Communities have long been concerned about the environmental health and environmental quality of their neighborhoods. Community-based exposure assessments have the potential to be an effective way to address these concerns. The success of such studies depends critically on the effective translation and communication of study results back to the study participants and the community. In this article we describe the communication approach applied as part of the South Baltimore Community Exposure Study. Specifically, in conjunction with collecting measurements, we asked the community to define questions they wanted answered and the way in which they wanted to receive study results. To meet their needs, we applied the risk assessment framework. The approach we developed helped residents interpret exposure assessment measurements and gave them the raw materials to effect change in their community. The risk-based approach to presenting participant and community results provides the means to move beyond traditional reporting of concentration values in three important ways. First, risk takes into consideration toxicity, thereby enabling a dialogue about community health concerns. Second, risk provides a common denominator so that exposure and risk can be compared and priorities identified. Third, exposure and risk can be summed, thereby meeting the community’s need for information regarding cumulative exposure. This approach may be a useful model for other researchers conducting exposure assessments in response to community concerns. Key words: personal exposure monitoring, risk communication, urban communities, volatile organic compounds.

Effective communication of results to policy makers, if they so choose. The approach presented here was applied successfully in conjunction with the South Baltimore Community Exposure Study. In this article, we describe the context of the South Baltimore study and the communication approach developed, concluding with a discussion of the lessons learned.

Community Context
The South Baltimore Community Exposure Study was conceived and implemented in response to community concerns about exposures to volatile organic compounds (VOCs) from industrial and mobile sources. The South Baltimore, Maryland, communities of Brooklyn, Brooklyn Park, and Curtis Bay are located primarily in the southeastern quadrant of the City of Baltimore. A significant number of Baltimore’s heavy industrial operations are located in South Baltimore, proximate to residences. These industrial facilities both improve understanding of the results and gives communities the resources necessary to communicate the implications of the results to policy makers, if they so choose.

We dedicate this article to D. McGuigan, a South Baltimore community leader who fought generously and tirelessly for community environmental health concerns.

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emit 360,479,759 pounds of pollutants into the environment annually, placing South Baltimore 12th among U.S. communities, as defined by in the top 100 ZIP codes, for total pollutant releases (Environmental Defense 2001). The communities’ industrial air toxic pollutant burden is compounded by intense mobile source emissions from major interstate highways that are also in close proximity to residential areas.

According to the 2000 U.S. Census, approximately 24,000 people live in South Baltimore; 80% are white, 15% African American, 2% Asian, 2% Latino, and 1% are 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/year (U.S. Census Bureau 2003).

Over the past two decades, a number of community leaders from South Baltimore neighborhoods have been fighting against the increased industrialization of their community, asserting that pollution associated with local industries is at least partly responsible for high rates of cancer and other serious health problems within the community. These concerns led the U.S. EPA to work with community leaders to conduct a risk-based screening analysis of toxic air pollutant concentrations, including VOCs and metals, measured at a local air monitoring station. This pollutant-by-pollutant analysis revealed that levels of ethylbenzene, 1,3-butadiene, carbon tetrachloride, chromium, hydrochloric acid, manganese, and methyl chloride exceeded the U.S. EPA’s cancer-risk screening levels and warranted further investigation (U.S. EPA 2000b).

Cumulative risk, however, was not assessed. U.S. EPA researchers struggled with deciding on the format to present the results to the larger community (U.S. EPA 2000b). The project ended without community-wide dissemination of results and recommendations. Therefore, for some community leaders, these past efforts by the U.S. EPA and similar efforts by the state environmental agency (Maryland Department of the Environment 1996) did not adequately answer their questions about levels of pollutants to which residents are actually exposed and the impact of exposure to more than one air pollutant.

Community leaders approached researchers at the Johns Hopkins Bloomberg School of Public Health seeking assistance in finding out “what is in the toxic soup that makes up the air that we breathe” (D. McGuigan. Personal communication). Through initial discussion with community leaders, it became clear that many residents were particularly concerned about whether their proximity to intense industrial emissions was leading to elevated exposure levels, and about the potential for those exposures to affect their health. As a result, we engaged with the community to design an exposure study to measure VOC exposure levels for a randomly selected population of nonsmoking adults and a subsequent communication approach that focused on expression of the population-level risks associated with the exposure levels monitored.

