and three response factors for the relative risk ratio of 1.3. Also presented by the author but not shown in this figure were the same calculations using a relative risk ratio of 1.14. The range of estimates of lifetime individual risk (shown in Figure 7, column I) for nonsmokers is based on three separate estimates of the coefficients in the multistage model and the range in exposure calculated for the 30 different exposure histories. Population risks expressed as the total number of lung cancer deaths per year among nonsmoking women (or men) (AN) attributable to ETS in 1985 were calculated by Robins (1986) using the following equation:

$$AN = \sum_{t} AF(t) \times I_o(t) \times N(t)$$

where:

$N(t)$ = the number of nonsmoking women (or men) at risk at age t in 1985

$I_o(t)$ = the age-specific death rate among nonsmoking women (or men) in 1985

$AF(t)$ = the age-specific fraction of lung cancer deaths due to ETS exposure in nonsmoking women (i.e., the average relative risk minus 1, divided by the age-specific relative risk)

In order to estimate age-specific relative risk among nonsmoking women (or men), age-specific estimates of the probability of being married to a smoker and the true relative risk in exposed and unexposed subjects are needed. The age-specific estimates of the probability of being exposed are taken from Garfinkel et al. (1985). Relative risk estimates were calculated in the same way as described for lifetime individual risk estimates.

Robins (1986) provides estimates of ETS attributable lung cancer deaths among U.S. nonsmokers in 1985 based on epidemiologic data, as shown in column K of Figure 7. Estimates are presented for females and males separately with a maximum and minimum across all exposure histories and five choices of dose-response coefficients. As shown in Figure 7, the maximum and minimum estimates calculated based on epidemiological data are 3,220 and 1,768 for females and 1,942 and 721 for males respectively.

Similar sensitivity analyses are presented by Robins (1986) for ex-smokers and continuing smokers for an alternative true relative risk ratio of 1.14 and for dosimetric measurements rather than epidemiological studies. For example, the author estimates the individual lifetime risk of lung cancer attributable to other people's cigarette smoke for an ex-smoker who smoked one pack per day from age 18 to 45 and was moderately exposed to other people's cigarette smoke lies between 5.2×10^{-3} and 2.03×10^{-2}. Note these risks are about 30 percent higher than the estimates shown in Figure 7, column I, for lifetime nonsmokers. Similarly, use of a relative risk ratio of 1.14 rather than 1.3 provides a range of estimates of annual lung cancer deaths for female nonsmokers of 935 to 1,730 and for male nonsmokers of 360 to 980. These estimates are approximately 50 percent lower than the estimates shown in Figure 7, column K.

Repace and Lowrey

The Repace and Lowrey (1985) estimates of annual risk of lung cancers attributable to exposure to ETS are based on estimates that U.S. nonsmokers are exposed to between 0 and 14 mg of tobacco tar per day with the typical nonsmoker exposed to 1.4 mg per day. A calculation based on age standardized differences in lung cancer mortality rates between Seventh-Day Adventists (SDAs) who never smoked and demographically comparable non-SDAs who never smoked (from studies by Phillips et al., 1980) yields a passive smoking risk rate of 7.4×10^{-5} per year. This was computed from an estimate that about 4,700 lung

cancer deaths per year were attributable to exposure to ETS among the 62.4 million U.S. nonsmokers. Results are shown in Figure 8. Note that data from the authors' reference are in columns I, J, K, and E. Column H was back-calculated in this work.

Seventh-Day Adventists have a lifestyle which avoids smoking and is oriented toward socialization with co-religionists. Moreover, 40 percent of the study group worked for church-run organizations; so the SDA lifestyle involves less exposure to ETS than demographically comparable nonsmokers from the general population.

The phenomenological exposure-response relationship derived by Repace and Lowrey (1985) was shown by them to predict to within 5 percent the lung cancer mortality rate and mortality ratio reported in the American Cancer Society cohort studied by Garfinkel (1981) in his study of passive smoking and lung cancer, as well as to explain the differences between the study of Garfinkel (1981) and the cohort studied by Hirayama (1981). Repace and Lowrey (1986), in a refinement of their estimates to adjust for sex and standardized to the total nonsmoking population, calculated a total of about 4,891 lung cancer deaths per year from passive smoking, of which 1,441 were estimated to be male and 3,450 female. These estimates are shown in parentheses in Figure 8, column K. These refined estimates are within 5 percent of their earlier estimates and are consistent with the results of a later case-control study by Garfinkel et al. (1985) to within 5 percent and to within 5 percent for the weighted lung cancer mortality ratios of the 13 epidemiologic studies of passive smoking and lung cancer as analyzed by the National Research Council report in 1986 (Repace and Lowrey, 1987).

The methodology for calculating these adjusted estimates of lung cancer deaths attributable to passive smoking is to start with the age-specific estimates for both sexes combined. These age-specific estimates are then separated into male and female estimates of lung cancer deaths, assuming 28.6 percent occur in men as presented in Arundel et al. (1986). The percent of over- or under-estimation for each sex in each age group is derived from Arundel et al. (1986). These percentages are used to adjust the estimates of lung cancer deaths for each sex in each age group. For three of the age groups (e.g., 35-44, 45-54, and 55-64) both male and female estimates of lung cancer deaths were adjusted downward while for the remaining two age groups (65-74 and 74+) estimates of lung cancer deaths for both sexes were adjusted upward.

