# PERSONAL EXPOSURE TO INDOOR AIR POLLUTION

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### INTRODUCTION

The term *personal exposure* refers to pollutant contact with an individual as he or she moves through various environmental settings, and is represented by the concentration at the boundary prior to ingestion, dermal uptake, and/or inhalation of that contaminant by the individual.

Accurate characterization of personal exposure is needed for valid assessment of health effects and the design of more effective intervention strategies. Misclassified exposures reduce the sensitivity of epidemiologic studies to detect the effects of pollutants or lead to spurious associations. For example, use of ambient air pollution levels to characterize exposures for residents of a community will not classify personal exposures accurately if there are indoor sources of the same pollutant and/or a large proportion of time is spent indoors. Further, exposure to pollutants of outdoor origin will be modified by infiltration and reaction indoors. In the context of an epidemiologic study, if these factors are randomly distributed across communities (i.e., exposure groups), then the estimate of the magnitude of the health effect might be underestimated (Shy, Kleinbaum, and Morgenstern 1978; Ozkaynak et al. 1986). However, if there are systematic differences in the distribution of indoor sources, or mitigating factors, then it is possible that positive or negative associations might be incorrectly attributed to the "assumed" exposure variable.

Personal exposure data might improve the cost effectiveness of control and mitigation strategies. If a personal exposure study indicates that the major portion of the total exposure is attributable to automobiles, one control strategy would be to restrict motor vehicle emissions, reducing exposures to those people in transit or pursuing activities near traffic. Restrictions on stack emissions from a local power #

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plant, although effective in reducing the total ambient pollutant burden of the community, might have little impact on total exposure to respirable particles inside residences. Investigations of personal exposures may also identify subgroups of the population whose particular behaviors would place them at risk for elevated exposures. Evaluation of activity patterns and exposures to specific sources may facilitate understanding of the determinants of the exposures and serve as a basis for intervention.

Several factors have contributed to the growing awareness of the importance of adequate estimation of personal exposure. The first is the development of new personal monitoring instrumentation, which is small and unobtrusive (Wallace and Ott 1982). The measurements using personal monitors have demonstrated clearly the inadequacies of assuming personal air pollution predicted by measurements made at outdoor sites, the usual approach for many community air pollutants. The error is particularly large for pollutants such as carbon monoxide (CO) and nitrogen dioxide (NO<sub>2</sub>), which are emitted from localized sources such as automobiles. kerosene space heaters, and gas cooking ranges (Akland et al. 1985; Quackenboss et al. 1986), but also has been demonstrated for more uniformly distributed regional pollutants such as ozone  $(O_3)$  and fine particulate matter, whose concentrations in indoor settings are mediated by building structures and surfaces (Spengler and Soczek 1984; Spengler et al. 1985; Contant et al. 1987). Second, the complexities of human behavior and movement may play a major role in determining personal exposure. Yet it has proven difficult to develop mathematical models for estimating individual exposure based on outdoor fixed-site or area measurements. Further, even for modeling population exposures, there is a lack of population-based data on activity patterns suitable for exposure risk analysis (World Health Organization 1982; Ott 1985; Sexton and Ryan 1988). This chapter develops a conceptual framework for exposure assessment in the indoor setting. The current monitoring methods are reviewed as they relate to strategies for personal exposure assessment, and exemplary applications are described. A more detailed treatment of air pollution measurement is provided in Chapter 4.

### CONCEPTS AND DEFINITIONS

Figure 5.1 presents a conceptual framework for understanding personal exposure within the sequence of events between the emission of a pollutant from its source and the health effect experienced by a person who comes into contact with that pollutant. After release of a pollutant at a source, the pollutant moves through an environment in which it may be diluted and transformed by physical and chemical processes. As illustrated in the third component of the sequence, some of the pollutant (or the product of a transformation) eventually comes into contact with people, resulting in an "exposure." The link between the presence of a chemical contaminant in the environment and its contact with people is complex and in part determined by patterns of human behavior. The portion of exposure which is adsorbed, ingested, or inhaled into the body is termed the *dose*. It is this final





amount of the chemical contaminant which produces the health effect. In the following sections, the terms *concentration*, *exposure*, and *dose* are more fully defined.

