

Personal Exposure Meets Risk Assessment: A Comparison of Measured and Modeled Exposures and Risks in an Urban Community

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Human exposure research has consistently shown that, for most volatile organic compounds (VOCs), personal exposures are vastly different from outdoor air concentrations. Therefore, risk estimates based on ambient measurements may over- or underestimate risk, leading to ineffective or inefficient management strategies. In the present study we examine the extent of exposure misclassification and its impact on risk for exposure estimated by the U.S. Environmental Protection Agency (U.S. EPA) Assessment System for Population Exposure Nationwide (ASPEN) model relative to monitoring results from a community-based exposure assessment conducted in Baltimore, Maryland (USA). This study is the first direct comparison of the ASPEN model (as used by the U.S. EPA for the Cumulative Exposure Project and subsequently the National-Scale Air Toxics Assessment) and human exposure data to estimate health risks. A random sampling strategy was used to recruit 33 nonsmoking adult community residents. Passive air sampling badges were used to assess 3-day time-weighted-average personal exposure as well as outdoor and indoor residential concentrations of VOCs for each study participant. In general, personal exposures were greater than indoor VOC concentrations, which were greater than outdoor VOC concentrations. Public health risks due to actual personal exposures were estimated. In comparing measured personal exposures and indoor and outdoor VOC concentrations with ASPEN model estimates for ambient concentrations, our data suggest that ASPEN was reasonably accurate as a surrogate for personal exposures (measured exposures of community residents) for VOCs emitted primarily from mobile sources or VOCs that occur as global "background" source pollutant with no indoor source contributions. Otherwise, the ASPEN model estimates were generally lower than measured personal exposures and the estimated health risks. ASPEN's lower exposures resulted in proportional underestimation of cumulative cancer risk when pollutant exposures were combined to estimate cumulative risk. Median cumulative lifetime cancer risk based on personal exposures was 3-fold greater than estimates based on ASPEN-modeled concentrations. These findings demonstrate the significance of indoor exposure sources and the importance of indoor and/or personal monitoring for accurate assessment of risk. Environmental health policies may not be sufficient in reducing exposures and risks if they are based solely on modeled ambient VOC concentrations. Results from our study underscore the need for a coordinated multimedia approach to exposure assessment for setting public health policy. **Key words:** hazardous air pollutants, personal exposure monitoring, risk assessment, urban communities. *Environ Health Perspect* 112:589–598 (2004). doi:10.1289/ehp.6496 available via <http://dx.doi.org/> [Online 22 December 2003]

The absence of human exposure information constitutes a critical source of uncertainty for risk-based regulatory decision making. Risk assessments are used by the U.S. Environmental Protection Agency (U.S. EPA) to estimate the likelihood that exposure to a given pollutant will produce an adverse health effect and to determine what regulatory actions are necessary to protect public health. In the absence of human exposure data, policy makers, risk assessors, regulators, researchers, and public health officials often must rely on estimates or surrogates of human exposure levels, such as proximity to a hazardous waste site or regional ambient air quality data. Such estimates may be derived from models that predict levels of environmental contamination in the air. These approaches are limited in identifying health risks because they rely on assumptions about actual exposures experienced by people, thus introducing uncertainty in their risk estimates and ensuing policies. Although monitoring is

generally recognized as providing a more reliable estimate of exposure, it carries its own limitations, such as cost for implementing on a large population scale over long periods of time to estimate long-term exposures.

In 1995, the U.S. EPA released the results of its Cumulative Exposure Project (CEP). Under the CEP, the U.S. EPA used an air dispersion model, the Assessment System for Population Exposure Nationwide (ASPEN) model, and 1990 emissions inventory data to characterize the magnitude, extent, and significance of airborne outdoor concentrations for 148 hazardous air pollutants (HAPs) listed under the 1990 Clean Air Act Amendments (CAAA 1990) for each of the 60,803 census tracts in the contiguous United States (Woodruff et al. 1998). Although the model estimated exposures to HAPs of ambient origin, by default they were assumed to represent total human exposure forming the basis for human health risk estimation.

Therefore, not only the validity of the ASPEN estimate relative to ambient measurements of interest but also the magnitude of the difference relative to personal and indoor exposure and the significance of this difference in risk estimation are important to understand. Results from the CEP suggested that HAP exposures were prevalent nationwide and that, in some locations, concentrations were significantly higher than concentrations associated with the one-in-one million excess cancer risk, levels considered by U.S. EPA researchers as a benchmark for acceptable "de minimus" risk (Caldwell et al. 1998; Woodruff et al. 1998). U.S. EPA researchers also concluded that HAP concentrations estimated by the model may pose a significant public health problem, especially in urban census tracts and census tracts of predominantly low-income and minority populations (Morello-Frosch 1997; Morello-Frosch et al. 2000). The main sources of the HAPs were found to be mobile

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(e.g., automobiles and trucks) and area sources (e.g., dry cleaners and gas stations).

The CEP has provided critical information about possible population exposures to HAPs and their relationship with population demographics (race, ethnicity, and income) never before revealed on a national scale. In addition, the CEP has served as a prototype for the National-Scale Air Toxics Assessment (NATA). The U.S. EPA released the NATA modeling and risk assessment results in two phases: first, in September 2000, the ASPEN modeling data only; and then later in May 2002, results from a human exposure module [Hazardous Air Pollutant Exposure Model 4 (HAPEM4)] added to ASPEN and related risk estimates using 1996 air toxics emissions data as input for the ASPEN model. Given the paucity of comprehensive ambient air monitoring data for HAPs and even fewer human exposure data, national air toxics modeling as carried out by the U.S. EPA will play a significant role in identifying effective control strategies to reduce public health risks from exposure to HAPs as required by 1990 CAAA, and in shaping national policies to reduce air pollution emissions.

Many volatile organic compounds (VOCs) are listed by the U.S. EPA as HAPs (e.g., benzene, carbon tetrachloride, and chloroform) and were included in the CEP. Beginning in the 1980s with the U.S. EPA's Total Exposure Assessment Methodology (TEAM) studies of VOCs, it has been demonstrated repeatedly that personal exposures typically exceed outdoor air concentrations and that levels of human exposure to VOCs depend on people's locations, especially indoors, where people spend up to 90% of their time (Aklund et al. 1997; Buckley et al. 1997; Clayton et al. 1999; Cohen et al. 1989; Kinney et al. 2002; Leung and Harrison 1998; Liroy 1990; Otson et al. 1994; Ott 1990; Pellizzari et al. 1999; Seifert et al. 1989; U.S. EPA 1987; Wallace 1993; Wallace et al. 1985; Weisel 2002). Therefore, assessment of potential public health impacts from HAPs is limited by the uncertainty in exposure estimates based on fixed-site ambient monitoring or models that use ambient concentrations to estimate exposure. Environmental policies that focus solely on reducing HAP emissions from stationary or point sources may not be effective in reducing human exposures and risks when the indoor environment is a significant contributor to exposures.

At the same time that the U.S. EPA released results from the CEP, a community-based VOC exposure study, conducted in partnership with South Baltimore, Maryland, community leaders, was in the planning phases. As a result, an opportunity arose to examine *a*) whether ambient concentrations of VOCs based on the U.S. EPA's ASPEN model were adequate estimates of ambient air

toxic concentrations in South Baltimore and *b*) the magnitude of the differences between ambient estimates and the more health relevant indoor and personal exposures and the differences between their associated health risks. In this article we present the results of this investigation.