Thirty-seven adult residents and a few children from South Baltimore used passive air sampling badges to measure personal exposures for 3 days, in conjunction with outdoor and indoor residential air monitoring. The analysis focused on the concentrations of 11 VOCs (Table 1). More detail on the study design and monitoring results are described in Buckley et al. (2003).

**Process**

We began the communication process by partnering with a well-recognized community organization, Concerned Citizens for Better Brooklyn. The president of that association was instrumental in the design and implementation of the exposure study and the communication strategy. Before initiating the field study, two meetings with community residents were held to discuss the type of environmental study that would be conducted and to clarify expectations, particularly that this was not a health study. These meetings, which a total of approximately 50 residents attended, also alerted residents that study personnel would be recruiting in the neighborhoods. The community association helped organize and publicize the community meetings held. They also helped us identify an individual from the community to work (as a paid employee) on the study as a recruiter, who also proved instrumental in applying her community outreach experience on educational issues in reviewing our communication instruments for appropriate literacy level.

It was important that the community itself define how it would like the results presented. A community advisory committee (CAC) made up of four local leaders from each of the South Baltimore communities and one delegate of the Maryland state legislature served this function. In addition, study participants were interviewed about their information needs at the conclusion of monitoring. All of the subjects participating in the study expressed interest in both their individual and the community-wide results. The CAC and study participants both then raised additional questions during the study, including how the specific air pollutants monitored affect or have affected community health, whether community members were exposed to these pollutants other than through air, whether they were at a high level of exposure, and what they could do to reduce those exposures. In addition, participants who also agreed to have their children’s exposure monitored raised questions about how exposures could affect their children’s health. The underlying theme of these questions was the need for a public health interpretation of the exposure monitoring results. Despite very limited resources to take on this task, we worked successfully with community leaders to develop a communication strategy that addressed many of their concerns.

The challenge was how to go beyond simply presenting exposure results, toward providing information that could support individual and community action to improve the environmental health and quality of South Baltimore neighborhoods. We discussed scientific and resource limits. For example, regarding children’s health, we explained that the number of children monitored was too small to allow us to understand the implications for children specifically; the monitoring results would allow us to determine only whether children’s exposures were likely to be very different from those of adults. We continued to reinforce these messages and to manage expectations regarding the results throughout the planning and communication process.

**Table 1. Presentation format for summary of personal exposure results.**

| Air pollutant | Population participant exposure monitoring results | Comparison with personal exposures measured in other states | Cancer risk per million concentration | Noncancer risk comparison with U.S. EPA reference concentration |
|--------------|---------------------------------------------------|---------------------------------------------------------|--------------------------------------|---------------------------------------------------------------|
| Benzene      | 3.8                                               | Same                                                    | 30                                   | Below                                                         |
| Carbon tetrachloride | 1.25                                           | No comparison                                           | 19                                   | Below                                                         |
| Chloroform   | 2.17                                              | Same                                                    | 50                                   | Below                                                         |
| Ethylbenzene | 4.24                                              | No comparison                                           | 2                                    | Below                                                         |
| Methylene chloride | 1.5                                               | No comparison                                           | 1                                    | Below                                                         |
| Methyl tert-butyl ether | 7.8                                              | No comparison                                           | 2                                    | Below                                                         |
| Tetrachloroethylene | 1.6                                              | Low                                                     | 9                                    | Below                                                         |
| Toluene      | 127                                               | High                                                    | 2                                    | Below                                                         |
| Trichloroethylene | 1.18                                            | No comparison                                           | —                                    | Below                                                         |
| Styrene      | 1.26                                              | Same                                                    | 3                                    | Below                                                         |
| Xylenes      | 1.45                                              | —                                                       | —                                    | Below                                                         |
| Cumulative health risks | 117                                           | —                                                       | —                                    | Below                                                         |

*Exposure data are examples only. *Comparisons to NHESXAS Region 5 Study: low (< 25th percentile), medium (25th–75th percentile), high (> 75th percentile), no comparison (these pollutants were not measured). *Participants were asked, “Assuming a population of 1 million people had the same exposure as you, the risk estimates would be as follows ... .”*
Through discussions, we realized that residents were really asking for an understanding of what the U.S. EPA and others define as “cumulative risk” (Corburn 2002; Fox 2002; Morello-Frosch 2000, 2002). Specifically, instead of only evaluating exposure on a traditional pollutant-by-pollutant basis, the community leaders and the study participants were looking for an evaluation of simultaneous health implications, particularly for cancer, of exposure to the air pollutants monitored. Jointly, we defined the goals of the communication strategy to provide the community with a) an awareness of toxic air pollutants, b) a health risk interpretation of exposure measurements, c) information around which community residents could organize to improve the air quality of their neighborhoods, d) advice for individual actions to reduce exposures, and e) results in a timely manner to both individual participants and the community at large. Because the CAC was concerned that the participants receive their individual reports as soon as the data were available, we agreed to take a phased approach for implementing the communication strategy. Specifically, we developed participant reports first and distributed them as soon as data were available. The community-wide report was disseminated only after all of monitoring had been completed and data analyzed.