Russell et al. (1986)

Urinary nicotine concentrations in smokers and nonsmokers are used to estimate exposure and risk to nonsmokers from ETS in Russell et al. (1986). This analysis is summarized in Figure 9. Urinary nicotine levels were chosen as a chemical marker for smoke intake because nicotine is specific to tobacco smoke, and urinary nicotine is stable and thus is suitable as a measure of tobacco smoke exposure over several hours. The levels easily differentiate "exposed" and "non-exposed" nonsmokers. The average concentration in nonsmokers who reported some exposure was 15.5 ng/ml, three times the value of 5.2 ng/ml for those who reported no recent exposure. The average urinary nicotine concentration of 188 nonsmokers from four studies was 10.8 ng/ml compared with 1,471 ng/ml in a combined sample of 229 smokers. On the basis of these measurements they estimated that nonsmokers receive on average about 0.7 percent of the nicotine dose of active smokers.

The authors assume that the dose-response effect at low levels is linear. This leads to a further assumption that a fair estimate of deaths due to passive smoking can be based on a proportion of deaths attributed to active smoking. In other words, the risk of death from passive smoking was assumed by the authors to be approximately 0.7 percent of that due to active smoking. The authors calculate the number of premature deaths per year among nonsmokers is estimated to be about 1,000 in Britain and over 4,000 in the U.S. as shown in column K of Figure 9.

Wigle et al. (1987)

Information on the proportion of people who had never smoked among victims of lung cancer in Canada is compiled in Wigle et al. (1987). This analysis is summarized in Figure 10. The total number of lung cancer deaths per year attributable to ETS in Canada was estimated to be 330. This number was derived by applying the age- and sex-specific rates of death from lung cancer attributable to ETS that were estimated by Repace and Lowrey to the Canadian population at risk as determined by the 1983 survey on smoking habits of Canadians. The U.S. population is approximately 10 times the population of Canada; therefore 3,300 deaths from lung cancer were estimated in the U.S. based on this study.

Wells (1986)

The epidemiological literature on ETS and adult lung cancer, other cancers, and heart disease is reviewed in Wells (1986). The primary model that was used by the author to estimate risk combined relative risks from the various studies that pertained to a given sex and disease and assumed that the combined relative risk was constant with age. A combined relative risk was calculated using the following equation:

$$R_{cb} = \exp \frac{W_{co} \ln R_{co} + W_{cc} \ln R_{cc}}{W_{co} + W_{cc}}$$

where:

R_{cb} = the combined relative risk

R_{co} = the relative risk for cohort studies

R_{cc} = the relative risk for case control studies

W_{co} = the weight for cohort study

W_{cc} = the weight for case control study

The weights for the cohort and case control studies are the inverses of the respective variances. The excess death rates attributable to lung cancer for never smokers for passive smoking (D_{px}) for each sex are shown in Figure 11, column K. These estimates were calculated for each sex and five-year age range from never smoker death rates (D_{ns}) by the following equation:

$$D_{px} = D_{ns} (R-1)/[F_p(R-1)+1]$$

where:

F_p = the fraction of the population that is exposed

R = the combined relative risk (R_{cb} from above)

Deaths were then calculated by multiplying the passive smoking excess death rate by the exposed population for each sex and five-year age interval, then summed. The sum for males and females is shown in Figure 11, column K.

For female lung cancer three cohort studies and 14 case control studies were combined. The overall combined relative risk is 1.44 with a 95 percent confidence limit of 1.3 to 1.7. The male lung cancer relative risk was based on a combination of two cohort studies and seven case control studies. The overall combined relative risk is 2.1 with a 95 percent confidence limit of 1.3 to 3.2.

In addition to lung cancer risk, the author estimates passive smoking related deaths for other cancers and for heart disease. By disease, the total consists of 700 lung cancer deaths, 11,000 other cancer deaths, and 32,000 deaths due to heart disease. For each million of the total population, the deaths by disease are 13 for lung cancer, 46 for other cancers, and 134 for heart disease.

Fong (1982)

The hazard of ambient cigarette smoke to nonsmokers is compared to the hazard of primary smoke to smokers in Fong (1982). Figure 12 provides a summary of his analysis. Fong assumed the nonsmoker worked in an office for 11 hours per day where 37 percent of the workers smoke and commuted on an unventilated bus 1 hour each day where 37 percent of the occupants smoke. The remaining 12 hours of each day was assumed to be unexposed. The ratio of the exposure of nonsmokers to the exposure of smokers is represented by the following equation:

$$R = r_1 \times r_2 \times r_3 \times r_4$$

where:

R = the ratio of exposure of a nonsmoker to a smoker

r_1 = the ratio of the density of secondary smoke to primary smoke

r_2 = the ratio of the times of exposure of the two cases

r_3 = the ratio of toxicities of the two types of smoke

r_4 = the ratio of the amounts of the ambient smoke (secondary plus side stream) a nonsmoker is exposed to and the primary smoke a smoker is exposed to

The maximum value of the ratio R calculated for this scenario is 1/13.

Since the effect of smoking in the U.S. has been stated as an excess of deaths of 350,000 per year, the maximum risk of ambient smoke to nonsmokers in the U.S. was estimated at 50,000 excess deaths per year (based on a smoker to nonsmoker ratio of 37:63). Fong (1982) assumes that 16 percent of the total deaths due to smoking are caused by lung cancer and 4 percent are due to emphysema. The remaining 80 percent are caused by heart disease and other cancers. The author notes, however, that other factors such as diet confound the effects of ETS on heart disease and cancers at other sites. He, therefore, assumes that only 20 percent of the risk of death from all causes attributed to ETS exposure as calculated by the above methodology is the realistic state-of-the-art estimate of the excess risk of death attributable to ETS. Thus the excess risk of death from ETS exposure is 1/60 that of primary smoke. This corresponds to an excess of 10,000 deaths per year.