### CONCENTRATION

The amount of a chemical contaminant at a particular location in a particular medium is termed the *concentration*. The concentration of an air pollutant is the amount of the material contained in a specified volume of air. Most air pollutant concentrations are expressed in mass per volume units (e.g.,  $\mu g/m^3$ ); however, gaseous pollutants may also be presented in units of a mixing ratio with air, typically in parts per million by volume (i.e.,  $ppm_v$ ). For certain particulate contaminants such as asbestos, the actual number of particles per unit volume is used (i.e., number count/m<sup>3</sup>).

### EXPOSURE

*Exposure* is defined as the contact of pollutant with a susceptible surface of the human body (Duan 1982; Ott 1985). For most air pollutants, this is the contact of pollutant with the skin, eyes, tissue in the nose, mouth, or throat, or the epithelium of the respiratory tract, the lining of the airways and alveoli. Thus, exposure can be simply defined as the simultaneous presence of a person and a pollutant in his or her immediate environment.

Exposure normally is considered to include within its definition an element of time. For example, exposures are typically given units of concentration multiplied by time (e.g.,  $\mu g/m^3$ -h), connoting an equivalent exposure experienced by an individual subject to a fixed concentration for a period of time. This allows exposures to be placed on a scale and quantified. One may see from this that a complete description of exposure requires knowledge of three components: magnitude of pollutant concentration in the exposure environment; duration of the exposure; and the time pattern of the exposure. The first two components require little further explanation. The pattern of exposure is of importance because of possible differences in the effects of varying concentrations relative to fixed values. Further discussion can be found below.

Several commonly used means of characterizing exposure are presented in



Figure 5.2. Graph A depicts the five-minute mean NO2 concentrations (parts per billion or ppb) measured by a continuous monitor worn by an individual over a twenty-four-hour period. Some periods of the exposure profile are characterized by highly variable exposures to peak levels as high as 200 ppb, whereas other periods are characterized by fairly constant low levels of exposure. These exposures may be compartmentalized by averaging the concentrations within the time period of a specific activity. As graph B illustrates, an individual moves through several diverse exposure settings in the course of a day. Graph C shows the cumulative integrated exposure as the individual moves during the day. The rate of increase in integrated exposure is greater for certain exposure settings, such as cooking meals on a gas range. Note that the twenty-four-hour integrated exposure for this individual is 960 ppb-h. Graph D presents average exposure measurements for various lengths of averaging times. The longer averaging times effectively dampen the variation in personal exposure. Although the twenty-four-hour mean exposure was 40 ppb, mean exposure during the six-hour interval comprised of night sleep was 25 ppb, and the three-hour interval comprising the evening commute and meal preparation was 65 ppb In this particular example of the different ways of averaging personal exposure, the biologically relevant measure of exposure is not known. Transient exposures to peak levels of NO<sub>2</sub> and/or long-term chronic exposures may be associated with oxidant damage and increased susceptibility to respiratory infection.

### DOSE

Dose refers to that amount of chemical contaminant which crosses a boundary of the body and reaches the site of toxic action. Time is implied in the concept because dose is typically expressed as mass or number of molecules. Dose, therefore, varies not only with the exposure profile (i.e., concentration and time course) but also with the physiologic state of the individual. For example, consider two individuals who are indoors at home. One sits in a chair and watches television, and the other rides an exercycle for one-half hour while also watching television. Although both individuals are equally exposed to radon present in the room air, the physically active person who is breathing faster, more deeply, and through the mouth receives a greater pulmonary dose relative to the person at rest.

If the site of toxic action is the lung epithelium, as for ozone, the amount of pollutant deposited on the lung epithelium is equivalent to the dose. If the pollutant is absorbed across the lung epithelium (see Chapter 8 on CO) into the blood, where it is transported to the target organ, the amount absorbed is the dose to the body, while the pollutant reaching the site of action is considered to be the biologically effective dose.