Materials and Methods

Study area. The South Baltimore communities of Brooklyn, Brooklyn Park, and Curtis Bay are located in the southeastern quadrant of the City of Baltimore and adjacent counties. According to the 2000 U.S. Census, approximately 24,000 people live in South Baltimore: 80% white, 15% African American, 2% Asian, 2% Latino, and 1% of other ethnic backgrounds (U.S. Census Bureau 2003). Most (60%) of these residents have a high school education, and the median family income in 2000 was about \$37,000 per year (U.S. Census Bureau 2003).

South Baltimore (Figure 1) presents a unique exposure scenario because of the intensity of large chemical industries in close proximity to residential areas. According to the U.S. EPA, there are approximately 189 permitted or registered stationary air pollution sources [including Toxic Release Inventory (TRI) reporting facilities and smaller facilities] located in South Baltimore (U.S. EPA 2000b). South Baltimore (as defined by ZIP codes 21226 and 21225) is ranked 12th in the top 100 ZIP codes for total pollutant releases into the environment, with 360,479,759 lb released to air, land, and water bodies annually (Environmental Defense 2001). The communities' industrial air toxics pollutant burden is compounded by intense mobile-source

emissions from nearby interstate highways and local truck traffic servicing industry.

Participant recruitment and data collection. A population-based random sampling strategy was used to recruit 37 adult residents from the study area for personal exposure monitoring during January 2000 through June 2001. Nonsmoking residents were recruited into the study to limit the influence of active tobacco smoking on personal exposure measurements, because the constituents of tobacco smoke include a number of our target VOCs (Miller et al. 1998). Passive air sampling badges (#3500 organic vapor monitor; 3M Co., St. Paul, MN) were used to assess 3-day time-weighted-average (TWA) personal inhalation exposures as well as outdoor and indoor residential air concentrations of 11 VOCs (Table 1) for each study participant.

Study participants were asked to wear the sampling badges on a shirt lapel or collar near their breathing zone whenever possible. Indoor residential sampling badges were placed in the room where the participant usually spent his or her most time when not sleeping. Residential outdoor sampling badges were placed in a protected but unobstructed location just outside the home.

Questionnaires were used to collect participant demographic information, including age, race, occupation, and household income, as well as exposure determinants—for example, use of air fresheners, dry cleaning, and mode of transportation. Each subject was asked to maintain a daily time-activity diary to determine the time spent indoors and outdoors and to identify VOC sources, such as environmental tobacco smoke, occupational exposures, or car refueling during the 3-day monitoring period.

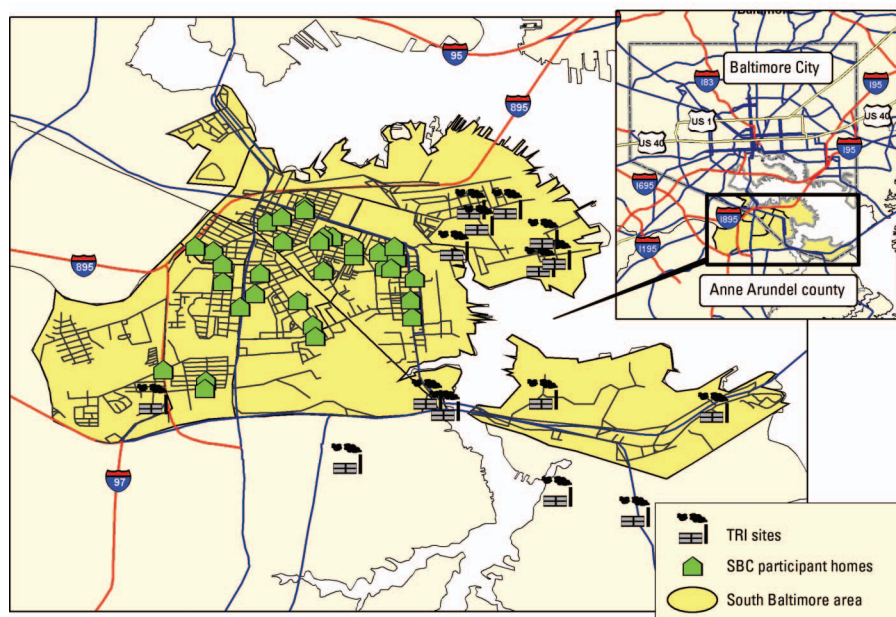


Figure 1. Map of South Baltimore. SBC, South Baltimore Community Study.

Informed consent was obtained from all study participants following procedures established by the Johns Hopkins University Bloomberg School of Public Health Human Subjects Review Board.

VOC Sample analysis. At the conclusion of the 3-day sampling period, the sampling badges were collected, sealed with plastic covers and transported to the lab and stored at -20°C . Analysis was conducted using gas chromatography/mass spectrometry (5890/5971 series II; Hewlett Packard, Palo Alto, CA) following a standard method outlined by Chung et al. (1999). After adding an internal standard, the adsorbed VOCs were extracted from the sampling badges using a 2:1 solution of acetone:carbon disulfide containing the surrogate 4-bromofluorobenzene. The method detection limit (MDL) was determined from field blanks as the value corresponding to the 99% confidence interval. VOC sample concentrations below the MDL were set to one-half the MDL. Further details on the sample analysis methods are discussed elsewhere (Buckley et al. 2003). Descriptive statistics including central tendency and variability were generated to characterize personal exposures and indoor and outdoor concentrations for each of the 11 VOCs.

Modeled ambient VOC data. The most recent (1996) ambient modeling results from the U.S. EPA's ASPEN model for the 11 target VOCs were obtained from the U.S. EPA Office of Air Quality Planning and Standards (U.S. EPA 2000c). For each pollutant, ASPEN provides modeled ambient annual average

concentrations in units of micrograms per cubic meter for three source categories (area, mobile, and point sources) and the total modeled ambient concentrations (U.S. EPA 2000c). These values were abstracted from the database for the eight census tracts that define the study communities. Pollutant concentrations from the ASPEN model were weighted by the number of participants monitored in each census tract and summarized by measures of central tendency and distribution percentiles.

Comparison of model with measured exposure concentrations. Although our measured exposure results and the ASPEN model could not be statistically compared because information was lacking on the uncertainty associated with ASPEN model estimates, we could describe the magnitude and direction of differences. For each VOC, ratios were calculated for each individual measurement (indoor, outdoor, and personal) to the ASPEN model estimates corresponding to the individual's home census tract. The ASPEN model estimates were judged reasonable surrogates for personal, indoor, and/or outdoor exposures if they were within a factor of 2 (median ratios of exposure to ASPEN concentrations, ranging from 0.5 to 2.0). The U.S. EPA generally applies this criterion for model-to-monitor comparison when comparing modeled ambient concentrations with data from air monitoring stations (U.S. EPA 2001). The accuracy of the ASPEN estimates across the census tracts was assessed by plotting median VOC concentration obtained from the personal, indoor, and outdoor

monitoring against the median concentration from ASPEN and determining the Pearson correlation coefficients.