VOLATILE ORGANICS

This section includes risk estimates of indoor exposure to volatile organic compounds other than formaldehyde, which is addressed separately in the next section. Three studies are included in this review: Tancrede et al. (1987), Wallace (1986), and McCann et al. (1986). EPA's Total Exposure Assessment Methodology (TEAM) Study provides indoor exposure concentration data used in all three studies. However, the first two studies use TEAM Study data exclusively to estimate exposure, whereas the McCann et al. (1986) study uses multiple literature sources as the basis for determining exposure concentrations to be used in the risk analyses.

A major difference among the three studies is the methodology employed to estimate carcinogenic potency. Wallace (1986) uses unit risk values developed by EPA's Carcinogen Assessment Group (CAG). As part of a sensitivity analysis, he compares these unit risk estimates with those developed by the "Harvard Group" which explicitly incorporates uncertainty into the estimate of potency. The potencies estimated by the Harvard Group can

be several orders of magnitude higher than the CAG estimates. Tancrede et al. (1987) use the Harvard Group methodology to estimate carcinogenic potency. Because this methodology is different from the methodology employed by CAG, Tancrede et al. estimate potencies for 52 chemicals, of which only nine have corresponding CAG values. McCann et al. (1986) provide a sensitivity analysis on carcinogenic potency for 24 chemicals, based on four methodologies. Nine of the chemicals have corresponding CAG values. Figures 13, 14, and 15 provide a detailed review of these three studies.

Tancrede et al. (1987)

This study by Tancrede et al. uses indoor air pollutant exposure data from the Total Exposure Assessment Methodology (TEAM) Study conducted in Bayonne and Elizabeth, NJ, and Los Angeles, CA, as well as data from a study by Lebret et al. (1984) in the Netherlands to predict cancer risk from exposure to 52 organic chemicals. Median estimates of cancer potency were calculated from human data (benzene) or animal bioassay data (20 chemicals) when such information was available. Cancer potency was estimated from toxicity and mutagenicity data and other information on biological activity for the remaining 31 chemicals using the analogical theoretical methodology developed by Fiering and Wilson (1983). For most of the 31 chemicals, studies of comparative toxicities and activities in promotion and co-carcinogenesis experiments of the chemicals and related compounds were used to estimate potencies. For a few of these chemicals, potencies were estimated from an inhalation or oral LD_{50} taken from the literature.

The review presented in Figure 13 focuses on Tancrede's risk estimates using exposure data from the Los Angeles, CA, TEAM study. These data are based on personal exposure monitoring of 200 people in the Los Angeles area during the winters of 1983 and 1984. Although not presented in Figure 13, the authors present a similar analysis based on the New Jersey TEAM study and the study by Lebret et al. (1984) of organic chemicals in four Dutch homes over a six-month period. Figure 13 also presents only those risk estimates for which the potency factor was based on human or animal bioassay data, and excludes all risk estimates for which the potency factor was based on the analogical theoretical methodology since this methodology is not in accord with the EPA Risk Assessment Guidelines (U.S. Environmental Protection Agency, 1987a). Tancrede et al.'s (1987) analysis of lifetime

median, mean, and 98th-percentile risk to residents of Los Angeles, CA, for individual organic chemicals is presented on separate framework pages in Figure 13.

The median risk estimate (pages 1 to 3 of Figure 13) is based on median exposure and potency estimates, without incorporating uncertainty, using the following equation:

$$\tilde{R} = 3 \times 10^{-4} \; \tilde{\beta} \; \tilde{d}$$

where:

$\tilde{R}$ = the median estimate of lifetime risk

$\tilde{\beta}$ = the median estimate of carcinogenic potency

$\tilde{d}$ = the median estimate of exposure dose

3×10^{-4} is a dosimetry factor which accounts for a breathing rate of the exposed individual of $20m^3$ per day; a 100 percent absorption rate; a 70 kg average body weight; and the conversion of micrograms to milligrams.

The mean risk estimates (pages 4 to 6 of Figure 13) incorporate uncertainty information into the risk calculation by using median estimates of the potency factor and dose along with an estimate of σ^2 in the following way:

$$\bar{R} = 3 \times 10^{-4} \; \tilde{\beta} \; \tilde{d} \exp(\sigma^2/2)$$

where:

$\tilde{\beta}$, $\tilde{d}$ and 3×10^{-4} are defined above.

The authors assume that potency and dose are lognormally distributed, and thus risk is assumed to be lognormally distributed with a variance σ^2 given by adding the variance of each of the following four factors:

$$\sigma^2 = \sigma_x^2 + \sigma_y^2 + \sigma_t^2 + \sigma_z^2$$

where:

σ = the standard deviation of the risk estimate

σ_x = the uncertainty in measuring potency in animal experiments

σ_y = the uncertainty in extrapolating from animals to humans, assumed to be $\log_e(4.5)=1.5$

σ_t = the uncertainty due to different routes of administration, is assumed to be $\log_e(5)=1.6$

σ_z = uncertainty (variability) in dose

This results in a mean risk estimate which is $\exp(\sigma^2/2)$ greater than the median estimate.

The 98th-percentile risk estimate (pages 7 to 8 of Figure 13) incorporates uncertainty into the risk calculation in a similar manner using the following equation:

$$R_{98} = 3 \times 10^{-4} \; \tilde{\beta} \; \tilde{d} \exp(2.0537\sigma) \; .$$

Thus, the 98th-percentile risk estimate is $\exp(2.0537\sigma)$ greater than the median estimate. A result of this methodology is that estimates with the most uncertainty (largest standard deviation) in the input parameters have the highest estimated risk.