For particulate matter and water soluble gases, the route of breathing will affect the amount of chemical contaminant that reaches the lung. During nasal breathing, particles with an aerodynamic diameter of  $2-5 \,\mu\text{m}$  are more likely to be filtered out in the nasal turbinates by impaction and adsorption onto-mucus whereas particles of a smaller diameter pass through the nasal passageways of the head and on to the

Figure 5.2. Examples of different ways to characterize an individual's personal NO<sub>2</sub> exposure profile. *Source:* Adapted from Sexton and Ryan (1988), with permission.

lower airways and alveoli where they may be deposited (Schlesinger 1988). Removal in the nasal passages is bypassed during mouth breathing. For nonreactive gases such as CO, the route of breathing does not affect the delivery of nonreactive gases to the deep lung. Therefore, for certain pollutants, estimates of pulmonary dose should consider ventilation and the route of inhalation along with the physical and chemical characteristics of the contaminant. Direct monitoring of breathing rates or level of physical exertion may be used to make a crude correction for oral breathing.

The definition of *biologically effective dose* can be refined further. Some inhaled contaminants undergo chemical transformation, and it is the metabolic products that are actually responsible for the toxic effect. Different metabolites may be formed depending upon the received dose, the rate of dosing, and the physiologic conditions. Hence, the effective biologic dose may be a fraction of the pollutant initially inhaled.

### TIME-ACTIVITY PATTERNS

People encounter different concentrations in different settings, and depending upon source use and ventilation, among other factors, the concentrations in these settings will change over time. It may be important, therefore, to understand the patterns of human behavior relevant to exposures. Thus, an understanding of the settings and activities in which people spend their time could identify populations and/or behaviors at risk of high exposure. Such studies may reveal effective exposure mitigation opportunities, while providing the basis for modeling exposures which incorporate data from fixed location microenvironmental monitoring. For example, human behavior related to source use, such as the use of an exhaust fan while cooking, the use of a gas range for space heating, the substitution of microwave ovens for gas ranges will result in differential exposures for subgroups.

### MODELING PERSONAL EXPOSURE

Personal exposure may be modeled by considering a series of locations with air pollutant concentrations present. A person moves through these locations over time. A given location could be subdivided if activities, ventilation, or mixing cause changes in source use, strength, or dilution. In the generalized model (Ott 1985; Duan 1982; Fugas 1986), the mean concentrations experienced in successive settings, or *microenvironments*, are time weighted and summed to generate a total integrated exposure for some specified time interval, usually a twenty-four-hour period:

 $E = \Sigma f_i C_i$ 

Where,

 $\Sigma =$  Sum over all times and concentrations

- E = Total exposure
- $C_i$  = Concentration of pollutant in microenvironment *i*
- $f_i$  = Fractional time spent in microenvironment *i*

When this model is applied to an individual's daily exposure profile, the total exposure is identical to the twenty-four-hour integrated exposure, or the cumulative exposure described in Figure 5.2, graph C. Microenvironments are specific situations of exposure, and as defined by Duan (1982), they are locations in space and time over which pollutant concentrations are assumed uniform and constant. Therefore, a kitchen location with cooking activity on a gas range is a microenvironment that is different and distinct from the same location before cooking began. Levels of pollutant concentration at certain locations (e.g., kitchen, garage, or traveling inside a car) may display high temporal variability, and therefore the choice of classification of the microenvironment, and hence averaging time, will influence the variability of the exposure measure. Quackenboss et al. (1986) suggested that although some variability in exposures may be lost by combining microenvironments into broad classes, the differences in variability within a class are likely to be smaller than those between microenvironmental classes (e.g., between indoor and outdoor locations or between residences and workplace). For certain pollutants, such as CO, continuous monitoring or a high resolution microenvironmental classification may be needed to characterize exposure adequately for accurate estimates of uptake by the body due to the timeexposure relationship for carboxyhemoglobin (COHb). In comparison, longer averaging times and more coarse characterization of microenvironments may be appropriate for pollutants such as lead, where body burden is the measure of interest.

The generalized model of total personal exposure may be applied to a specific group of people, or a *community*. The distribution of individual personal exposures made on a sample is combined with time-activity data on the population to estimate the distribution of total exposures for the population. The upper tail of the distribution for some pollutant exposures may identify a subgroup of the population with higher-than-average risk. This is of particular interest to decisionmakers concerned with public health. It should be recognized that this area of the distribution is somewhat more difficult to characterize than is the mean (Sexton and Ryan 1988), and many personal exposure measurements must be obtained to estimate accurately the frequency, magnitude, and duration of high-exposure events, which may be relatively rare. Examples of relatively lower frequency situations include faulty auto exhaust systems that result in high in-vehicle CO concentrations and the improper venting of furnaces that then leak emissions into residences.