As part of our discussion with the CAC, we reviewed the components and assumptions often employed in risk assessment, such as adult body weight and inhalation rates used to estimate a dose and the application of data from animal studies where extrapolation from higher dose to lower dose is done. The CAC felt we should pursue the health risk interpretation as long as we were clear about the caveats when we distributed results to participants. We met regularly with the CAC to go over communication strategy (timing, format, and content) and discuss progress in the study.

**Risk Assessment Approach**

In response to the challenge set before us by the community, we reviewed the literature regarding risk communication and community-based research in search of a suitable model. Akland et al. (1997) reported personal exposure measurements for a variety of contaminants in Lower Rio Grande Valley by comparing individual measurements with values from other exposure assessment studies, as well as with Texas Effects Screening Levels—concentrations equivalent to a 1 in 10,000 cancer risk level. We drew on this approach, comparing individual and community exposure measurements to the U.S. EPA’s National Human Exposure Assessment Survey (NHEXAS), which was conducted in 1995 across six midwestern states (Illinois, Indiana, Ohio, Michigan, Minnesota, and Wisconsin; Clayton et al. 1999). We then developed risk-based estimates for the target population based on the South Baltimore exposure measurements and toxicity values from the database developed by the U.S. EPA for their Cumulative Exposure Project (Caldwell et al. 1998; Morello-Frosch et al. 2000; Woodruff et al. 1998, 2000). To meet the CAC goals, the approach was extended to address cumulative risk.

Risk assessment is a way of organizing information that bridges science and policy. It is an accepted way to consider the potential toxicity of a given pollutant to estimate the likelihood that a population exposed to that level of pollution will experience a given health effect (National Research Council 1983; U.S. EPA 1986, 1999). The potential toxicity values that we used were the unit risk estimates (UREs) associated with carcinogens and the reference concentrations (RfCs) associated with noncancer effects for each of the pollutants monitored in the South Baltimore study. 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-year exposure (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). The URE values in this database follow the U.S. EPA default assumption: In the absence of model-specific information, the slope is based on a no-threshold model. UREs based on animal data are the upper 95% confidence bound of the estimated cancer potency or slope of the dose–response curve. The URE is a plausible upper-bound estimate of the risk (i.e., the risk is likely to be lower, but may be greater; U.S. EPA 1999). For air pollutants, the URE is typically expressed as excess cancer risk per microgram of pollutant per cubic meter of air.

When the URE is multiplied by the lifetime average daily exposure to a carcinogen in the population, the result is the upper-bound probability of cancer. However, when a URE is based on human data it is typically defined as the maximum likelihood estimate, and that represents a “best estimate” of the dose response in the occupational study population, and is somewhat less conservative than upper-bound estimates (Woodruff et al. 2000). Inhalation cancer potency estimates for two of the pollutants in this present analysis—benzene and chloroform—are based on human data. For example, if a population of one million people were exposed to benzene at a level of 3.8 µg/m³ over their lifetimes, multiplying the exposure by the URE for benzene of 7.8 × 10⁻⁶ risk/µg/m³ would result in a risk of approximately 30/1 million people exposed. Or in other words, 30 people are estimated to develop cancer as a result of their lifetime exposure.

For noncancer effects, we employed the U.S. EPA’s default assumption that, in the absence of better information, the dose–response model has a threshold (i.e., an exposure level exists where no health effects occur). Therefore, it is not possible to calculate a URE. The RfC is an estimate of a continuous inhalation exposure to human populations (including sensitive subgroups) that is likely to be without an appreciable risk of ill health effects during a lifetime (Caldwell et al. 1998). The U.S. EPA’s standard practice is to use the most sensitive target organ for a given pollutant to estimate the RfC. For noncancer risk, we calculated hazard indices (HIs) by dividing the exposures by RfCs. For example, assuming a community-average toluene exposure of 127 µg/m³, the HI would be 127 µg/m³ divided by toluene’s RfC of 400 µg/m³ (i.e., 0.31). The example exposure level is 30% of a level that is expected not to be associated with a health effect.