Two key assumptions are made in this analysis. First, exposure is assumed to be continuous for a 70-year lifetime. This assumption is based on evidence that individuals spend 80 to 90 percent of their time indoors. However, the exposure data used in the analysis are from overnight personal air samples taken during the winter months in each of the three locations. Thus, a second assumption is that the overnight personal air sample data for the home are reasonably representative of air concentrations from all indoor micro-environments, over long time periods.

Tancrede et al. (1987) also provide estimates of the mean annual total risk for the mixture of contaminants measured in the TEAM Study at each of three locations: Bayonne, NJ; Elizabeth, NJ; and Los Angeles, CA. The basic assumption used to derive these estimates is that the risk from the mixture of organics presented in the analysis is the sum of the lifetime individual risk posed by each of the chemicals separately. Thus, risks are

assumed to be additive. These risk estimates are not presented in Figure 13 since they include many compounds other than those shown here.

Wallace (1986)

This study uses EPA's TEAM Study data to construct a rough nationwide risk assessment for the following six chemicals: benzene, chloroform (both air and water), carbon tetrachloride, trichloroethylene, tetrachloroethylene, and *para*-dichlorobenzene. The TEAM study measured personal air and drinking water exposures to these six chemicals and approximately 15 other chemicals for 600 residents of New Jersey, North Carolina, North Dakota, and California between 1981 and 1984. This sample represented a total population of ~700,000 in seven cities. The author cautions that although this is the largest personal exposure data set available, its use in providing rough estimates of population risks should be considered in light of the many and great uncertainties in these estimates.

The review presented in Figure 14 focuses on the author's estimates of individual lifetime risk and population risk for the six chemicals for both metropolitan and nonmetropolitan residents. The equation used by Wallace (1986) for calculating the risk of excess cancer incidence (y_i) for an individual from exposure to a carcinogen is the following:

$$y_i = a\, x_i$$

where:

a = potency of the carcinogen, in units of excess cases per unit concentration normalized to a 70 kg male with a 70-year lifetime (i.e., unit risk factor)

x_i = mean lifetime concentration

The equation used to estimate aggregate or population risk (Y) where the sample size is N and the sum of the weights equals a target population (P) is the following:

$$Y = Pa\,\overline{wx}$$

where:

a = potency as defined above

$\overline{wx}$ = the arithmetic mean of the population-weighted exposure distribution

The author notes that these equations ignore differences in individual susceptibility; the possible multistage nature of cancer; the different effects of early exposure versus late exposure; and the different actions of initiators, promoters, and carcinogens, among others.

The potency estimates used by the author for five of the chemicals were unit risk estimates [expressed as $(\mu g/m^3)^{-1}$] developed by EPA's Carcinogen Assessment Group (CAG). For benzene, the unit risk estimate is based on human epidemiological data and are a best (maximum likelihood) estimate. For tetra chloroethylene, trichloroethylene, chloroform, and carbon tetrachloride, the unit risk estimates are based on animal studies and are calculated from the 95-percent upper confidence limit on the potency factor. The unit risk estimate for *para*-dichlorobenzene is not a CAG estimate but was calculated by the author based on a National Toxicology Program mouse bioassay. For comparative purposes, the author also presents mean and 95-percent upper confidence limit potency estimates developed by a group at Harvard. Their methodology incorporates uncertainties of various types into the calculation. A large variance exists between the CAG 95-percent upper confidence limit estimates and the Harvard estimates.

The author calculates both lifetime individual risk and population risk for those living in U.S. metropolitan areas and nonmetropolitan areas. The author assumed that the TEAM Study data for New Jersey and California are representative exposures for 178×10^6 metropolitan residents, and the North Carolina and North Dakota data are representative exposures of 56×10^6 nonmetropolitan residents.

In addition to what is shown in Figure 14, the author presents similar estimates for outdoor exposures to these chemicals and comparative risk estimates using the potency factors developed by the Harvard group.

McCann et al. (1986)

The literature for indoor air concentrations for 140 organic compounds is reviewed in McCann et al. (1986). In Figure 15 the risk estimates calculated using both maximum and mean concentrations combined with the maximum likelihood (MLE) and 95-percent upper confidence level (UCL) unit risk factors are shown for eight carcinogens. These eight chemicals (plus formaldehyde shown in the next section) were included in Figure 15 because they are considered carcinogens by EPA and have unit risk factors estimated by EPA's Carcinogen Assessment Group. The MLE estimate is shown because in most cases it is the lowest or similar to the lowest unit risk estimate of the four. Similarly, the 95-percent UCL is similar to the highest unit risk estimate, in most cases. However, EPA unit risk estimates are the highest for several compounds including benzene, dichloromethane, dimethylnitrosoamine, and vinylidenechloride. Figure 15 gives the general range of risk estimates made by the authors.

The primary sources for the full list of compounds examined in this study were the published literature and presentations made at the 1984 Indoor Air Quality meetings in Sweden. Based on this literature review, the authors performed preliminary assessments of cancer risks for 24 chemicals. All concentration data presented by the authors are direct field measurements in homes and public buildings that the authors judged to reflect everyday exposure in normal, noncomplaint homes and offices. They did not include, for example, concentrations of formaldehyde in UFFI homes or concentrations measured in traditional occupational settings. When available, maximum and median or mean concentration measurements were recorded by the authors.