Cumulative risk analysis. U.S. EPA cancer classification and the critical end points considered in the risk assessment for each of the target VOCs are presented in Table 1. For carcinogenic hazard, both the unit risk estimate (URE; risk per microgram per cubic meter) and the equivalent concentrations (micrograms per cubic meter) representing generally the upper bound of a one-in-one million excess risk or probability of contracting cancer over a lifetime of exposure are also presented in Table 1 (Caldwell et al. 1998). These concentrations posing a one-in-one million cancer risk are presented as a benchmark value for cancer effects, consistent with sections 112(f) and 112(c)(9) in the CAAA (1990) allowing exemption from regulation of source categories when posing less than a one-in-one million lifetime risk to the most exposed individual (Caldwell et al. 1998). Specifically, under the CAAA, a cancer risk of one-in-one million is considered negligible risk. The non-cancer "health benchmarks" were defined by the inhalation reference concentrations (RfCs). An inhalation RfC is defined as an estimate (with uncertainty spanning perhaps one order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious noncancer health effects during a lifetime (U.S. EPA 1994). These toxicity data were obtained from

Table 1. Target VOCs and associated toxicity values

VOC pollutant	Weight of evidence ^a	Cancer health effects			Source	Target organ for critical chronic effects	Noncancer health effects		
		Unit risk estimate (per $\mu\text{g}/\text{m}^3$)	One per million cancer risk equivalent concentration ($\mu\text{g}/\text{m}^3$)	Source			Target organs for other critical chronic effects	RfC ($\mu\text{g}/\text{m}^3$)	Source
Benzene	A	7.8×10^{-6}	0.13	IRIS ^b	Blood; nervous system; immune systems	Reproductive/developmental	60	Cal EPA ^c	
Carbon tetrachloride	B2	1.5×10^{-5}	0.067	IRIS ^b	Liver	Kidney; reproductive/developmental; nervous system	40	Cal EPA ^c	
Chloroform	B2	2.3×10^{-5}	0.043	IRIS ^b	Liver	Kidney; reproductive/developmental	35	CEP ^d	
Ethylbenzene	Positive in NTP study but not classified	5.0×10^{-7}	2.0	CEP ^d	Reproductive/developmental	Liver and kidney	1,000	IRIS ^b	
Methylene chloride	B2	4.7×10^{-7}	2.1	IRIS ^b	Cardiovascular system	Central nervous system	400	Cal EPA ^c	
MTBE	—	2.6×10^{-7}	3.84	Cal EPA ^c	Liver/kidney		3,000	IRIS ^b	
Styrene	C	5.0×10^{-7}	2.0	CEP ^d	Nervous system	Liver	900	Cal EPA ^c	
Perc	B2, C	5.6×10^{-6}	0.18	Cal EPA ^c	Nervous system	Liver and kidney	35	CEP ^d	
Toluene	D	—	—	IRIS ^b	Nervous system	Liver; reproductive/developmental	400	IRIS ^b	
Trichloroethylene	B2, C	2.0×10^{-6}	0.59	Cal EPA ^c	Nervous system	Respiratory; liver	600	Cal EPA ^c	
Xylenes	D	—	—	IRIS ^b	Nervous system	Respiratory	300	CEP ^d	

MTBE, methyl *tert*-butyl ether; perc, tetrachloroethylene; —, no data because the compounds were not classifiable as to cancer risk.

^aGroup A, known carcinogen; group B1, probable carcinogen; group B2, probable carcinogen; group C, possible carcinogen; group D, not classifiable; group E, evidence of noncarcinogenicity. ^bU.S. EPA's Integrated Risk Information System (www.epa.gov/iris/index.html; U.S. EPA 2000d). ^cCalifornia Environmental Protection Agency, Office of Environmental Health Hazards Assessment (<http://oehha.ca.gov/>; Cal EPA 2002). ^dU.S. EPA's Cumulative Exposure Project (Caldwell et al. 1998).

various sources, including *a*) the CEP, *b*) the U.S. EPA's Integrated Risk Information System (IRIS), and *c*) the California Environmental Protection Agency (Cal EPA) (Caldwell 1998; Cal EPA 2002; U.S. EPA 2000d). Preference was given to the most current U.S. EPA-derived toxicity data for inhalation exposures from the CEP. For HAPs without a U.S. EPA or Cal EPA toxicity estimate, but with a U.S. EPA or International Agency for Research on Cancer weight of evidence indicating a potential for carcinogenicity or clear evidence of carcinogenicity in animals in a National Toxicology Program study, a default potency value equal to that of methylene chloride—the lowest of the 82 available carcinogenic U.S. EPA-derived potency values for individual HAPs—was assigned per the approach used for the U.S. EPA's CEP (Caldwell et al. 1998).

Cancer and noncancer risks were assessed using conventional approaches (U.S. EPA 1986, 1999, 2003) applied to the exposure estimates based on our measured exposures and the ASPEN model estimates for comparison purposes. The common approach to estimating inhalation cancer risk is to multiply the estimated annual average pollutant concentration by its URE, the inhalation cancer potency estimate based on the slope of the dose–response curve. In the absence of information to the contrary, a nonthreshold, linear model is assumed for cancer when extrapolating from high dose to low dose and/or from animals to humans (U.S. EPA 1986, 1999, 2003). The URE represents the excess cancer risk over background associated with continuous lifetime exposure to a pollutant and is typically expressed as risk or probability of cancer for a 70-yr exposure per 1 µg pollutant/m³ air (U.S. EPA 1986). UREs are derived either from occupational studies in humans, typically adult males, when available, or from toxicologic studies in animals (Woodruff et al. 2000). A URE based on human data is typically defined as the maximum likelihood estimate representing a “best estimate” of the dose response in the occupational study population and are somewhat less conservative than upper bound estimates. (Woodruff et al. 2000). For example, inhalation potency estimates for two of the pollutants in this present analysis, benzene and chloroform, are based on human data. UREs based on animal data are the upper 95% confidence bound of the estimated cancer potency. The use of upper bounds is generally considered a health-protective approach for covering the risk to susceptible individuals (U.S. EPA 2003).

Cancer risk for each VOC and each study participant was calculated using Equation 1:

$$E_{ij} \times URE_j = R_{ij} \quad [1]$$

where R_{ij} is the estimated risk from pollutant j for study participant i , E_{ij} is the measured exposure (indoor, outdoor, or personal) or ASPEN-modeled estimated exposure concentration (micrograms per cubic meter) for pollutant j for study participant i , and URE_j is the inhalation URE for pollutant j from Table 1. Summary statistics (e.g., mean, median, percentiles) of the cancer risk estimates for each pollutant were calculated across study participants.

To derive estimates of lifetime population excess cancer incidence or number of cancer cases expected over a lifetime, we applied Equation 2:

$$R_{ij} \times P = CC_{ij} \quad [2]$$

where R_{ij} is the risk associated with each VOC pollutant j and each study participant i from Equation 1, P is the total population size of the study communities (estimated to be 24,000 based on 2000 Census; U.S. Census Bureau 2003), and CC_{ij} is the estimated number of excess cancer cases expected over a lifetime for pollutant j based on exposure for study participant i . CC_{ij} estimates were summarized (e.g., mean, median, percentiles) across study participants for each pollutant to calculate population-level estimates of lifetime excess cancer cases. We compared the mean number of estimated cancer cases because the mean has public health relevance for exposure and risk, because it takes into account the full distribution of values including extreme values, whereas the median and geometric mean lack physical meaning and would underestimate the true risk (Ott 1994).

To evaluate cumulative cancer risks associated with exposures to the target VOCs, compound-specific cancer risk estimates for all of the known, possible, and probable carcinogenic VOCs (all but two of the target VOCs) for each study participant were summed as defined below in Equation 3, assuming cumulative cancer risks are additive per U.S. EPA guidelines (Caldwell et al. 1998):

$$\sum_j R_{ij} = CR_i \quad [3]$$

where R_{ij} is the estimated risk from pollutant j for study participant i and CR_i is cumulative cancer risk for participant i . Summary statistics (e.g., mean, median, percentiles) of the cumulative cancer risk estimates CR_i were calculated across study participants to estimate population-level cumulative cancer risk estimates.