# ASSESSMENT OF PERSONAL EXPOSURE

Techniques for the assessment of personal exposure to air pollution can be divided into two major classes. The first approach measures the concentrations of the pollutant using monitors worn on the person or located in specific settings frequented by the person (i.e., home, workplace, or car), and the second estimates exposures from measurements of biologic markers such as the pollutant concentrations in blood and breath samples (Sexton and Ryan 1988).

#### AIR POLLUTANT MONITORING

In his review of total exposure assessment, Ott (1985) separated exposure measurement into two general methodologies, direct and indirect.

*Direct Method* In this approach, individuals wear personal monitors that measure the concentrations of pollutants in their breathing zone. While wearing the monitor, the subject maintains a diary record of locations visited and activities pursued. A variety of passive sampling devices that can provide integrated measurements of personal exposure is available, and continuous monitoring instrumentation with data-logging capacity continues to evolve (Wallace and Ott 1982; see Chapter 4 on environmental monitoring). However, implementing the direct method is labor intensive and time consuming, which may preclude its application to large samples, and personal monitors are not presently available for all pollutants of concern.

The direct method of personal exposure assessment has been applied in several surveys. The Environmental Protection Agency (EPA) obtained personal CO measurements on large probability samples of the residents of metropolitan Denver and Washington, D.C. (Akland et al. 1985; Wallace et al. 1988), which allowed the evaluation of the efficacy of outdoor monitoring networks to estimate actual population exposures. Personal exposures to CO have also been measured in a subgroup of Los Angeles men who have ischemic heart disease (Lambert 1990). The exposure patterns of these susceptible individuals were comparable to those measured in the general population at Denver and Washington, D.C. Nitrogen dioxide exposure has been characterized by direct monitoring carried out in conjunction with the Harvard six cities study (Quackenboss et al. 1986) and in probability samples of residents in Boston and Los Angeles (Ryan et al. 1989; Spengler et al. forthcoming). In the Total Exposure Assessment Methodology (TEAM) studies, the EPA surveyed personal exposure to various species including CO, volatile organic compounds (VOCs), pesticides, and particulate matter in several U.S. metropolitan areas (Wallace 1987; see also Chapter 11). The methodology of the EPA's carbon monoxide and VOC studies will be presented in detail in a later section of this chapter.

*Indirect Method* This method avoids the practical and logistic constraints of direct monitoring. The indirect approach uses area or microenvironment monitors and time-activity data to estimate personal exposures. Ideally, a mathematical model relating personal exposure to area measurements and behavioral parameters should be developed and validated prior to the implementation of a large-scale monitoring program.

The indirect method has been applied to estimate the ozone exposure of asthmatics residing in Houston (Contant et al. 1987). This study will be discussed later under "Applications of Personal Exposure Monitoring Techniques." A simplified application of this method has also been used to study the exposures of infants to  $NO_2$  in residences in Albuquerque, New Mexico (Harlos et al. 1987). Mothers reported the time-activity patterns of their children inside the residence, and total personal exposure to  $NO_2$  was weighed according to the time that the child spent in the particular rooms in which the samplers were located (Table 5.1). The timeweighted estimate of personal exposure agreed closely (R = .81) with measurements made by a sampler worn on the child. This result supported the choice of area monitors for a larger scale study that will longitudinally measure the child's exposure from birth to age 18 months.