This risk-based approach was adapted for interpreting the population-based implications of individual participant exposure monitoring results. The risk assessment paradigm is meant to assess population-level risk. By presenting the individual-level exposure in the context of statements such as “assuming that a population of a million individuals had the same exposure as yours, we would predict x effects/cases,” we allow participants to move past individual exposure measurements to an understanding of potential implications for their community. In the past, exposure researchers have been reluctant to do more than simply provide individuals with their exposure results.

To meet the community’s interests in risk from exposure to multiple chemicals, cumulative cancer risks associated with exposures to all of the target VOCs were calculated. The approach involved summing the compound specific cancer risks estimates for all known, possible, and probable carcinogens. Given that there are limited data to establish relationships between multiple exposures and health effects, we followed the currently used default assumption that cancer risks are additive (U.S. EPA 1999, 2002).

We assessed cumulative noncancer risks by aggregating the HIs across the VOCs that affect the same target organ. Aggregation in this way produces a “target-organ-specific hazard index” (TOSHI), defined as the sum of HIs for individual VOCs that affect the same organ or organ systems (U.S. EPA 2001).

**Interpretation**

Our approach for interpreting the results from the risk analysis was based on the recent air toxics work by the U.S. EPA and others, specifically the U.S. EPA’s Cumulative
Exposure Project and National Air Toxics Assessment (Caldwell et al. 1998; U.S. EPA 2001; Woodruff et al. 1998, 2000) in which VOC exposures posing a one-in-a-million cancer risk or more were interpreted as posing possible public health concerns. A one-in-a-million cancer risk as a health benchmark is consistent with provisions in the 1990 Clean Air Act Amendments, sections 112(f) and 112(c), which allow hazardous pollutant emission source categories to be exempted from regulation when posing less than a one-in-a-million lifetime risk to the most exposed individual (Clean Air Act of 1990).

For noncancer hazards, HIs > 1 were flagged to indicate that the VOC concentration exceeded the RfC and may be of public health concern. If the HI was ≤ 1, no harm was expected because the exposure was below the threshold for an adverse effect (Caldwell et al. 1998).

Whereas the Clean Air Act establishes a one-in-a-million cancer risk benchmark for single chemical pollutant emissions, there is no guidance for interpreting cumulative risk resulting from multiple pollutant emissions. We used the one-in-a-million cancer risk benchmark to interpret risk from exposure to multiple VOCs because the CAC position was that exposures should pose negligible risk to be protective of the vulnerable members of the community (e.g., children, the elderly, impoverished, those without health care).

Presentation Format

Individual-level results communication. We communicated the exposure measurement results and risk interpretation to the study participants during home visits to deliver the written reports and discussed the findings with each participant. The individual-level reports included seven types of information: a) actual personal exposure concentrations that we measured, b) exposure comparisons of individual results with results from other studies reported in the literature, c) risk-based interpretation based on the individual exposure results, d) general information on potential health effects of VOCs, e) general information on VOC sources, f) individual-level indoor/outerdoor ratios, and g) local, state, and federal resources for understanding and reducing exposure and risk. Examples of some of the actual written materials that were distributed to the study participants are shown in Table 1 and Figures 1–3.

Individual reports were prepared using a narrative, tables, and graphs. Exposure results were presented in concentration units (micrograms per cubic meter), as shown in Table 1. In presenting individual risk estimates, we explained in writing and orally the limitations of the exposure data and the risk estimates. The major uncertainties and underlying assumptions associated with estimating risk that we discussed with study participants included the following:

- Risk estimates were based on single 72-hr measurement of exposure, which may not be representative of annual exposures or lifetime exposures.
- The toxicity data were based on animal or human studies that used VOC exposures much higher than levels typically measured in a community setting.
- Risk was estimated only for the few pollutants that we measured—there are other air pollutants, such as metals, that may present additional risk for health problems (U.S. EPA 2000b).
- Risk was estimated only for inhalation exposures. Personal inhalation exposures represent VOCs from all sources, indoor, and outdoor sources. Food, water, and soil pathways were not assessed.
- Risk estimates were not designed to be applied to individuals—no personal information about demographic characteristics, lifestyle factors, other exposures, or preexisting conditions was used to formulate the risk estimates.
- Risk estimates provided are only validly applied when thinking about a large number of people who had the same exposures.
- We provided advice to study participants on how best to approach the risk estimates. We were very careful to point out that the risk estimates did not mean that individuals would develop cancer or other serious health effects: Risk estimates based on participants’ personal exposures were not described or promoted as “individualized” risk predictions. Rather, we focused on how the risk-based approach can be used as a basis for understanding the relative importance of exposure to various VOCs, which would in turn help individual study participants and the community leaders set priorities in reducing exposures.