The lifetime excess cancer cases associated with cumulative exposure to the target VOCs were estimated using Equation 4:

$$CR_i \times P = CCC_p \quad [4]$$

where CR_i is cumulative cancer risk for participant i from Equation 3, P is the total population size of the study communities, and CCC_i is the estimated excess cumulative cancer cases based on cumulative exposure for study participant i . Summary statistics (e.g., mean, median, percentiles) of the number of cumulative cancer cases CCC_i were calculated across study participants to estimate population-level lifetime cumulative cancer cases. Mean number of cumulative cancer cases has more public health relevance and is preferred to compare the mean based on our measured exposures and the ASPEN estimates.

For noncancer effects, the default assumption is that the dose–response model has a threshold below which no adverse health effects are expected to occur. Noncancer risks in this study were measured by a direct comparison of the exposure with a chemical-specific RfC. Each study participant's exposure was divided by the pollutant's RfC, the noncancer “health benchmark” to calculate a hazard quotient (HQ):

$$\frac{E_{ij}}{RfC_j} = HQ_{ij} \quad [5]$$

where E_{ij} is the measured exposure (indoor, outdoor, or personal) or ASPEN-modeled estimated exposure concentration (in µg/m³) for pollutant j for study participant i , RfC_j is the noncancer RfC for pollutant j (micrograms per cubic meter), and HQ_{ij} is the HQ for participant i for pollutant j . HQs > 1 indicated that the VOC concentration exceeded the benchmark concentration and could be of public health concern. If the HQ was ≤ 1, no harm was expected because the exposure was below the threshold (the RfC) for an adverse effect.

Cumulative noncancer risks were assessed by aggregating the HQs across the VOCs that affected the same target organ using Equation 6:

$$\sum_{\substack{j \text{ common} \\ \text{endpoints}}} HQ_{ij} = TOSHI_i \quad [6]$$

where HQ_{ij} is the HQ for participant i for pollutant j for a specific health end point (e.g., cardiovascular system, central nervous system), where $TOSHI_i$ is defined as the target-organ-specific hazard index and is the sum of HQs for individual VOCs that affected the same organ or organ systems for participant i (U.S. EPA 2001). Summary statistics (e.g., mean, median, percentiles) for $TOSHI_i$ were calculated across study participants to estimate population-level cumulative noncancer risks.

Results

Summary of exposure measurements. Most participants were women (70%) and white (84%). Median household income was in the

\$30,000–40,000 range. Median age of the participants was 53 years. Thirty-two percent of study participants had not completed high school, 35% completed high school only, and 22% had attended some college or technical school. The income and racial demographics of our study participants were comparable with the 2000 Census data for South Baltimore; however, our sample was slightly more educated, older, and mostly women compared with the 2000 Census.

More than half (54%) of the participants were not working during the exposure monitoring. The automobile was the most common mode of transportation among the participants. Housing stock tended to be older homes (73% of participants lived in homes built in 1950 or earlier) without an attached garage. On average, participants reported that they spent 80% of their time indoors. Based on questionnaire responses, four of the 37 study participants were found to be smokers. Of nonsmoking participants ($n = 33$), 42% reported environmental tobacco smoke exposure at some time during the monitoring period.

Analysis of exposure measurements (personal, indoor, and outdoor) and subsequent comparison with ASPEN and the risk analysis were restricted to the exposure measurements of the nonsmoking adult participants ($n = 33$). The frequency of VOC detection was greatest for personal, followed by indoor, and then outdoor measurements. The most frequently detected VOCs (e.g., > 80% of samples were above the MDL) in personal air were benzene, carbon tetrachloride, chloroform, ethylbenzene, methylene chloride, methyl *tert*-butyl ether (MTBE), styrene, tetrachloroethylene (perc), toluene, and the xylenes. Similar frequencies were found with the indoor samples except for styrene, where only 65% of indoor samples were above MDL. VOCs with > 80% of outdoor samples detected above the MDL were carbon tetrachloride, MTBE, and the xylenes.

Figure 2 presents a summary of the personal exposure and indoor and outdoor monitoring for each of the target VOCs. The box plots indicate the 10th, 25th, 50th, 75th,

and 90th percentiles for the 3-day TWA exposures for each VOC. For most of the VOCs, exposure concentrations spanned several orders of magnitude; however, the range from the 25th to the 75th percentile was generally no more than one order of magnitude. Trichloroethylene (TCE) tended to be the lowest in absolute concentration for all three sample types. MTBE, benzene, toluene, and ethylbenzene tended to be found in the highest absolute concentrations for all sample types. MTBE and toluene had the highest maximum personal (248.4 $\mu\text{g}/\text{m}^3$ and 195.6 $\mu\text{g}/\text{m}^3$, respectively), indoor (81.7 $\mu\text{g}/\text{m}^3$ and 114.80 $\mu\text{g}/\text{m}^3$, respectively), and outdoor (10.44 $\mu\text{g}/\text{m}^3$ and 8.61 $\mu\text{g}/\text{m}^3$, respectively) exposure measurements. On average, MTBE, toluene, and the xylenes contributed the most to total personal VOC exposures (28, 30, and 19% respectively) as proportion of average total VOC personal exposures.

As expected for most of the VOCs, personal exposures were greater than indoor concentrations, which were greater than outdoor concentrations. For example the median personal exposure was 14.6 $\mu\text{g}/\text{m}^3$ for toluene, compared with median indoor and outdoor toluene concentrations of 12.1 and 3.88 $\mu\text{g}/\text{m}^3$, respectively. In contrast, carbon tetrachloride and TCE were stable across the three locations as shown in Figure 2.

Comparison with the ASPEN model.

Table 2 presents the summary statistics from the ASPEN model across the eight census tracts in South Baltimore, along with results from the community monitoring. Figure 3 summarizes the observed ambient-to-model predicted concentration ratios for all 11 VOCs. The distribution of the ratios was positively skewed. For two of the VOCs, benzene and methylene chloride, ASPEN estimated concentrations were higher than our outdoor measurements, whereas the ASPEN estimates for chloroform, MTBE, and styrene were lower than our measurements. For carbon tetrachloride, ethylbenzene, perc, TCE, and xylenes, the ASPEN model provided reasonable central estimates of measured ambient concentrations. A comparison of the median

outdoor monitored concentrations versus the median ASPEN model predicted concentrations for each VOC resulted in a Pearson correlation coefficient of 0.97, demonstrating that ASPEN was capable of distinguishing the relative magnitude of ambient concentrations among the different VOCs, as reported by Rosenbaum et al. (1999). For most VOCs, the median ratios were within a factor of 2 (Figure 3), showing good agreement between ambient measurements and model predictions across South Baltimore.

ASPEN estimated ambient concentrations were generally lower than personal and indoor air measurements. However, for benzene, carbon tetrachloride, methylene chloride, MTBE, perc, and TCE, central estimates from the ASPEN model and indoor concentrations were comparable, with median ratios of observed indoor-to-model predicted concentrations within a factor of 2. Interestingly, for these same compounds, comparisons of measured indoor and outdoor concentrations revealed consistent VOC concentrations, indicating that ambient air infiltrated indoors and was an important driver for indoor concentrations posing the potential for influencing personal exposures. The ASPEN model and the personal exposure results for benzene, carbon tetrachloride, methylene chloride, and TCE were also similar, with median ratios of personal exposure to ASPEN model estimates in the factor of 2 range (0.5–2.0), as shown in Figure 4. For other VOCs, ASPEN model estimates were lower than personal exposure, especially for chloroform, toluene, and styrene (Figure 5).