Four different approaches were employed to estimate unit risk values for the 24 chemicals. Maximum likelihood estimates and 95-percent upper confidence level values were calculated using the multistage model based on the dose-response data given by Gold et al. (1984, 1986). The authors do not state which animal study these estimates are based upon when multiple studies are reported for the same chemical. Risks were also calculated using the most potent TD_{50} estimated by Gold et al. (1984, 1986). The authors assumed this value is a point on a linear dose-response curve and have divided 0.5 by the TD_{50} (estimated as the equivalent dose in $\mu g/m^3$) to obtain the risk per unit dose. When Gold et al. (1984, 1986) reported that curves were nonlinear, the authors modified the risk estimates by using a less

than (<) or greater than (>) sign. Also, unit risk factors reported in EPA's Health Assessment Documents (U.S. Environmental Protection Agency, 1985a-f, 1986) were used. Nine of the 24 chemicals have EPA unit risk numbers and eight of these chemicals are shown in Figure 15; formaldehyde is shown separately in Figure 18.

FORMALDEHYDE

Interagency Regulatory Liaison Group (1981)

The Interagency Regulatory Liaison Group's Task Group on Formaldehyde (1981) published an extensive summary of studies on the exposure and resulting health effects, environmental effects, and health risks attributable to formaldehyde. A summary is presented in Figure 16. The report provides a detailed breakdown for exposure of subpopulations including four occupational categories, three consumer categories (e.g., residents of mobile homes, conventional homes), and ambient air and water. The risk estimates shown in Figure 16, column I have been calculated by the Interagency Regulatory Liaison Group (1981) with potency factors that were estimated from an animal bioassay. No scaling factors were used in estimating human potency; therefore, it is difficult to present an accurate relationship between columns G, H, and I in Figure 16.

Tancrede et al. (1987)

Tancrede et al. (1987) present median, mean, and 98th-percentile individual risk estimates attributable to exposure to formaldehyde in the average U.S. home. The authors assume formaldehyde exposure follows a lognormal distribution with a median indoor concentration of 0.05 ppm and a logarithmic standard deviation of 1.8. This is an assumed value in agreement with several studies for all U.S. houses. The authors use a carcinogenic potency factor derived by Zeise et al. (1984) of $\beta_a = 0.11$ $(mg/kg\text{-}day)^{-1}$ with a standard deviation of 0.18. The authors also assume the standard deviation of human response associated with this potency factor to be 1.5. This analysis is shown in Figure 17.

McCann et al. (1986)

Formaldehyde was included in the McCann et al. (1986) study on indoor air concentrations and preliminary carcinogenic risk assessment for 24 organic compounds. This study has been previously mentioned in the section on volatile organic compounds.

The authors used four different approaches to obtain unit risk values. Maximum likelihood estimates (MLE) and 95-percent upper confidence level values (UCL) were estimated from experimental results and tumor data used by Gold et al. (1984, 1986) to calculate the most potent TD_{50}. A response factor was also calculated from the TD_{50} value, which is assumed to be a point on a linear dose-response curve. The fourth method (EPA) was to use the unit risk factor reported in EPA Health Assessment Documents (Environmental Protection Agency, 1986). The unit risk factors were applied to both average and maximum concentrations reported in literature to obtain the eight lifetime risk estimates which are shown in Figure 18. Lifetime individual risk using maximum measured concentrations ranged from a high of 9.0×10^{-2} to a low of 3.1×10^{-3}. (Figure 18, page 1, column I). Corresponding values for the mean measured concentrations (page 3, column I) ranged from a high of 9.5×10^{-3} to a low of 3.7×10^{-6}. Due to the nonlinearity of the dose-response curve for formaldehyde, the potency factor estimates are also dependent on the concentration. Thus, corresponding values in column H on pages 1 and 3 of Figure 18 are not identical. At lower concentrations there is a nonlinear change in risk (column I) for MLE and UCL estimates. Linearity is assumed in EPA and TD_{50} potency estimates.

Additionally, the authors estimated the percentage of population at "high risk" from formaldehyde exposure. "High risk" was defined as lifetime individual risks of greater than one in 1000 (10^{-3}). Populations at risk were calculated based on geometric mean and geometric standard deviation of concentration data by DeBortoli et al. (1985). Those at high risk ranged from less than 0.01 percent of the total U.S. population for the MLE to greater than 99 percent for TD_{50}. These values have been included in Figure 18, column K, page 1, for estimates of carcinogenic risk for lifetime exposures to maximum measured concentrations. However, no attempt was made to quantify the number of excess cancers expected.

ASBESTOS

Several risk assessments for occupational exposures to asbestos have been performed (Hogan and Hoel, 1981; Nicholson et al., 1982; and Nicholson, 1983). These assessments focus on occupational exposures that have occurred in the past. The recent analysis by Mauskopf (1987), which is reviewed in this monograph, focuses on occupational and nonoccupational exposures to asbestos from products manufactured under current regulations for asbestos and asbestos products. This analysis draws on the work of Nicholson and others and was viewed as one of the few analyses addressing nonoccupational exposure. Risk characterization components are shown in Figure 19.

Mauskopf (1987)

This study presents an analysis of cancer risk attributable to exposure to asbestos from products manufactured during the years 1985 to 2000 for nine product categories (shown in column B of Figure 19). This analysis takes into account current government regulation of asbestos and projects cancer risk for the next 100 years attributable to exposure to products manufactured during the 16-year period, 1985 to 2000. Five occupational and four nonoccupational exposures identified in the lifecycle of each of the nine product categories are included in the analysis: occupational and nonoccupational exposure during primary manufacture, secondary manufacture (occupational only), installation, use, and repair/disposal.