As shown in Table 1, cumulative cancer risk for the VOCs reported was well above the one-in-a-million benchmark, approaching the risk level of 1 in 10,000 that the U.S. EPA generally views as warranting further action to reduce exposures (Caldwell et al. 1998). Typical text in the participants’ report to help interpret Table 1 included the following:

Based on your personal monitoring results, we estimate a cumulative cancer risk of 117 in 1 million. This means if a population of 1 million had the same exposure level as you, 117 people may develop cancer. This however does not mean that you will develop cancer. A cancer risk number above 1 in 1 million is an indication that you might want to look at ways to reduce or eliminate your exposures. There are no governmental standards as to what human exposure level to these pollutants should be. This report provides suggestions on what you can do to reduce exposure for you, your family and your community. Please note there are many causes of cancer including lifestyle, diet, genetic makeup and family history, possible interacting with environmental exposures.

Cumulative cancer risk estimates were discussed in conjunction with Figure 1, highlighting that the VOCs, among those that were measured for that individual, presented the most risk and therefore should be targeted for exposure reduction. In the example shown in Figure 1, benzene, carbon tetrachloride, and chloroform present the greatest risks. Therefore, the study participant would be advised that exposure reduction for these VOCs would yield the greatest benefit in risk reduction. Conversely, cumulative risk for serious health effects other than cancer associated with the all of the VOCs measured was low; that is, the cumulative HI or TOSHI levels were well below 1 (Figure 2).

Figure 1. Summary of cumulative cancer risk and apportionment among the VOCs. MTBE, methyl tert-butyl ether. (A) Shows which air pollutants are contributing the most to cancer health risk from the pollutants we measured. The bigger the slice in the pie chart, the bigger the contribution. (B) Shows how small environmental pollution as is a contributor to cancer in general compared with other known causes of cancer such as smoking, diet, family history, and occupation. Cancer health risk due to the air pollutants we measured contributes a small part to the pollution risk factor overall, which is just 2% of all cancers. (B) Is reprinted from Doll and Peto (1981), with permission from the Journal of the National Cancer Institute.
To provide a context for the risk estimates, the sizes of other important cancer risk factors [based on research by Doll and Peto (1981)] were presented in a pie chart format juxtaposed to the cumulative VOC cancer risk pie chart (Figure 1). Specifically, Doll and Peto (1981) used evidence from epidemiology to estimate the proportion of annual cancer mortality in the United States that might have been caused by or avoided by factors such as diet, tobacco smoke, occupational exposures, and environ-mental pollution. Based on their analysis of cancer mortality data at that time, on a popu-lation level, between 2 and 4% of cancer deaths may result from exposures to toxic chemicals in the workplace or environmental exposures.

The indoor/outdoor VOC ratio was pre-sented in individual participant reports. Figure 3 illustrates typical ratios observed for South Baltimore homes. This illustration was impor-tant in educating individuals and the commu-nity that many of the VOCs of concern in industrial emissions also have indoor sources. Although it is not possible to quantitatively determine the indoor source contribution without a corresponding measure of air exchange rate, this illustration provides a gen-eral indication of the indoor and outdoor source contribution; for example, there is no evidence of an indoor source of carbon tetrachloride, whereas in the case of chloroform, indoor sources dominated. This analysis complements both the exposure comparison (with NHEXAS) as well as the risk interpretation by providing individuals with specific information about how and where to target exposure reduc-tion efforts (i.e., at the home, community level, or both) for exposures or risks of concern. For instance, carbon tetrachloride is a chemical whose use has declined over time since it was banned from consumer products in the early 1970s (U.S. EPA 2001). However, it is an environmentally persistent compound. According to the U.S. EPA, the main source of exposure to carbon tetrachloride is “background” levels in ambient air from past emissions (U.S. EPA 2001). In this example, ambient concentrations of carbon tetrachloride drive personal exposure and risk. Elevated chloroform levels are found mainly indoors, resulting from chlorinated household cleaning products and off-gassing from chlorinated tap water. Exposure to benzene results from both mobile and industrial sources. We focused on inhalation sources of these VOCs, given the community’s interest in air pollution exposure. We explained that inhalation was the primary means of exposure for most VOCs monitored. For some VOCs, such as chloroform, that occurs as a by-product of disinfection in drinking water, ingestion can provide an additional important route of exposure.