Health risk estimates. Summary statistics of the estimated lifetime excess cancer risks based on exposure monitoring results from our adult participants and exposure estimates from the ASPEN model are presented by exposure category in Table 3. Cancer risks are all expressed as excess risk per one million population. Chloroform, benzene, and carbon tetrachloride presented the highest median cancer risks at 53, 23, and 12 per one million population, respectively, based on personal exposures, and were similar for risk estimates

Table 2. Comparison of ASPEN (1996) estimated VOC concentrations and measured (2000–2001) exposure ($\mu\text{g}/\text{m}^3$).

VOC pollutant	ASPEN model ^a		Outdoor monitoring ($n = 33$)				Indoor monitoring ($n = 33$)				Personal monitoring ($n = 31$)			
	Mean	Median	Mean	Median	10th ^b	90th ^b	Mean	Median	10th ^b	90th ^b	Mean	Median	10th ^b	90th ^b
Benzene	2.81	2.69	1.84	1.79	0.57	3.14	3.70	2.45	1.03	8.34	4.06	2.94	1.44	7.30
Carbon tetrachloride	0.88	0.88	0.94	0.90	0.60	1.48	0.98	0.85	0.51	1.66	0.94	0.82	0.60	1.70
Chloroform	0.10	0.09	0.44	0.22	0.07	0.89	4.36	2.30	0.61	7.89	4.79	2.29	0.70	7.80
Ethylbenzene	1.02	0.84	1.26	1.00	0.55	2.00	3.22	1.95	0.90	7.33	4.42	2.53	1.18	9.45
Methylene chloride	0.80	0.68	0.35	0.35	0.06	0.60	2.86	0.95	0.08	7.73	2.07	0.93	0.08	5.58
MTBE	2.86	2.64	4.41	4.30	0.99	8.70	10.80	4.25	1.06	21.99	24.74	8.80	2.66	66.57
Styrene	0.12	0.12	0.50	0.25	0.05	1.68	2.72	0.43	0.16	8.96	2.51	1.30	0.20	7.40
Perc	0.42	0.35	0.47	0.28	0.10	1.09	2.55	0.50	0.10	5.68	3.00	0.91	0.19	8.23
Toluene	6.05	4.94	4.10	3.88	1.66	6.43	21.90	12.12	5.79	50.13	26.81	14.65	7.75	41.33
TCE	0.20	0.18	0.19	0.17	0.08	0.24	0.36	0.18	0.08	0.56	0.41	0.20	0.09	0.83
Xylenes	3.92	3.44	4.68	3.97	2.36	7.09	12.36	7.60	3.57	23.01	17.75	9.50	4.61	30.85

^aWeighted by the number of exposure monitoring samples taken per census tract. ^bPercentiles.

based on indoor concentrations. Cancer risk estimates based on measured outdoor VOCs concentration were generally lower than personal exposures and indoor concentrations, except for carbon tetrachloride. Cancer risk estimates for carbon tetrachloride were similar across all three exposure monitoring categories.

Cancer risk estimates at the 90th percentile for chloroform were higher for personal and indoor exposures than for outdoor exposures, at 181 and 183 versus 20 per one million, respectively. Maximum cancer risk estimates based on personal exposures for benzene, chloroform, and perc were high. For example, the maximum estimated cancer risk based on personal exposures was 133, 801, and 135 per one million for benzene, chloroform, and perc and are in the range that the U.S. EPA would consider warranting action to reduce exposures.

Cancer risks based on results of the ASPEN model are also presented in Table 3. Again, chloroform, benzene, carbon tetrachloride, and perc presented the highest median cancer risks. Although the median cancer risks based on the ASPEN model estimates for benzene and carbon tetrachloride were comparable

with the risks based on personal exposures, median cancer risk estimates for the other VOCs were lower, especially for chloroform. The differences between risks based on ASPEN and exposure measurements were even greater at the 90th percentile and maximum risk estimates, corresponding to the earlier comparison between exposure estimates.

Table 4 shows estimated cumulative cancer risk and estimated number of cancer cases based on measured exposures and the ASPEN model. Assuming risks are additive, the median cumulative cancer risk based on personal exposures was 120 per one million. The average cumulative cancer risk based on personal exposures was 183 per one million, indicating that the distribution was skewed. The difference in the cumulative cancer risk between personal and indoor exposure at the 90th percentile may be the result of individual activities magnifying the variability in personal exposure concentrations. In applying Equation 4, we estimated on average the number of predicted cancer cases based on the distribution of cumulative exposures and risks to be four cancer cases over a lifetime

(70 years) within the South Baltimore population. Cumulative risk and cancer incidence estimates were similar for personal and indoor VOC concentration. In contrast, the ASPEN model and measured residential outdoor concentrations resulted in much lower cumulative risk estimates. Based on ASPEN, median cumulative cancer risk was 42 per 1 million, and the expected mean lifetime cancer incidence is estimated as one cancer case within the South Baltimore population.

Community exposure monitoring results and ASPEN model results were also evaluated for potential noncancer health risks. Here there was good agreement: Neither the ASPEN results nor the average exposure measurements (personal, indoor, or outdoor) exceeded the RfCs for any of the VOCs (results not shown). The HQs were all < 1, with average HQs ranging from 8.12×10^{-4} for MTBE based on the ASPEN model estimates to 0.14 for chloroform based on personal exposures. The maximum hazard index (HI) was 0.98 for personal exposure to chloroform. For cumulative noncancer effects, the median TOSHI were all < 1, across all pollutants and exposure estimators.

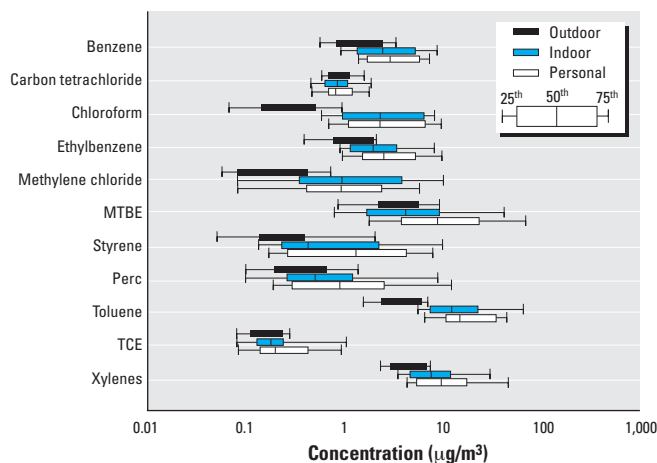


Figure 2. Distribution of personal VOC exposures and indoor and outdoor VOC concentrations for 25th, 50th, and 75th percentiles.

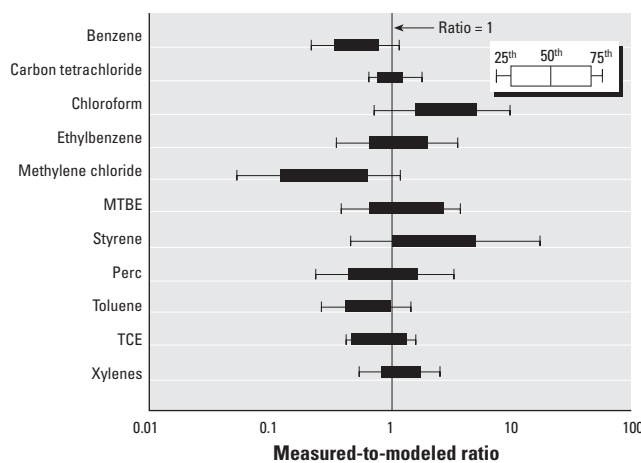


Figure 3. Distributions of measured-to-ASPEN modeled ambient VOC concentration ratios for 25th, 50th, and 75th percentiles.