### **BIOLOGIC MONITORING**

In performing a biologic assessment of exposure, samples of sputum, urine, blood, or expired breath are obtained and analyzed for the presence of the pollutant or its metabolite. Biologic monitoring is particularly useful if highly sensitive and specific markers of exposure are available, and it may be considered an indirect method of exposure assessment. Good markers of exposure are available for CO (Coburn, Forster, and Kane 1965; Joumard et al. 1981; Lambert, Colome, and Wojciechowski 1988), lead (Annest 1983; Billick 1983), and the nicotine component of environmental tobacco smoke (U.S. Department of Health and Human Services 1986). Biologic monitoring may be a more relevant measure than ambient concentrations for defining populations at risk or for conducting health effects research. However, although providing a surrogate measure of dose and an integrated measure of exposure, relating the biologic marker's level to personal exposure is often problematic for some contaminants due to the complex metabolic pathways involved and the variability in physiologic parameters affecting uptake

### Table 5.1 Time-Weighted Contribution of Exposures to NO<sub>2</sub> in Several Residential Locations to Total (Twenty-Four-Hour) Exposures

Location	Timea		Mean NO <sub>2</sub> <sup>b</sup>		Exposure Contribution	
	Hours	S.D.	ррь	S.D.	ppb-hour	Percent Total
Bedroom	14.1	6.2	42.9	15.3	604.9	61.4
Living room	6.3	4.5	50.2	22.4	316.3	32.1
Kitchen	0.78	0.73	65.5	31.5	51.1	5.2
Outdoor	0.22	0.32	12.2	8.6	2.7	0.3
Travel	0.80	0.98	12.2	8.6	9.8	13
Total	24.0		10000000	(7.5 M)	948.8	1.5

Estimated average infant exposure = 41 ppb

Source: Harlos et al. (1987), reprinted with permission.

"Time-location for all forty-six infants.

\*NO2 levels for twenty homes with complete data.

Outdoor and travel levels are the seven-day average outdoor NO2 values for the twenty homes.

and elimination (Wallace et al. 1988). Nevertheless, it must be recognized that environmental controls and mitigation strategies will be predicated upon reducing concentrations, and perhaps exposures. It is these more conventional measurements that lend themselves to the precise definitions necessary for enforcement. On the other hand, reduction of a biological marker like blood lead or COHb provides useful trends data and displays the effectiveness of source reduction.

# TIME-ACTIVITY MONITORING

Human beings are not stationary, and the environments people inhabit may support several kinds of activities. Therefore, accurate estimates of exposure require assessments of the movements of people and the activities undertaken at various locations. Sociologists and geographers have collected information on activities and movement using self-administered diaries and recall interviews (Robinson 1988). With the diary method, subjects record activities and locations as they engage in activities through the day (Figure 5.3). If faithfully performed, this method can provide information with fine time resolution and good reliability (Michelson 1985). An alternative approach is the twenty-four-hour recall interview in which the respondent recalls the activities and locations of the preceding day within a structured line of questioning by an interviewer. Although providing a record of activities at a more coarse level of time than the diary approach, the interviewing process is generally regarded to produce a more complete and logical

TIME BEGAN	WHAT WERE YOU DOING? (ANYTHING ELSE AT THE SAME TIME?)	WHERE WERE YOU? (ROOM IN HOUSE, OR NEAREST INTERSECTION.)	WERE YOU NEAR ANY OF THESE ACTIVITIES? CHECK (1/).	L 0 T:C
			<ul> <li>( ) Running Autos</li> <li>( ) Ges stove/oven</li> <li>( ) Tobacco smoking</li> <li>( ) Voodburning</li> <li>( ) Running engines</li> </ul>	
			<ul> <li>( ) Running autos</li> <li>( ) Gas stove/oven</li> <li>( ) Tobacco smoking</li> <li>( ) Woodburning</li> <li>( ) Running engines</li> </ul>	
			<ul> <li>( ) Running autos</li> <li>( ) Gas stove/oven</li> <li>( ) Tobacco smoking</li> <li>( ) woodburning</li> <li>( ) Running engine</li> </ul>	

Figure 5.3. Example of a page from a time-activity diary to monitor personal activity while wearing an air pollution monitor.

sequence of information (Michelson 1985). Standard formats for diaries and interviews have been described (Michelson 1985; Robinson 1988).

## APPLICATIONS OF PERSONAL EXPOSURE MONITORING TECHNIQUES

In this section, several study designs are presented to illustrate some specific methodologic aspects of the measurement of personal exposure. The first study is the Denver-Washington, D.C., CO study conducted by the EPA. This study represents the first large-scale application of the direct and indirect methods of population exposure assessment and creatively uses direct exposure measurement and biologic markers to characterize exposure. The second study presented uses the indirect method to estimate personal exposure to ozone for asthmatics living in Houston. The third study considers the measurement and modeling of personal exposure to nitrogen dioxide in Boston and Los Angeles. The fourth study is also one of the TEAM studies, conducted by the EPA to characterize population exposures. This chapter briefly focuses on the TEAM study of exposures to VOCs, although the EPA has conducted other exposure studies on pesticides and particulates that utilize the TEAM concepts.