To help individuals understand and inter-pret measured indoor and outdoor concentra-tions and personal exposures, we presented their results relative to normative exposure distribu-tions. VOC exposure results from NHEXAS were included as part of the tabular summary of individual participant exposure measure-ments (Table 1). This comparison allowed study participants and the community to evaluate whether their exposures were high or low relative to those of other communities. We color coded and assigned a qualitative descriptor of “low” to the individual partici-pant VOCs exposures if they were less than or equal to the 25th percentile, of “medium” if they were between the 25th and 75th percentiles, and of “high” if they were greater than the 75th percentile measured in NHEXAS.

As part of the written materials that were given the participants, we included information on sources of the pollutants, how to reduce exposures to the air pollutants, and health effects that have been observed through animal testing and human studies for each of the pollu-tants that were monitored. This information was obtained from the Agency for Toxic Substances and Disease Registry’s ToxFAQs (ATSDR 2001) and the U.S. EPA’s Integrated Risk Information System website (U.S. EPA 2000c) and Air Toxics website (U.S. EPA 200a). For instance, in the case of benzene, we listed leukemia as a health effect that has been associated with high levels of benzene from occupational exposures. We discussed sources of benzene, which included automobile exhaust; environmental tobacco smoke; paint; industries that make detergents, drugs, dyes, and rubber; and solvents. Suggestions for reducing personal benzene exposures that are in the individual’s control included “Don’t let people smoke around you,” “Stand back from the gas pump when refueling,” “Call or write your state and federal government representa-tive and tell them you are concerned about air quality in your neighborhood,” and “Work with local city council and state agencies about rerouting traffic away from residential areas.” In addition, we provided ideas for community organizers to reduce exposures, such as educat-ing neighbors about indoor and outdoor air pollution, and identifying businesses in the community that use hazardous compounds and launching a campaign to persuade them to find less toxic alternatives. We provided a list of consumer guides to toxic chemicals that could be obtained from the local library. Because the 2000 U.S. Census data indicated that most South Baltimore residents did not have more than a high school education (U.S. Census Bureau 2003), our written materials were crafted to read at the eighth- to tenth-grade level.

Communication of community-level results. Four months after the study was completed, we held a community meeting to discuss the overall study results, including health risk interpretations based on mean exposures for the population. About 40 resi-dents attended the meeting. We followed the same approach for interpreting and presenting risk for the community meeting as with the individual reports. We presented the same suggestions for individual-level and community-level actions to reduce VOC exposures as described above, which were drawn from citizen guides on toxic chemicals (Dadd 1997; Harte 1991), Environmental Defense’s Scorecard (Environmental Defense 2001), and activities we were aware of taking place in other communities (e.g., Roxbury, MA, and Harlem, NY). When asked whether the local industries were responsible for the exposures, we had to inform the audience that VOCs are emitted from a variety of sources that include both indoor and outdoor sources. As discussed with the community before beginning the study, available technology would not allow us to identify a single source responsible for their exposures. Therefore, we could not conclude that the local industry was solely responsible for the exposures we measured. This was a disappointment for some community leaders and residents.
After the community meeting, we mailed a study results fact sheet to all the participants and community leaders, which included the same elements as the individual report (exposure results and indoor/outdoor ratios, population risk estimates, sources of exposures, health effects information, and exposure reduction strategies).

Evaluation and Discussion

**Individual level.** The risk-based approach and the communication materials that we prepared were well received by the study participants. Individual participants expressed gratitude that we took the time to return to their homes to give them their results. For example, one male participant stated that our returning to his home to review his results was reassuring and meant that we were not the stereotypical university researchers only concerned about “getting” our data. By hand-delivering the participant reports, we provided an opportunity for dialogue and to build trust in the community. Specifically, the interactive approach allowed the study participants to ask clarifying questions and for us to receive instant feedback on the communications materials, and thus for us to ensure that our messages were being appropriately interpreted. Most participants did not “overreact” to the risk estimates. Rather, participants focused on the information we provided on sources and ways to reduce exposure.