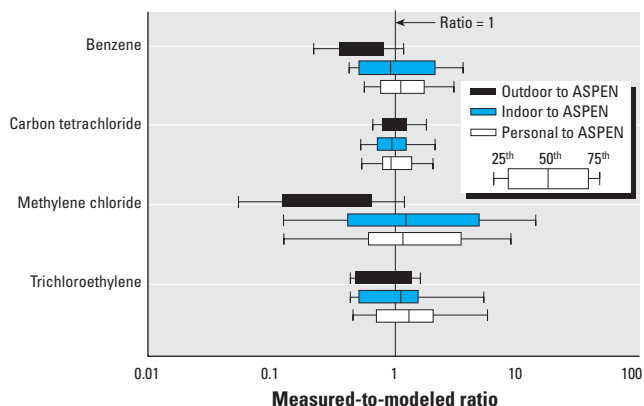


Figure 4. Distributions of measured-to-modeled ratios for VOCs for 25th, 50th, and 75th percentiles, showing good agreement.

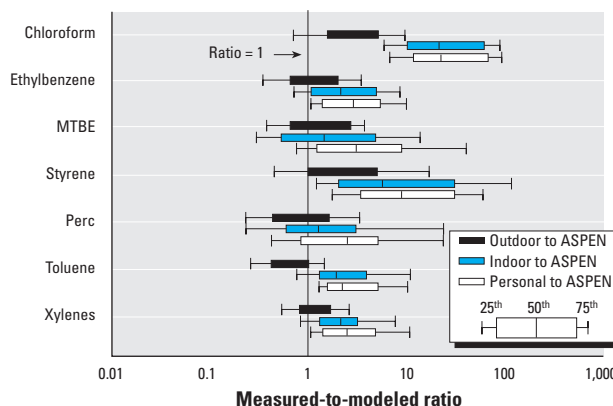


Figure 5. Distributions of measured-to-modeled ratios for VOCs for 25th, 50th, and 75th percentiles, showing poor agreement.

Discussion

Accurate exposure assessment is critical to a credible and scientifically sound assessment of risk (National Research Council 1983; Sexton et al. 1995). For the CEP, the U.S. EPA adopted the ASPEN model to estimate exposure and risk associated with census tract-level ambient air pollution levels. Because previous studies have shown that, for most VOCs, indoor and other microenvironmental concentrations (e.g., inside automobiles) are primary determinants of exposure and risk (Ott 1995; U.S. EPA 1987; Wallace 1990), the present study was conducted to explore the differences in measured exposure (indoor, outdoor, and personal) with ASPEN model ambient exposure estimates. This is the first direct comparison of the ASPEN model (as used by the U.S. EPA for the CEP and subsequently for the first phase of NATA) with human exposure data to estimate health risks.

The relative concentrations of VOCs measured indoors, outdoors, and on persons is a function of relative indoor and outdoor source contribution and time-activity patterns. This was initially identified by the TEAM studies where the indoor source contribution greatly exceeded that from outdoors (U.S. EPA 1987). Many VOCs are emitted from both outdoor sources (e.g., industrial facilities, power plants, dry cleaners, and mobile sources) and indoor sources (e.g., environmental tobacco smoke, paint, pesticides, varnishes, and household cleaners). In comparing our measured personal exposures with measured indoor and outdoor VOC concentrations, certain patterns emerged consistent with previous human exposure studies. For chloroform, toluene, methylene chloride, and styrene,

indoor concentrations dominated personal exposures. Ratios of personal to indoor concentrations for these VOCs were close to 1, whereas both indoor and personal exposures were six to seven times higher than measured outdoor concentrations. On the other hand, similar indoor and outdoor concentrations were observed for VOCs usually associated with motor vehicle emission, including benzene and MTBE. In those instances, ambient air may be an important driver for personal exposures, as documented by Kinney et al. (2002). In addition, similar VOC concentrations were observed across indoor, outdoor, and personal measurements for carbon tetrachloride and TCE. On the basis of the observed pattern of measured personal, indoor, and outdoor exposures, we could anticipate the outcome of the comparisons with the ASPEN model.

Consistent with what it was designed to estimate, the best agreement between ASPEN and our measurements was observed for outdoor measured VOCs, where all but two of the median ratios (measured to modeled concentrations) were within a factor of 2. The agreement of the modeled estimates to measured ambient concentrations was also indicated by the similarity in their relative ranking of VOCs. The favorable agreement between ASPEN and ambient VOC concentrations suggests that the VOC emission inventories that formed the basis of ASPEN have been well characterized for the study area. Styrene and chloroform, however, were the exceptions, with median ratios > 2, indicating that the ASPEN model underpredicted the measured ambient concentrations. The reason for the poor prediction is unclear; however, it may be due to source changes for styrene and

chloroform from 1996 (date of the emissions inventories) to 2001 (time period of our community monitoring). These findings are consistent with those in Rosenbaum et al. (1999), who reported that despite a tendency of the model to underpredict, the frequency of agreement in ranking between predicted concentrations and the observed concentrations obtained from stationary monitoring programs across the United States suggest reasonable good performance by ASPEN for most of the primary hazardous air pollutants. Rosenbaum et al. (1999) reported an R^2 statistic of 0.59 for model predicted versus observed HAP concentrations for monitors in the northeastern states. Pratt et al. (2000), in a comparison of ASPEN model estimates with ambient monitoring data for Minnesota, also concluded that the model tended to underestimate the monitored values. As with the present study, the monitor-to-model ratios were within a factor of 2 for most of the pollutants measured. Overall results from the Minnesota study suggest that the monitor and model results were in good agreement (Pratt et al. 2000). A similar ASPEN underestimation was observed in a study conducted by the U.S. EPA (2000a). Both the relative and absolute comparisons we conducted between model and monitored concentrations are important. For instance, if the U.S. EPA is interested in relative risk, then the relative ranking may be especially important. If the goal, on the other hand, is to assess whether risk exceeds some threshold, then the accuracy of the estimate as reflected by the median ratios is critical.

Even though ASPEN is designed to estimate exposure to air toxins of ambient origin, its comparison with measured indoor VOC

Table 3. Comparison of estimated individual pollutant cancer risk by exposure category in South Baltimore : all units in excess cancer risk per 1 million.

