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 (LA) show a significant correlation with the presence of a smoker in the home, and then only with $p = 0.1$ (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 $\mu g/m^3$ for smoking and nonsmoking, respectively, not 11 and 6.5 $\mu g/m^3$ 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 unsurmountable assumptions. For example, the text appears to say that two-thirds of the population are passive smokers, which we take to be 160 x 10^6 individuals. The footnotes to Table 3 imply that 80% of the population is exposed to environmental tobacco smoke, which we take to be 190 x 10^6. Table 3 claims a population at risk of 200 x 10^6. Footnote c of Table 3 and the text imply that the average increase in benzene due to environmental tobacco smoke is 3 $\mu g/m^3$ 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 x 10^6 smokers must be contained within the 200 (2) x 10^6 passive smoker population.

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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 (7) 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,
are fairly common in Los Angeles in the winter months, and any estimate of exposure must include both high and low exposure seasons. We made a return visit to Los Angeles in 1987 (2,3) and observed similar differences between high winter and lower summer exposures (Table 1).

The Maryland data (4) were collected by two contractors using two different sampling techniques (Tenax and evacuated canisters). I chose to report only the data collected by the same sampling technique and the same contractor as in all other TEAM studies, in the belief that these data would be the most comparable to data from other cities. However, because Rosebrook and Worm dispute the central finding of increased personal and indoor exposures compared with outdoor exposures, it is appropriate to compare indoor values collected with the canister to outdoor values collected with the canister (Table 2). These data, like those from the Tenax samplers, showed increased indoor values compared to outdoor.

It is unclear why Worm brings up the additional Maryland information only to then suggest discarding both it and the original Maryland data.

The North Carolina data are indeed more uncertain than the remaining data due to high and variable background contamination of benzene. If the North Carolina data are removed, the conclusions of the paper would be based on a total of about 600 different persons instead of about 620 and would be unchanged. Because all of these persons were selected on a probability basis to represent much larger populations, the total population represented is about 600,000 persons, even without the 130,000 residents of Greensboro, North Carolina.

Rosewood and Worm state that "...important unanswered questions remain regarding the true impact of proximity to industrial sources. ..." The New Jersey study involved 350 persons, many of whom lived close (<1 km) to major petroleum refining and petrochemical facilities, and many others lived farther away. No difference in exposure of the two groups was evident. For both groups, personal exposures were approximately triple the outdoor concentrations, putting a stringent upper limit on the portion of exposure that could be provided by the nearby industrial sources. Both the Los Angeles and Antioch-Pittsburg, California, locations were selected for proximity to major petroleum refining operations, and again no impact of these facilities on personal exposure could be discerned.

Recently, one additional large-scale personal monitoring study in Valdez, Alaska (5) has come to a similar conclusion: the single major source in that community (a pipeline terminal facility) contributes about 90% of the benzene emissions in that area but contributes only about 10% of residents’ exposure to benzene. That estimate was based on a tracer gas emitted from the facility, providing additional objective evidence for the conclusion. The Valdez study also found that indoor sources and personal activities accounted for the majority of personal exposure to benzene, concluding that its findings confirm those of the TEAM studies.

Rosewood and Worm state that the correlations between passive smoking and benzene are weak, quoting from a table based on a subset (about one-quarter) of the available data. However, when all the data are included in the regressions, much stronger correlations are noted. Tables A-1 and A-2 of Wallace (6) show that the 258 New Jersey residents with one or more smokers in their homes had about double the daytime benzene personal air concentrations (p < 0.0006) and about 68% increase in their overnight benzene personal air concentrations (p < 0.006) compared to the 90 persons with no smokers in their homes. Table A-11 shows a similar increase (about 50%) in the February 1984 overnight benzene personal air concentrations for the Los Angeles residents with a smoker in the home (p < 0.001). The increase was not observed in California homes in May and June of 1984; however, windows in these homes were generally left open for ventilation and therefore had high air exchange rates, reducing the concentration of benzene in indoor air. I have discussed the relationship of benzene to active and passive smoking in greater detail elsewhere (7).

The median values for smoking and nonsmoking homes quoted by Rosewood and Worm from Krause et al. (8) were based on only 230 of the 488 homes eventually monitored. The values used in my paper (1) were based on the full 488 homes. Geometric means in the TEAM studies have consistently been very close to median values, as would be expected for log-normally distributed data, and as can be seen in the accompanying tables.

The estimates of the number of passive smokers were indeed inconsistent, ranging from 160 to 200 million. This number includes active smokers because all active smokers are also passive smokers, breathing increased benzene from their own sidestream smoke. If the lower figure were accepted, the estimated nationwide benzene exposure budget would be lowered by about 1%. In view of the increasing restrictions on smoking in the workplace and in public facilities, a still lower estimate of the
number of passive smokers might be appropriate today.

If we omit the breath measurements for New Jersey, which may have been elevated for both smokers and nonsmokers, the mean breath levels measured at other locations are remarkably consistent at about 12–14 μg/m³ for smokers and 1–2 μg/m³ for nonsmokers. It is impossible to observe these data without concluding that smoking is the most powerful single source of exposure to benzene for many millions of persons.

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