actions (e.g., exposure assessments, epidemiologic investigations, exposure registries, surveillance) that should be pursued in communities. Even with the efforts to measure exposure, the important question about latent, adverse health effects remains unanswered. As public health professionals in environmental health committed to protect the health of communities living near hazardous waste sites, we therefore strongly disagree with the authors’ statement that “No further assessment of the health risks is needed.”

Legator and Strawn also make two other points to which we wish to respond. They state “If information on each site were available in sufficient detail, populations from exposed communities could be aggregated or combined. Unfortunately, the data that would help determine the multiple sites for which similar effects could be anticipated does not yet exist.” The ATSDR agrees with the approach of combining populations from sites with reasonably common characteristics; this is the exact approach taken in our National Exposure Registry program (8). As an example, the ATSDR Subregistry of Persons Exposed to Trichloroethylene consists of a registry of about 5000 persons in 13 communities. Chemical-specific exposure subregistries provide ATSDR with health information on persons with common chemical exposures and also provide a means for communicating health information back to the registrants.

In addition, more recently, ATSDR has developed the database necessary to combine site-specific information. The database is called HazDat. It contains all the environmental contamination, toxicology, and human health effects data from about 1300 Superfund sites. Recently, in conjunction with four state health departments, we conducted a study of lead exposure and toxicity in four different populations that were identified through use of HazDat. We anticipate releasing HazDat to the public later this year.

Ascertaining the dangers to public health of hazardous waste sites, together with implementing public health actions to protect against the effects of hazardous substances, is a challenging responsibility. The ATSDR’s public policies and public health practices must be based on sound scientific principles and data. This must involve the communities affected by releases from waste sites and other sources of hazardous substance releases. We believe the statutory mandates in the Comprehensive Environmental Response, Compensation, and Liability Act that bear on public health are consistent with sound public health practices. The translation of these mandates into actions, to some extent in ways inferred by Legator and Strawn, is ATSDR’s challenge. We believe we have made progress, but much awaits.

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REFERENCES

1. Susten AS. Findings from ATSDR’s health assessments. J Environ Health 53:17-21 (1992).
2. National Research Council. Environmental epidemiology: public health and hazardous wastes. Washington, DC: National Academy Press, 1991:
7-8.
3. Johnson BL. A precis on exposure assessment. J Environ Health 55:6-9 (1992).
4. Johnson BL, Jones DE. ATSDR’s activities and views on exposure assessment. J Exp Anal Environ Epidemiol (suppl 1): 1-17 (1992).
5. Agency for Toxic Substances and Disease Registry. Public health assessment guidance manual. Chelsea, Michigan: Lewis Publishers, 1992.
6. U.S. GAO. Public health assessments—Incomplete and of questionable value. Washington, DC: U.S. Government Accounting Office, 1991.
7. ATSDR. ATSDR’s response to the GAO report on health assessments. Atlanta, Georgia: Agency for Toxic Substances and Disease Registry, 1991.
8. ATSDR. Policies and procedures for the National Exposure Registry. Atlanta, Georgia: Agency for Toxic Substances and Disease Registry, 1987.

Industrial Sources of Benzene Exposure?

In volume 82 of EHP, Wallace presented some of the results of the EPA’s Total Exposure Assessment Methodology (TEAM) study in an attempt to identify the major sources of exposure to benzene (1). He contended that the results showed “... that personal activities or sources in the home far outweigh the contribution of outdoor air to human exposure to benzene” (L: 166). Two tables of statistical data were presented to demonstrate this point.