VOC pollutant	ASPEN model ^a					Outdoor concentration					Indoor concentration					Personal monitoring				
	Mean	Min	Median	90th ^b	Max	Mean	Min	Median	90th ^b	Max	Mean	Min	Median	90th ^b	Max	Mean	Min	Median	90th ^b	Max
Benzene	24.5	3.8	18.4	46.0	51.3	14.1	4.21	13.8	24.2	36.9	28.5	4.42	18.85	64.2	81.5	31.2	5.38	22.6	56.2	133
Carbon tetrachloride	15.1	2.9	17.5	24.1	29.4	14.1	1.49	13.4	22.1	26.9	14.6	1.49	12.7	24.8	40.3	14.1	4.48	12.3	25.4	31.3
Chloroform	2.64	0.49	2.77	4.55	5.48	16.9	1.55	5.12	19.9	79.1	101	2.33	53.5	183	695	111	6.98	53.3	181	801
Ethylbenzene	0.55	0.09	0.42	1.07	1.29	0.63	0.07	0.50	1.00	1.85	1.61	0.29	0.98	3.67	10.0	2.21	0.56	1.27	4.73	13.4
Methylene chloride	0.44	0.08	0.38	0.82	0.98	0.17	0.03	0.16	0.29	0.81	1.36	0.03	0.45	3.68	8.95	0.99	0.02	0.44	2.66	6.39
MTBE	0.51	0.08	0.39	0.93	1.15	0.73	0.05	0.72	1.45	1.74	1.80	0.05	0.71	3.67	13.6	4.12	0.16	1.47	2.66	41.4
Styrene	0.07	0.01	0.05	0.15	0.19	0.25	0.03	0.13	0.84	1.35	1.36	0.03	0.21	4.48	11.6	1.25	0.03	0.65	3.70	5.25
Perc	2.73	0.43	2.24	4.99	6.03	2.61	0.46	1.55	6.04	11.7	14.2	0.46	2.78	31.6	141	16.7	0.56	5.05	45.7	135
TCE	0.35	0.06	0.30	0.73	0.82	0.33	0.10	0.28	0.41	1.74	0.61	0.10	0.30	0.95	4.97	0.70	0.10	0.34	1.40	4.36

Abbreviations: Max, maximum; Min, minimum.

^aASPEN concentrations weighted by the number of exposure monitoring samples taken per census tract. ^bPercentiles.

Table 4. Comparison of cumulative cancer risk by exposure category for the VOC pollutants

	Cumulative risk (risk per 1 million)					Corresponding number of cancer cases (over a lifetime)				
	Min	Average	Median	90th percentile	Max	Min	Average	Median	90th Percentile	Max
ASPEN model	8	47	42	85	86	0	1	1	2	2
Outdoor concentrations	16	43	36	69	124	0	1	1	2	3
Indoor concentrations	26	165	120	193	740	1	4	3	7	18
Personal exposures	33	183	120	337	862	1	4	3	8	21

Abbreviations: Max, maximum; Min, minimum.

concentrations is of interest for evaluating the magnitude of the difference. Median indoor-to-ASPEN ratios for chloroform, ethylbenzene, toluene, styrene, and xylenes were 24.7, 2.24, 2.03, 6.30, and 2.25, respectively. However, favorable agreement between measured and modeled ambient concentrations was observed for VOCs, with indoor:outdoor ratios close to 1. Six of the 11 VOCs fell into this category: benzene, carbon tetrachloride, methylene chloride, MTBE, perc, and TCE. Similar to what was observed for the outdoor measurements, median indoor-to-ASPEN ratios across all six VOCs did not depart from unity by more than a factor of 2. For benzene and methylene chloride, the good agreement between indoor measurements and ASPEN is probably a result of ASPEN's higher estimation of ambient levels (median ratios of outdoor concentrations to ASPEN were 0.43 and 0.63 for methylene chloride and benzene, respectively).

The utility of the ambient concentration estimates given by ASPEN to predict exposure is most directly and comprehensively assessed by its comparison with personal monitoring. This comparison suggests that for VOCs with indoor:outdoor ratios near unity (e.g., benzene, carbon tetrachloride, and TCE), ASPEN provided reasonable central estimates for human exposure. This follows the pattern of agreement between indoor and outdoor measurements and ASPEN estimates discussed above. Although ASPEN provided a reasonable estimate of personal exposure to methylene chloride, median indoor concentrations were three times higher than outdoor concentrations. It was expected for carbon tetrachloride that ASPEN and personal exposures would be somewhat comparable because the main source of exposure to carbon tetrachloride is ambient "background" levels from past emissions (Rosenbaum et al. 1999; U.S. EPA 2000a). Ratios of the VOC concentrations for indoor-to-personal, personal-to-outdoor, and indoor-to-outdoor measurements of carbon tetrachloride were close to 1, suggesting that outdoor air was the only source for indoor and personal exposures. For TCE, the data suggest that there were no significant indoor sources that would affect personal exposures and thus ambient levels were adequate surrogates for exposure. As with the indoor comparison, the suitability of ASPEN for estimating personal exposures for benzene and methylene chloride is probably an artifact of ASPEN's minor overestimation of ambient concentrations for these pollutants, which is consistent with a comparison of the ASPEN model with monitor data (stationary monitors in this case) for northeastern states (including Maryland) conducted by Rosenbaum et al. (1999), based on 1990 emission inventory. For all the other measured VOCs, including chloroform, ethylbenzene, MTBE, perc, toluene, styrene, and

xylenes, ASPEN estimated concentrations were less than personal exposures by a factor of 3 (median of the ratio of ASPEN to personal measurements across these pollutants). For some VOCs, (e.g., MTBE and xylenes), personal exposures exceeded both indoor and outdoor concentrations, suggesting exposure in an unmonitored microenvironment such as work or the automobile. Gasoline or refueling is an unlikely source because other mobile source characteristic VOCs (e.g., benzene, toluene, and ethylbenzene) were not similarly elevated. Therefore, as expected, the agreement between the ambient VOC estimates provided by ASPEN and measured personal exposure varied by source: there was better agreement for VOCs with global background source (e.g., carbon tetrachloride) than for VOCs primarily emitted from mobile sources (e.g., benzene) than for VOCs from indoor sources (e.g., chloroform).

An additional important distinction between the ASPEN model estimates and the actual measurements (outdoor, indoor, and personal) is the spatial resolution of their assessment. ASPEN provides resolution to the census tract level, whereas monitoring provides within-tract spatial resolution at the level of the individual and residence, thereby including interindividual variability. Therefore, the utility of approach will depend on the variability of interest.

Because ASPEN was developed and has been used to assess risk and support policy development, the consequence of exposure misclassification on risk is of primary importance. Our results indicate that South Baltimore residents are routinely exposed to a number of VOCs that are considered toxic air pollutants by the U.S. EPA and at levels above public health benchmarks (approaching 1 in 10^4 excess cancer risk). Cancer risk estimates based on outdoor VOC monitoring are similar to risk estimates based on ASPEN model results, as expected. By comparing risks based on the ASPEN model with risks based on personal and indoor exposures, we demonstrated that the model underestimates exposures and therefore risks. This underestimation is especially pronounced for chloroform, ethylbenzene, MTBE, styrene, and perc, whereas estimates were comparable for benzene, carbon tetrachloride, methylene chloride, and TCE.

The present study shows that a modest underestimation of exposure by the ASPEN model on an individual pollutant basis resulted in a proportional underestimation of cumulative cancer risk. Although the numbers of estimated excess lifetime cancer cases due to cumulative exposure are not large in and of themselves, the 4-fold difference between the ASPEN estimate and that derived from measured exposure could have a large impact on risk management or policy decisions.

Risk based on the model also underestimated projected cancer incidence for individuals at the upper end of the exposure distribution. Identifying high-exposure groups is important because this population subgroup is at greatest risk, and it is for this group that intervention strategies will be most effective. Cancer risks based on ASPEN model concentrations did not adequately characterize risk of South Baltimore populations at the extreme end of exposure distributions.