For instance, a comment we received about reducing chlorine use and chloroform exposure during a home visit was, “When I was growing up we used Bon Ami and that doesn’t contain chlorine. We should go back to using that. But I have not seen Bon Ami in the stores in a long time.” This comment highlights the difficulty in making what might appear to be “simple” changes in daily activities to reduce exposure. Although upper-middle-class families might have access to organic or “green” alternatives, for those with less access to specialty stores and their prices, alternatives may not be readily available.

In another example, delivering the results report allowed us the opportunity to inquire about unusually high exposure measurements and learn about occupational exposures. When we discussed exposure measurements with one elderly male participant, he informed us that he used solvents to clean off grease and oil from his arms and hands after working on car and motorcycle engines with his son. We suggested that he find an alternative for cleaning up after working on his hobby. Again, we would have felt better if we had had an easily available substitute to recommend.

For the South Baltimore study, we relied largely on anecdotal feedback from participants and the CAC. For the most part, our goals for the communication effort were met. A formal evaluation was not conducted. However, evaluating the audiences’ (participants, CAC, and the community at large) response to the message and their comprehension of our materials would be an excellent component to add to our approach for future applications. Additionally, a follow-up survey of the study participants and community leaders on what actions, if any, were taken to reduce exposures would help to ascertain whether changes occurred as a result of the communication effort.

**Community level.** Although the individual results communication effort was positively received, the community-level communications effort met with mixed reactions. Community meeting attendees and some members of the neighborhood association were disappointed that we did not pinpoint a particular local industry as the source of VOC exposures and health risks. This reaction was not necessarily surprising, even though we worked hard to manage expectations and provide the resulting information in a format that would allow the community to advocate for themselves once we completed the study. But despite disappointment on the part of some residents, there was interest at the meeting in the suggestions we presented on community-level action, including neighborhood associations conducting outreach and education to residents about indoor and outdoor air quality in general; the value in identifying consumer products that also emit VOCs and influence indoor air quality; and identifying small businesses that may also contribute to outdoor air pollution, such as the numerous autobody repair shops in South Baltimore.

Our experience at the community level provides another example of the limitations associated with conducting health and exposure investigations in response to community concerns, and the tension between science and politics. As Ng and Hamby (1997) describe, risk communication can take on many forms:  

- a) information and education, where people are informed and educated about risks and risk assessments in general;  
- b) behavior changes and protective action, which encourage risk reduction behavior by trying to influence the perceptions of the audience;  
- c) disaster warning and emergency information; and  
- d) joint problem solving and conflict resolution, which involve the public in risk management decision making and in resolving health, safety, and environmental controversies (Ng and Hamby 1997).

We made a conscious decision to put ourselves somewhere in the middle of what we consider a continuum between traditional field research and science-based advocacy. Our approach provided information, education, and suggestions for risk reduction. Although we did not meet some residents’ desire for a clear industrial pollutant/health risk link, the communication approach presented here enables researchers to put exposure data into a context and format that facilitates the community’s ability to credibly represent the study’s results. Communities benefit from a fuller understanding of exposure research results as they move forward to “confronting risk” and engaging stakeholders to attempt to cope with the physical or medical and social consequences posed by the environmental health problem (Leviton et al. 1998). Researchers should be aware of the options available and select the level of community interaction that is appropriate to the problem at hand and can be implemented given the resources and experience of both the community and the researchers.