### CARBON MONOXIDE

During the winters of 1982 and 1983, the EPA measured personal exposures to CO in statistically representative samples of the Denver and Washington, D.C., metropolitan areas (Akland et al. 1985). The goal of the research program was to generalize the direct measurement of personal exposures to the entire adult nonsmoking population residing in these areas. The sampling scheme was stratified and included disproportionately large numbers of individuals who commuted and who lived in residences with gas-fueled appliances or an attached garage. In each urban area, five hundred individuals were monitored; subjects wore a portable, continuously recording instrument and maintained a time-activity diary for one day in Washington, D.C., and two days in Denver. End-expired breath samples were collected from subjects at the end of each twenty-four-hour monitoring period to estimate blood COHb levels. The population estimates of personal exposure were derived from adjusted sampling weights. The results indicated that more than 10 percent of the personal exposures of residents of Denver, and 4 percent of the Washington, D.C., residents exceeded the eight-hour 9-ppm federal standard. Ambient fixed site monitoring data underestimated the distribution of these personal exposures (Figure 5.4). The exposures experienced in transit and outdoors near active roadways were identified as important contributors to total CO exposure. The observation that people spent more than one hour per day in transit and more than twenty-two hours per day indoors is important. The mean levels of CO measured in specific indoor microenvironments are presented in Chapter 9.

The breath samples were used to provide an additional measure of exposure for the Washington, D.C., sample (Wallace et al. 1988). Carbon monoxide levels in end-expired breath were used to estimate blood COHb concentration, a measure of



Figure 5.4. Frequency distribution of maximum eight-hour CO personal exposures and ambient concentrations for population samples in Denver, Colorado, and Washington, D.C., during the winters of 1982 and 1983. *Source:* Reprinted with permission from Akland G.G., et al. Measuring human exposure to carbon monoxide in Washington, D.C., and Denver, Colorado, during winter 1982–1983. *Environ Sci Technology* 19:911–18. Copyright 1985 American Chemical Society. the cumulative exposure to CO. Exposure measurements from the continuous monitors were input into the pharmacokinetic model to calculate COHb levels at the end of the twenty-four-hour monitoring period (Coburn, Forster, and Kane 1965). The modeled COHb levels were 40–50 percent lower than those estimated from samples of end-expired breath. The availability of this alternative measure of exposure prompted the investigators to reevaluate the accuracy of the electronic monitors. Differences in the sensitivity of monitoring instrumentation, declining sensitivity with battery discharge, and improper calibration methods may have biased the measurements low in some monitors. Therefore, the investigators used the breath measurements to calculate individual correction factors to revise upwardly the distribution of personal exposures. Without the biologic marker data, the monitoring instrumentation would have underestimated the sample's exposures.

The efficacy of outdoor monitoring networks to estimate personal exposure was tested using the data derived from the Denver field survey. The exposure profiles were used to validate a population exposure model, the simulation of human activity and air pollution exposure (SHAPE) model (Ott 1988). Two days of personal monitoring data were available for each of 336 individuals living in Denver. The distributions of microenvironmental exposures and the ambient monitoring network data from the first day of monitoring of each individual were used to predict the personal exposures on the second day. The distribution of microenvironmental concentrations on the second day were calculated by adding microenvironmental source inputs onto the ambient background concentration measured on the second day. Using the actual time-activity data from the second day of monitoring, exposures in microenvironments were assigned by Monte Carlo sampling from the microenvironmental CO distributions. SHAPE was successful at predicting the mean of the cumulative distribution, but it tended to underestimate exposures in the tails of the distribution (Figure 5.5). Of particular concern was the underestimation of high exposures.