Exposure data used by the author vary in duration from 1 to 30 years among the categories and therefore cannot be presented directly in Figure 19. A weighted yearly lifetime average exposure was calculated and is presented in Figure 19, column E in units of fibers per year. These exposure values must be converted, using the dosimetry factor in Figure 19, column F, to units applicable to the risk models used (fibers per ml), normalized for an occupational work exposure (Figure 19, column G). The risk models used were developed from occupational studies. The unit of measure for dose in the model is in fibers per ml over a typical work year. Since data from occupational exposure studies indicate that excess mortality is proportional to both the level and duration of exposure, it is reasonable to normalize indoor air exposure to an occupational equivalent. The total exposure in fibers per year were normalized to an occupationally equivalent concentration, as if that exposure

occurred during a typical work year of 40 hours per week, 50 weeks per year with a breathing rate of 1 m^3/hr.

Two health effects were evaluated, lung cancer and mesothelioma. The author used a life table risk assessment model which utilized actual data on the time, level, and duration of exposure and which provided estimates of individual lifetime risk (Figure 19, column I) and expected cancer cases (column K) among total exposed population (column J). Estimates were also generated for when in the future those cancer cases would occur and the mean ages of those persons contracting these diseases as a result of asbestos exposures. These types of estimates are not shown in Figure 19.

Cancer cure rates and annual death rates after diagnosis for the terminal patients were assumed to be constant across demographic groups, exposure categories, and products. Values estimated and used in the analysis were the following: cure rates of 8 percent and 2 percent for lung cancer and mesothelioma respectively, and, for those dying from the diseases, annual death rates after diagnosis of 81 percent and 71 percent for lung cancer and mesothelioma respectively. The following dose response factors from Selikoff et al. (1979) were used: $K_L = 1.0 \times 10^{-2}$ (fibers/milliliter)$^{-1}$ for the lung cancer relative risk model and $Km = 1.5 \times 10^{-8}$ for the mesothelioma absolute risk model. These are not the same as the response factors shown in Figure 19, column H. The calculated response factors shown in Figure 19, column H represent the average lifetime risk from asbestos exposure of an occupationally normalized exposure of 1 fiber/ml. These response factors were back-calculated by dividing the lifetime individual risk (Figure 19, column I) by the calculated yearly average lifetime dose (Figure 19, column G).

The author conducted a sensitivity analysis using alternative dose-response factor. Upper bound estimates were from Seidman et al. (1979) and obtained from a study of workers exposed less than a year. Seidman values were $K_L = 6.8 \times 10^{-2}$ and $K_m = 5.7 \times 10^{-8}$. Values of $K_L = 3.1 \times 10^{-3}$ (Hughes and Weif, 1980) and $K_m = 0.7 \times 10^{-9}$ (Peto, 1980) were used to obtain lower bound estimates. The author predicted a total of 1,541 cancer cases, and the sensitivity analysis revealed a range of 268 to 8,090 total cases.

CONCLUSIONS/RECOMMENDATIONS

CONCLUSIONS

- **Few complete quantitative risk characterizations appear in the literature.**
  While many scientific studies were found which deal with individual components of risk assessment, only a few studies combine exposure assessment data with dose-response evaluations to produce quantitative risk characterizations (as defined in the U.S. Environmental Protection Agency Risk Assessments Guidelines of 1986).

- **Most of the risk characterization studies which were found and reviewed focus on cancer risk.**
  For radon, formaldehyde, and asbestos, the cancer risk estimates are for lung cancer. The analysis of cancer risk from exposure to asbestos includes mesothelioma as a separate health effect in addition to lung cancer. The analysis of cancer risk from exposure to specific volatile organics does not specify any particular type of cancer. Three of the ETS analyses address diseases other than cancer including heart and lung diseases.

- **Few published risk characterizations are strong in all components of risk assessment.**
  Often a rigorous dose-response evaluation has been combined with a very cursory exposure assessment (or vice versa).

- **Pollutants may be ranked for comparison by individual lifetime risk or by risk to exposed populations.**
  The two approaches (Figures 2 and 3) give different results and are best analyzed in combination and with regard to weight-of-evidence evaluations. These summary figures provide useful insights into the relative significance of indoor air pollutants such as radon, asbestos, organic compounds, and environmental tobacco smoke.

- **A large quantity of data essential to future risk characterizations is emerging from the current work.**

  While few *completed* risk characterization studies were found, a large number of studies containing one or more essential components of risk characterization were identified.

- **The review of risk characterization studies and analyses presented in this work must be considered preliminary.**

  While considerable effort was expended to identify risk characterization studies (from EPA and RTI data bases, technical libraries, and computerized library resources), the authors cannot be sure that the universe of all relevant studies has been captured.

RECOMMENDATIONS

- **Continued work should focus on:**
  - **refining the systematic review and presentation of the complex components of risk assessment.**

    This should be done so that critical assumptions and data strengths and weaknesses can be further understood by more people. The Risk Characterization Framework (presented in Figure 1 and utilized in Figures 4 through 19) provides a start in that direction.

  - **combining existing data on exposure and dose-response relationships to make new risk estimates.**

    Such estimates should be based on a methodology and assumptions designed to provide risk estimates that are as comparable as possible between pollutants. A best currently available risk characterization for a particular pollutant could be developed by combining the most scientifically defensible information on hazard identification, exposure assessment, and dose-response relationships.

- **performing sensitivity analyses.**

  This would provide an understanding of the importance of input risk parameters to the resulting risk characterizations. Careful study of the uncertainties in the risk characterization process will lead to improved risk estimates and greater confidence in those estimates.