We have previously commented on the severe problems affecting the benzene data for New Jersey (2) and on the confounding effects of the apparent inversion that occurred concerning the data for Los Angeles, California (J: Tables 1 and 2; LA1). We believe that further comment is necessary regarding the

| Source | Arithmetic mean | Geometric mean | Median |
|--------|-----------------|----------------|--------|
| Night  |                 |                |        |
| Upwind | 9.3             | 2.85           | 5.81   |
| Downwind | 20.7          | 12.3          | 13.0   |
| Day    |                 |                |        |
| Upwind | 10.1            | 3.03           | 5.87   |
| Downwind | 16.4          | 8.38          | 11.0   |

Table 1. Comparison of data on levels of benzene (μg/m³) from different segments of the TEAM study

the study, equal in size, from an area upwind of potential industrial sources has apparently not been reported except in the final report prepared for EPA (6). Table 1 compares data from the upwind segment of the study to data from the downwind segment of the study. Outdoor benzene levels are not reported because they were obtained by a different sampling technique.

There is no serious question about the values from the second group of data from Los Angeles (LA2) and from Antioch-Pittsburg, California (AP), but subsequent comments and conclusions regarding benzene exposure or breath differences should be reconsidered based only on results from the remaining total of 30 smokers and 89 nonsmokers. These remaining subjects can hardly be considered to be representative of the U.S. population.
The correlations between passive smoking and benzene are very weak. This weakness is further demonstrated in another EPA report (7) that shows when the New Jersey and the California data for matched indoor and outdoor samples are regressed, only the first group of data for Los Angeles (LA1) show a significant correlation with the presence of a smoker in the home, and then only with p = 0.1 (1), the probability that a smoker in the home was a significant variable. A later study conducted in Los Angeles (8), continuing primarily with the same homes at two different times of the year, was unable to show a significant difference between benzene in the air in the homes of smokers and those of nonsmokers. This result held true regardless of the season, the time of day, and the area of the house that was studied. What this later study did show was that the location of the outside samplers was important because there was not a good correlation between fixed or area samples and individual samplers located outside homes. This implies that the location of individual samples outside of homes is critical. We know of no published work in which this variable has been studied. Hence, when examining earlier data, the emphasis should be on matched indoor–outdoor results, and even then one should not be overly confident in the results.

Wallace’s Figure 2, which compares West German data to U.S. data, appears to contain an error. Krause et al. (9) give the concentrations of benzene in West German homes as 9.3 and 6.9 µg/m³ for smoking and nonsmoking, respectively, not 11 and 6.5 µg/m³ as quoted by Wallace. We are also suspicious of the practice of comparing two different statistics, i.e., U.S. geometric means and West German medians.

We are aware of the breath levels of benzene in self-reported work exposure as discussed by Wallace et al. (10). Those results, obtained by the TEAM study in New Jersey when exhaust fumes infiltrated the van containing the spirometer, indicated that nonsmokers exposed to passive smoke more than 50% of the time at work could probably reduce their exposure by becoming smokers! Neither the experimental conditions during the New Jersey study nor the finding about the equivalent workplace exposure to benzene inspires much faith in the passive smoking conclusions from the TEAM study.

Readers with a need to incorporate the results of the TEAM study into their own findings would be well advised to critically review all of the TEAM study reports to determine when problems detracted from the significance of the study’s conclusions and to what extent this occurred. Some important unanswered questions remain regarding the true impact of proximity to industrial sources, the potential for indoor sinks to ballast the effects of outdoor concentrations of benzene, whether smokers and nonsmokers have different lifestyles, and how representative these data are of the subjects’ average day. Until these questions can be answered more conclusively, one can put little faith in risk analyses that use TEAM data.

We contend that the problems enumerated above invalidate the benzene exposures and risks shown in Wallace’s Table 3. In addition to the problems with the appropriateness of the bases for the numbers in the calculations, examination of the numbers used in the exposure budget and risk analysis reveals some contradictory and unsupported assumptions. For example, the text appears to say that two-thirds of the population are passive smokers, which we take to be 160 × 10⁶ individuals. The footnotes to Table 3 imply that 80% of the population is exposed to environmental tobacco smoke, which we take to be 190 × 10⁶. Table 3 claims a population at risk of 200 × 10⁶. Footnote e to Table 3 and the text imply that the average increase in benzene due to environmental tobacco smoke is 3 µg/m³ for 17 hr spent at home and at work. But the data in the EPA report indicated that there was essentially no difference between the homes of smokers and nonsmokers in the second Los Angeles and Antioch-Pittsburg studies (10), and these are the only data not subject to serious questions. Finally, the variables presented in Wallace’s Table 3 are not independent. The 53 × 10⁶ smokers must be contained within the 200 (2) × 10⁶ passive smoker population.