### OZONE

Ideally, exposure models are constructed and validated with actual personal exposure data. It is not always necessary to perform this validation on a sample the size of that used in the EPA CO studies. For example, the  $O_3$  exposure model developed by the University of Texas School of Public Health was validated in a community sample of relatively small size (Stock et al. 1985; Contant et al. 1987). Data to construct the model were obtained from twelve homes of asthmatics by measuring indoor and outdoor residential levels of  $O_3$  with a mobile monitoring van and by measuring personal exposure with portable instruments carried by a field technician who followed the research subject. Fixed-site monitoring data were regressed on the indoor and outdoor residential measurements to define the relationship between levels of  $O_3$  from the ambient monitoring network and the concentrations occurring at the residential sites. Hourly averages of  $O_3$  concentration at indoor and outdoor residential sites were computed. The exposures of each individual were weighted according to records of personal activity maintained by



# CUMULATIVE FREQUENCY, %

Figure 5.5. Logarithmic probability plot of cumulative frequency distributions of the maximum moving average eight-hour personal exposure to CO predicted by SHAPE using the ambient background concentration calculated from a composite measure from all fixed-site stations along with observed frequency distribution of the measured personal exposures for day 2 in Denver, Colorado. *Source:* Adapted from Ott et al. (1988), with permission.

the subjects. When compared with the actual hourly measurements of personal exposure, the model underestimated exposure by approximately 20 percent (Figure 5.6). However, use of the model to estimate personal exposure is considerably more accurate than using untransformed fixed-site measurements, for the indoor concentrations of  $O_3$  were, on average, substantially less (<10%) than levels simultaneously measured at the nearest fixed site. Outdoor  $O_3$  concentrations at homes were approximately 80 percent of those measured at fixed sites, but a



Figure 5.6. Scatter plot of  $O_3$  exposure model estimate of maximum hourly average personal exposure versus maximum hourly average measurement of  $O_3$  exposure by personal monitoring for forty-nine daytime monitoring periods between 7 A.M. and 7 P.M., with a mean duration of eight hours. *Source:* Adapted from Contant et al. (1987), with permission.

relatively small proportion of daily time is spent outdoors around the place of residence. This research demonstrated that large improvements in the accuracy of  $O_3$  exposure assessments can be achieved by the simple weighting of personal activity patterns into indoor and outdoor classes.

### NITROGEN DIOXIDE

Direct and indirect approaches to exposure assessment have been combined to strengthen the design of surveys to measure personal exposure to  $NO_2$  in the community. The Harvard School of Public Health developed a model of personal exposure based on ambient monitoring information and coarse activity pattern information on time spent indoors at home, indoors at work, outdoors, and in transit (Ryan et al. 1988a, 1988b, 1989; Spengler et al. forthcoming). Comparison of the estimates of the initial model with actual personal monitoring data demonstrated the importance of refining the model to account explicitly for exposures in three other microenvironments with potentially elevated  $NO_2$  concentrations: in-home cooking areas with unvented combustion appliances, travel on roadways during commuting hours, and certain occupational settings. Personal activity diaries were

modified to collect information on the time spent in these special settings.

Personal exposure surveys on representative samples of urban residents were conducted to determine the population distributions of NO2 exposures. Utilizing an integrating diffusion badge, personal exposures were quantified for approximately three hundred individuals in Boston and seven hundred residents in Los Angeles. Subjects wore one badge while indoors and a different badge when outdoors. Outdoor measurements were also made at each subject's residence. Ambient levels of NO2 were higher in Los Angeles (30-70 ppb) than they were in Boston (20-30 ppb). Unlike Boston, in Los Angeles approximately 40 percent of the variance in personal exposures and 60 percent in indoor residential concentration was explained by ambient measurements made outside the residence. These results suggest that in areas of higher ambient pollution with substantial spatial variation, outdoor concentrations can influence exposure. This occurs through the contribution of outdoor concentrations to indoor concentrations because the modeled prediction does not improve when the fractional times spent outdoors are included as an independent variable.

#### VOLATILE ORGANIC COMPOUNDS

Wallace and co-workers at the EPA have measured personal exposures to VOCs in several metropolitan areas across the United States in a group of interrelated studies called the TEAM studies. In each metropolitan location, random stratified samples were selected on the basis of proximity to point sources and socioeconomic class, geographic area, and demographic characteristics including age, marital status, tobacco smoking status, and occupational class. Personal exposure to VOCs was measured with personal samplers; end-exposed breath samples were obtained at the end of each twenty-four-hour period; water samples from the homes were taken for VOC analysis; and outdoor sites were monitored (see Chapter 11 for further details). Sources of exposure were inferred by questionnaire data on personal activities and proximity to potential sources.