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APPENDIX A

GLOSSARY

Absolute risk projection model: a model which estimates the risk of exposure beyond the years of observation of a studied population by projecting the average observed number of excess cancers per unit dose into the future years of risk (U.S. Environmental Protection Agency, 1987).

Activity: the mean number of decays per unit time of a radioactive nuclide. Units: becquerel (Bq), curie (Ci) (National Research Council, 1988).

Alpha energy: the energy released when an alpha particle emitted during radioactive decay is halted by collision with a substance (e.g., lung tissue). The amount of energy depends on the velocity of the alpha particle, which in turn depends on the source of radioactive decay (e.g., decay of U-238 versus Ra-226) (U.S. Environmental Protection Agency, 1987).

Alpha particle: two neutrons and two protons bound as a single particle that is emitted from the nucleus of certain radioactive isotopes in the process of decay or disintegration (National Research Council, 1988).

Attached radon decay product: a radon decay product that is attached to a particle of dust or other material in air (U.S. Environmental Protection Agency, 1987).

Bequerel (Bq): the SI unit of radioactivity equal to one disintegration per second (U.S. Environmental Protection Agency, 1987).

Beta particle: a negatively charged subatomic particle (electron) emitted from a nucleus during some types of radioactive decay (U.S. Environmental Protection Agency, 1987).

Cohort: a large homogeneous group of people tested in epidemiological or socioeconomic studies. EPA's lung cancer estimates are based on calculations for a cohort of 100,000 people (U.S. Environmental Protection Agency, 1987).

Constant-relative-risk model: a risk model which assumes that, after a certain time, the ratio of the risk at a specific dose to the risk in the absence of the dose does not change with time (National Research Council, 1988).

Cumulative working level month (CWLM): a unit of cumulative radon exposure. The sum of lifetime exposure to radon working levels in total working level months (U.S. Environmental Protection Agency, 1987).

Curie (Ci): a unit of radioactivity, defined as that quantity of any radioactive nuclide which spontaneously undergoes 3.7×10^{10} disintegrations per second. One gram of radium-226 has an activity of one curie (U.S. Environmental Protection Agency, 1987).

Decay series: the consecutive members of a family of radioactive isotopes formed by sequential radioactive decay. A complete series commences with a long-lived parent such as U-238 and ends with a stable element such as Pb-206 (U.S. Environmental Protection Agency, 1987).

Electron volt (eV): a unit of energy = 1.6×10^{-12} ergs = 1.6×10^{-19} J; 1 eV is equivalent to the energy gained by an electron in passing through a potential difference of 1 V; 1 KeV = 1,000 eV; 1 MeV = 1,000,000 eV (National Research Council, 1988).

Equilibrium factor: an adjustment used in converting from picocuries per liter (pCi/L) to working-level concentration (WL), which takes into account the possible absence of radioactive equilibrium between radon and its decay products (U.S. Environmental Protection Agency, 1987).

Equilibrium fraction: ratio of the concentration of radon decay products to concentration of radon in the same sample of air at radioactive equilibrium. Typical measured values range from 0.3 to 0.7, with an average of about 0.5 (U.S. Environmental Protection Agency, 1987).

Gamma ray: short-wavelength electromagnetic radiation of nuclear origin (range of energy, 10 keV to 9 MeV) (National Research Council, 1988).

Ionizing radiation: subatomic particles or photons that have sufficient energy to produce ionization directly in their passage through a substance (U.S. Environmental Protection Agency, 1987).

Latent period: the minimum period of time between exposure and expression of the disease. After exposure to a dose of radiation, there is delay of several years (the latent period) before any cancers are seen (National Research Council, 1988).

Lifetime risk: the lifetime probability of contracting a specific disease (National Research Council, 1988).

Lifetime risk ratio: the ratio of the lifetime risk (R_e) of an exposed person to the lifetime risk of an unexposed person (R_o). This number minus 1 is the proportional increased risk associated with exposure (R_e/R_o - 1) (National Research Council, 1988).

Linear dose model: this model postulates that the excess risk is linearly proportional to the dose (National Research Council, 1988).

Linear energy transfer (LET): average amount of energy lost per unit track length (National Research Council, 1988).

Picocuries per liter (PCi/L): a unit of measurement of activity concentration. One picocurie per liter is equal to 10^{-12} curies per liter (U.S. Environmental Protection Agency, 1987).

Progeny: the decay products resulting after a series of radioactive decays. Progeny can also be radioactive, and the chain continues until a stable nuclide is formed. Radon progeny of primary concern to the public health community are 218-Po, 214-Pb, 214-Bi, and 214-Po (National Research Council, 1988).

Radiation dose: the total amount of ionizing radiation absorbed by material or tissues, in the sense of absorbed dose (expressed in rads), exposure (expressed in roentgens), or dose equivalent (expressed in rems) (U.S. Environmental Protection Agency, 1987).

Radioactive equilibrium: a state in which the rate of formation of atoms by decay of a parent radioactive isotope is equal to its rate of disintegration by radioactive decay, so that the activity of the parent and the decay product assume a constant proportion. This proportion is equal to one, if the parent has only one mode of radioactive decay. Because radon decay products tend to attach readily to surfaces, equilibrium between radon and its decay products is seldom reached. Using the average value of the equilibrium fraction (0.5), a ratio of about 200 pCi/L of radon to 1 WL of radon decay products is fairly typical for residences (U.S. Environmental Protection Agency, 1987).