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REFERENCES

1. Wallace LA. Major sources of benzene exposure. Environ Health Perspect 82:165–169 (1989).
2. Rosebrook DV, Worm GH. Personnel exposures, indoor-outdoor relationships, and breath levels of toxic air pollutants measured for 355 persons in New Jersey (letter to the editor). Atmos Environ (in press).
3. Pellizzari ED, Perritt R, Hartwell TD, Michael LC, Sheldon LS, Spacino CM, Whitmore R, Leninger C, Zelton H, Handy RW, Smith D, Wallace LA. Total exposure assessment methodology (TEAM) study: Elizabeth-Bayonne, New Jersey; Devils Lake, North Dakota; and Greensboro, North Carolina, vol 2. EPA 600/C87/002b. Washington, DC: U.S. Environmental Protection Agency, 1987:662.
4. Pellizzari ED, Perritt R, Hartwell TD, Michael LC, Sheldon LS, Spacino CM, Whitmore R, Leninger C, Zelton H, Handy RW, Smith D, Wallace LA. Total exposure assessment methodology (TEAM) study: Elizabeth-Bayonne, New Jersey; Devils Lake, North Dakota; and Greensboro, North Carolina, vol 2. EPA 600/C87/002b. Washington, DC: U.S. Environmental Protection Agency, 1987:appendix B.
5. Pellizzari ED, Perritt R, Hartwell TD, Michael LC, Sheldon LS, Spacino CM, Whitmore R, Leninger C, Zelton H, Handy RW, Smith D, Wallace LA. Total exposure assessment methodology (TEAM) study: Elizabeth-Bayonne, New Jersey; Devils Lake, North Dakota; and Greensboro, North Carolina, vol 2. EPA 600/C87/002b. Washington, DC: U.S. Environmental Protection Agency, 1987:270–272.
6. PEI Associates, Inc. Baltimore total exposure assessment methodology (TEAM) study. Final report prepared for EPA under contract no. 68-02-4406. Research Triangle Park, North Carolina: U.S. Environmental Protection Agency, 1988.
7. Wallace L. The total exposure assessment methodology (TEAM) study: summary and analysis, vol 1. EPA 600j6-87/002a. Washington, DC: U.S. Environmental Protection Agency, 1987:appendix B.
8. Pellizzari ED, Michael LC, Perritt K, Smith DJ, Hartwell TD, Sebestik J. Comparison of indoor and outdoor toxic air pollutant levels in several southern California communities. Final report prepared for EPA under contract no. 68-02-4544. Research Triangle Park, North Carolina: U.S. Environmental Protection Agency, 1989.
9. Krause C, Mailahn W, Nagel R, Schulz, Seifert B, Ulrich D. Occurrence of volatile organic compounds in the air of 500 homes in the Federal Republic of Germany. In: Indoor air ‘87: proceedings of the fourth international conference on indoor air quality and climate, vol 1. Berlin: Institute for Water, Soil and Air Hygiene, 1987:102–106.
10. Wallace LA, Pellizzari E, Hartwell TD, Perritt R, Zienegus R. Exposures to benzene and other volatile compounds from active and passive smoking. Arch Environ Health 42:272–279 (1987).

Cigarettes: Point Source for Benzene Exposure?

In their letter, Rosebrook and Worm erroneously state that Tables 1 and 2 of my article in volume 82 of EHP (1) include values from the second batch of samples from New Jersey (which were taken in the summer of 1982). However, footnote a of Tables 1 and 2 clearly indicate that these values are for the fall of 1981. No data from the summer of 1982 are included in either table.

Rosebrook and Worm refer to the “confounding” effect of the inversion affecting the first group of Los Angeles, California, data. Such inversions, however,