Table 5.2 presents a summary of the results of the TEAM survey in one urban location, the Bayonne and Elizabeth, New Jersey, survey. The exposures experienced outdoors and indoors were highly variable, both from compound to compound and within a compound. However, indoor concentrations were consistently higher than outdoor concentrations. The higher concentrations observed indoors were unexpected because this study site has many industrial sources of VOCS

Breath measurements did not correlate well with ambient concentration measurements, further indicating that ambient data do not represent population a posures accurately. However, for some specific VOCs, elevated personal exposures as measured by breath samples were associated with certain types of activities. For example, personal exposure to benzene was correlated with visitate service stations, and tetrachloroethylene was correlated with visits to dry cleaning businesses.

The TEAM VOC studies have had a major impact on the way in which the research community views environmental exposures to VOCs. In a review of the

# Table 5.2 Summary of Median and Maximum Concentrations $(\mu g/m^3)$ for Elizabeth-Bayonne, New Jersey, TEAM Study of Volatile Organic Compounds

Compound	Outdoor <sup>a</sup>	Indoort	
Chloroform 1,1,1-Trichloroethane Benzene Carbon tetrachloride Trichloroethylene Tetrachloroethylene Styrene m.p-Dichlorobenzene :thylbenzene -Xylene n.p-Xylene	$\begin{array}{c} 0.74^{\prime\prime} \ (21.5) \\ 4.20 \ (40.0) \\ 7.00 \ (91.0) \\ 0.81 \ (14.0) \\ 1.34 \ (15.0) \\ 2.60 \ (27.0) \\ 0.67 \ (11.0) \\ 0.80 \ (13.0) \\ 3.20 \ (20.0) \\ 3.00 \ (27.0) \\ 9.90 \ (70.0) \end{array}$	2.94 (215) 15.60 (880) 13.00 (120) 1.38 (14.0) 2.00 (47.0) 5.60 (250) 1.80 (53.5) 2.80 (915) 6.10 (320) 4.98 (46.0) 15.50 (120)	Ratioe 3.97 (10.0) 3.71 (22.0) 1.86 (1.32) 1.70 (1.00) 1.49 (3.13) 2.15 (9.26) 2.69 (4.86) 3.50 (70.4) 1.91 (16.0) 1.66 (1.70) 1.57 (1.71)

Source: Adapted from Wallace et al. (1986) with permission.

"Outdoor heading corresponds to overnight outdoor air in TEAM nomenclature.

eIndoor heading corresponds to overnight personal air in TEAM nomenclature. Summary statistics presented include a small number of personal exposures not in indoor environ-

«Ratio of indoor median to outdoor median (ratio of indoor maximum to outdoor "Median (maximum).

EPA's research on total human exposure, Ott et al. (1986) acknowledged these major contributions of the TEAM studies: (a) Variability of two to three orders of magnitude in exposures is found over small geographic regions, suggesting a need to reconsider epidemiologic approaches that assume homogeneous exposures cross broad areas; (b) personal and indoor exposures consistently exceed outdoor concentrations; (c) although inhalation is the prime exposure route to many VOCs, ingestion by drinking water can be a major route of exposure to chloroform and other species; and (d) breath samples are a reliable biologic marker for VOC exposure and correlate well with personal exposures.

# SUMMARY

The majority of daily activity is spent in indoor settings, and therefore, on a timeweighted basis, indoor exposures may dominate the total exposure of most individuals when concentrations experienced in other microenvironments are of comparable magnitude. An understanding of the personal exposures in the indoor ctting is essential and will continue to be an important focus in air pollution epidemiology and public health planning. This chapter has reviewed the basic incepts of human exposure assessment and has presented some of the methadologic considerations. These approaches to the estimation of actual personal exposures offer encouraging prospects for improvement of our understanding of the relationship between air pollutant exposures and health effects and the potential for intervention to reduce those exposures.

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