Radioactive secular equilibrium: ideal steady state situation in which radon decay products would be formed and would decay at the same rate. Under such ideal conditions, the ratio of concentrations (or activities) of radon to decay products would be such that 100 pCi/L of radon and 1 WL of decay products would be present in the same sample of air. Secular equilibrium is never achieved because some of each of the decay products is removed from the air (due to attachment to walls, floors, etc.) before undergoing decay (U.S. Environmental Protection Agency, 1987; National Research Council, 1988).

Relative risk: the ratio of the rate of disease in exposed to unexposed populations (U.S. Environmental Protection Agency, 1987).

Relative risk projection model: a model which estimates the risk of exposure beyond the years of observation of a study's population by projecting the currently observed percentage increase in cancer risk per unit dose into the future years (U.S. Environmental Protection Agency, 1987).

Risk coefficient: the increase in the annual incidence or mortality rate per unit dose: (1) absolute risk coefficient is the observed, minus the expected number of cases per person-year at risk for a unit dose; (2) the relative-risk coefficient is the fractional increase in the baseline incidence or mortality rate for a unit dose (National Research Council, 1988).

Risk estimate: the number of cases (or deaths) that are projected to occur in a specified exposed population per unit dose for a defined exposure regime and expression period—for radon, the number of cases per person cumulative working-level month (National Research Council, 1988).

Synergistic model: a form of cancer causality whereby two or more carcinogens (for example, radon and tobacco smoke) act synergistically to cause cancer with a greater probability than if each were acting alone (U.S. Environmental Protection Agency, 1987).

Threshold hypothesis: the assumption that no radiation injury occurs below a specified dose (National Research Council, 1988).

Time-since-exposure (TSE) model: a model in which the relative or absolute risk is not constant but varies with the time after exposure (National Research Council, 1988).

Unattached radon decay product: a radon decay product that is not electrostatically attached to dust or particles in the air. Capable of attaching to lung tissue if inhaled (U.S. Environmental Protection Agency, 1987).

Working level (WL): any combination of short-lived radon progeny in 1 liter of air that will result in the ultimate emission 1.3×10^5 MeV of potential alpha energy. This number was chosen because it is approximately the alpha energy released from the decay of progeny in equilibrium with 100 picocuries of ^{222}Ra (National Research Council, 1988).

Working-level month (WLM): exposure resulting from inhalation of air with a concentration of 1 working level of radon progeny for 170 working hours (National Research Council, 1988).

APPENDIX B

Concentration Units and Conversion Factors for Radon and Radon Progeny

The quantity of radioactive species present in a given volume of air is typically specified by activity rather than by mass. The standard international (SI) unit for activity is the Becquerel (Bq), which is defined as 1 radioactive decay per second. The Curie (Ci) and picocurie (pCi) represent 3.7×10^{10} Bq and 0.037 Bq, respectively, and are often used in the U.S. to express quantity of radon.

Concentration of radon in air is typically expressed in Becquerels per cubic meter (Bq/m^3) or in picocuries per liter (pCi/L). Concentration of radon progeny (^{218}Po, ^{214}Pb, ^{214}Bi, and ^{214}Po) in air is typically expressed in working levels (WL). A working level is any combination of radon progeny which has the potential to release 1.3×10^5 mega-electron volts of alpha energy per liter of air (MeV/L). Concentrations of radon and radon progeny in indoor air are, of course, related, but the relationship is dependent upon a number of factors. Such factors include the rate at which progeny are removed from the air (by attachment to aerosols, dust particles, walls, ceilings, and floors). In indoor environments, one working level of progeny corresponds to about 150-300 pCi/L of radon, with 200 pCi/L representing about the average conversion factor for radon in homes.

Radon itself, because of its behavior as essentially a chemically inert gas with no affinity for attachment to other materials (including aerosols, dust particles, and lung tissue), does not appear to pose a serious threat to human health. Radon progeny, however, attach readily to other materials, including lung tissues. Because of this, epidemiological studies are usually concerned with exposure to radon progeny rather than with exposure to radon. Exposure to radon progeny is typically measured in working-level months (WLMs), where one WLM is equivalent to 170 hours of exposure to one WL of progeny.

TABLE OF CONVERSIONS

Description	Conversions
Activity: mean number of radioactive decays per unit time. Typically expressed in Becquerels (Bq), Curies (Ci), or picocuries (pCi).	1 Bq = 1 decay/sec 1 Ci = 3.7×10^{10} Bq 1 pCi = 0.037 Bq
Concentration: activity per unit volume. Typically expressed in Becquerels per cubic meter (Bq/m^3) or picocuries per liter (pCi/L) for radon and in working levels (WL) for radon progeny. A WL is defined in terms of potential alpha energy (from radon progeny) per liter of air. Some assumptions regarding relative concentrations of radon and progeny are required for conversions between pCi/L (or Bq/m^3) and WL. Progeny concentration may also be expressed in energy units of mega-electron volts per liter (MeV/L) or joules per cubic meter (J/m^3) with proper conversion of units.	1 pCi/L = 37 Bq/m^3 1 WL = 200 pCi/L (typically, in a home) 1 WL = 3.7×10^3 Bq/m^3 (at radioactive equil.) 1 WL = 1.3×10^5 MeV/L 1 WL = 2.1×10^{-5} J/m^3
Exposure: exposure concentration multiplied by the length of time exposed. Exposure to radon progeny is typically expressed in working-level months (WLM). A WLM is equivalent to exposure to a concentration of 1 WL for 170 hours.	1 WLM = 170 WL-h 1 WLM = 3.5×10^{-3} $J\text{-}h/m^3$

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