Underestimation of exposure and risk may also lead to different prioritization of pollutants for environmental regulatory action to reduce risks and protect public health. Among the 11 VOCs measured in this study, benzene, carbon tetrachloride, chloroform, and perc were identified by both ASPEN and the exposure measurements as major risk drivers. However, their relative contribution to cumulative risk differed greatly. Compared with personal exposures, ASPEN overestimated risk contributions of benzene and carbon tetrachloride by more than 2-fold (52 vs. 17%) and 4-fold (32 vs. 8%), respectively. In contrast, ASPEN's estimate of chloroform's cumulative risk was 6%, whereas personal exposures indicated 61% contribution to cumulative risk. Perc's relative contribution to cumulative cancer risk was similar, with 6% estimated from ASPEN and 9% based on personal exposure. If ASPEN-based estimates of exposure formed the basis for policies to reduce cancer risk from air toxics, the focus would be primarily on benzene, whereas the personal exposure measurements indicate that chloroform contributes more to cumulative cancer risk. Chloroform represents a unique but important indoor air exposure. Indoor chloroform is generally not the result of industrial emissions or consumer products. Rather, it is formed as a byproduct of drinking water chlorination and is subject to regulation under the Safe Drinking Water Act (Safe Drinking Water Act Amendments 1996). National efforts have substantially reduced chloroform levels in drinking water; however, its ubiquitous presence in indoor air underscores the need for a coordinated multimedia approach to regulation. Nonetheless, this is not to suggest that the contribution of benzene to personal exposures, particularly from mobile sources, should be disregarded. Benzene was identified as the second largest cancer risk contributor in South Baltimore and is highly correlated with VOCs typically found in auto and truck exhaust. The toxic effects of benzene are more certain [Group A, known human carcinogen (U.S. EPA 2000d)], and benzene is ubiquitous in ambient air. The results of this study suggest that the U.S. EPA should continue to focus on benzene as an air pollutant of concern, but should also consider policies to reduce risk from toxic air pollutants

in the indoor environment. These policies could require more extensive disclosure of cancer risks from chemicals in building materials and household consumer and cleaning products used indoors.

This comparison of measured and modeled exposure and risk is limited with respect to two dimensions of time. First, the ASPEN estimates are based on annual averages, whereas the measured values are based on 3-day integrated samples. This limitation is partially offset by the fact that sampling was conducted over one year, thereby encompassing both seasonal and individual variability. The second temporal limitation is that ASPEN's 1996 estimates are being related to measurements conducted in 2000–2001. Therefore, results from the present study need to be interpreted with caution, recognizing that true differences between modeled and measured estimates are potentially confounded by differences in averaging time and period. Additional research can address these limitations with *a*) concurrent measurement and model estimates and *b*) a repeated measure design providing a better estimate of long-term exposure (Wallace et al. 1994). Although there are limits to this study with respect to the temporal associations forming the basis of comparison, these results provide the basis for an important evaluation of the differences between population-measured exposures and risks and the model estimates.

Over the next 2 years, the U.S. EPA is scheduled to reassess the cancer and noncancer effects for carbon tetrachloride, chloroform, methylene chloride, and TCE. The impact of this reassessment is particularly important for chloroform because new data being considered under IRIS review suggest that chloroform's carcinogenicity occurs with a threshold and only at relatively high concentrations (U.S. EPA 2000d). Accordingly, the U.S. EPA is working to revise the URE assessment for inhalation exposure for chloroform.

Conclusion and Recommendations

The U.S. EPA relied upon ASPEN for its CEP to estimate air toxic exposure and risk for the U.S. population. Therefore, the reliability of the ASPEN exposure estimates has implications for risk management and public health policy. The present study provides an evaluation of ASPEN based on measurements of air toxic levels indoors, outdoors, and on individuals. Study results suggest that for pollutants primarily of ambient origin—benzene, carbon tetrachloride, methylene chloride, and TCE—ASPEN provides a reasonable (within a factor of 2) central estimate for personal exposures. However, for the remaining seven VOCs with significant indoor sources, ASPEN estimates are substantially lower than personal exposures.

The CEP approach of estimating exposure of ambient origin understates cumulative risks due primarily to the exclusion of indoor exposures. The present analysis suggests that regulation solely focused on exposure and risk from VOCs of ambient origin will address only a small portion of the actual exposure and risk, as has been previously stated by Wallace (1989, 1990, 1991, 1993).

Given the resources and time necessary for exposure monitoring of large populations, models to estimate exposures provide a necessary practical alternative. Exposure models are intended to complement results from direct exposure monitoring studies and to extend and extrapolate these findings to other locales and other situations. In recognition of the limitations of using ASPEN to estimate risk and the need to account for the time that people spend indoors and outdoors, the U.S. EPA recently developed an exposure module, HAPEM4, and included it in the second phase of NATA. The HAPEM4 model was designed to predict the “apparent” inhalation exposure for specified population groups and air toxics. The HAPEM4 exposure model calculates the concentration in specific microenvironments (e.g., in a home or in a car) based on the ambient air concentration predicted by ASPEN (U.S. EPA 2002). Through a series of calculation routines, the HAPEM4 uses census data, human activity patterns, ambient air quality levels, climate data, and indoor/outdoor concentration relationships to estimate an expected range of “apparent” inhalation exposure concentrations of primarily ambient sources for groups of individuals (U.S. EPA 2002). It also predicts nationwide census-tract-level annual average human exposures and is to be used in a screening-level inhalation risk assessment. As the U.S. EPA continues to apply the ASPEN and HAPEM4 models to identify air toxics of greatest public health concern, and assess progress in reducing exposures across the United States, comparison of exposure measurements with modeling estimates provides the basis for continued model development and refinement. Although the second-phase NATA data were not available at the time of our study, a review of the HAPEM4 data versus ASPEN estimate shows that, for a number of our target VOCs, the HAPEM4 estimates are lower than those from ASPEN. This difference would mean that, compared with our measured personal exposures, risks based on HAPEM4 would be underestimated. We plan to conduct a detailed comparison of HAPEM4 with our measured exposures in a future analysis.

Overall validation studies for exposure models would be useful for varying environmental scenarios (e.g., rural community vs. suburban), in different regions of the country, for specific subpopulations (elderly, children, and ethnic minorities), and for an expanded

number and more varied types of hazardous pollutants (assuming sample collection instruments and analytical methods are readily available). An existing human exposure monitoring framework such as the U.S. EPA's National Human Exposure Assessment Survey, the Centers for Disease Control and Prevention (CDC) National Health and Nutrition Examination Survey, and the CDC's Second National Report on Human Exposure might provide excellent opportunities for exposure model validations. Results from these comparisons would help refine the models.

Although indoor air is an important contributor to human exposure to VOCs, we do not suggest that ambient VOC concentrations be ignored. Reducing outdoor VOC concentrations results in public health benefits. First, outdoor air has been shown to infiltrate indoors, adding to the indoor pollutant concentration (Lewis 1991). It is reasonable to assume that ambient concentrations represent minimum exposure for a number of toxic air pollutants. In this study, measured indoor and outdoor concentrations for carbon tetrachloride, ethylbenzene, MTBE, styrene, perc, TCE, and xylenes were significantly positively correlated. Therefore, lowering outdoor concentrations would reduce indoor and personal VOC levels. In addition, controlling outdoor VOCs prevents the formation of secondary air pollutants such as ozone.

Exposure is the link between the release of a toxic agent into the environment and subsequent disease in humans. Accurate exposure estimates are critical inputs to risk assessment in evaluating the severity and probability of health impact. Measured and/or modeled ambient pollutant concentrations are appropriately used as surrogates of human exposure in risk assessment. Researchers need to be cognizant of the limitations of this approach, however, and work diligently to improve the accuracy of these exposure surrogates to best inform policy. The marriage of personal exposure monitoring and risk assessment, even on a limited scale, would help identify the weaknesses of models and surrogates of exposure in estimating cumulative risk, suggest improvements in these models, and possibly reduce some of the current uncertainty associated with risk estimates.

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