In conclusion, the risk-based approach to presenting participant and community results provides a means to move beyond traditional reporting of concentration values in three important ways. First, risk takes into consideration toxicity, thereby enabling a dialogue about community health concerns. Second, risk provides a common denominator so that exposure and risk can be compared and priorities identified. Third, exposure and risk can be summed, thereby meeting the community’s need for information regarding cumulative exposure. This approach can be applied to other community-based exposure studies. Access of a CAC is extremely important to gaining insight into community sociopolitical dynamics and information needs. The level of detail/complexity for presenting a health risk interpretation will depend on the risk literacy of the study participants and community. In our case, we relied on the fact that a previous risk-based study was conducted in the community, which allowed us to pursue the risk assessment approach.
to participants in an environmental exposure study: insights from clinical ethics. Environ Res 80(2 pt 2):S223–S229.
Doll R, Peto R. 1981. The causes of cancer: quantitative estimates of avoidable risks of cancer in the United States today. J Natl Cancer Inst 66:1191–1308.
Environmental Defense. 2001. Scorecard. Available: http://www.scorecard.org [accessed 30 November 2001].
Fox MA. 2002. Evaluating cumulative risk assessment for environmental justice: a community case study. Environ Health Perspect 110(suppl 2):203–209.
Harte J. 1991. Toxics A to Z: A Guide to Everyday Pollution Hazards. Berkeley, CA:University of California Press.
Israel BA, Schulz AJ, Parker EA, Becker AB. 1998. Review of community-based research: assessing partnership approaches to improve public health. Annu Rev Public Health 19:173–202.
Leviton LC, Needleman CE, Shapiro MA. 1998. Confronting Public Health Risks: A Guide to Everyday Pollution Hazards. Berkeley, CA:University of California Press.
Maryland Department of the Environment. 1996. Maryland Air Quality Data Report 1996. Baltimore, MD:Air and Radiation Management Administration.
Metzler MM, Higgins DL, Beeker CG, Freudenberg N, Lantz PM, Senturia KD, et al. 2003. Addressing urban health in Detroit, New York City, and Seattle through community-based participatory research partnerships. Am J Public Health 93:803–811.
Morello-Frosch R, Pastor M Jr, Perras C, Sadd J. 2002. Environmental justice and regional inequality in southern California: implications for future research. Environ Health Perspect 110(suppl 2):149–154.
Morello-Frosch RA, Woodruff TJ, Axelrad DA, Caldwell JC. 2000. Air toxics and health risks in California: the public health implications of outdoor concentrations. Risk Anal 20:273–291.
National Research Council. 1983. Risk Assessment in the Federal Government: Managing the Process. Washington, DC:National Academy Press.
Ng KL, Hamby DM. 1997. Fundamentals for establishing a risk communication program. Health Phys 72:473–482.
O’Fallon LR, Deary A. 2002. Community-based participatory research as a tool to advance environmental health science. Environ Health Perspect 110(suppl 2):155–159.
Schulte PA, Sweeney MH. 1995. Ethical considerations, confidentiality issues, rights of human subjects, and uses of monitoring data in research and regulation. Environ Health Perspect 103(suppl 1):69–74.
U.S. Census Bureau. 2003. United States Census 2000. Washington, DC:U.S. Census Bureau. Available: http://www.census.gov/main/www/cen2000.html [accessed 4 September 2003].
U.S. EPA. 1986. Guidelines for carcinogen risk assessment. Fed Reg 51:33992–35003.
———. 1999. Proposed Guidelines for Carcinogen Risk Assessment. NCEA-F-0644. Washington, DC:Office of Research and Development, National Center for Environmental Assessment.
———. 2000a. Air Toxics Website. Washington, DC/U.S. Environmental Protection Agency. Available: http://www.epa.gov/tri/ate/hapindex.html [accessed 26 November 2001].
———. 2000b. Baltimore Community Partnership Air Committee Technical Report. Community Risk-Based Air Screening: A Case Study in Baltimore, MD. EPA 744-R-00-005. Washington, DC:Office of Pollution Prevention and Toxics.
———. 2000c. Integrated Risk Information System (IRIS). Washington, DC:U.S. Environmental Protection Agency. Available: http://www.epa.gov/iris/index.html [accessed 1 November 2001].
———. 2001. National-Scale Air Toxics Assessment for 1996. Research Triangle Park, NC:Office of Air Quality Planning and Standards.
———. 2002. External Review Draft Framework for Cumulative Risk Assessment. EPA/630/P-02/001A. Washington, DC:Risk Assessment Forum.
Wakefield J. 2003. Growing pains for environmental justice movement. Environ Health Perspect 111:A38–A49.
Weed DL, McKeeown RE. 2003. Science and social responsibility in public health. Environ Health Perspect 111:1804–1808.
Woodruff T, Axelrad D, Caldwell J, Morello-Frosch R, Rosenbaum A. 1998. Public health implications of 1990 air toxics concentrations across the United States. Environ Health Perspect 106:245–251.
Woodruff T, Caldwell J, Cogliano V, Axelrad D. 2000. Estimating cancer risk from outdoor concentrations of hazardous air pollutants in 1990. Environ Res 82:194–206.