more detailed explanation of the basis for MCIA’s position.

Woodruff et al. (1) incorrectly suggest that industrial air emissions of methyl chloride present a significant health risk. On the basis of 1990 data for the Toxics Release Inventory (TRI) and the Cumulative Exposure Project (CEP), Woodruff et al. purport to identify listed hazardous air pollutants (HAPs) that are present in the environment above levels of concern based on cancer and noncancer effects. The authors further state that methyl chloride is one of eight pollutants identified as having “modeled concentrations exceeding the benchmark concentrations for cancer in 100% of the census tracts” (1).

These statements are inaccurate for the following reasons. First, methyl chloride air emissions and resulting concentrations should not be compared to a cancer health benchmark because available data are not sufficient to conclude that methyl chloride poses a human cancer hazard. Methyl chloride has been classified by the U.S. Environmental Protection Agency (U.S. EPA) as only a Group C possible human carcinogen (2); this is based on no human data and insufficient animal data. Further, the International Agency for Research on Cancer (IARC) (3) found that the evidence of carcinogenicity of methyl chloride to humans and to animals is inadequate; therefore, IARC classifies methyl chloride in Group 3 (not classifiable). A U.S. EPA Scientific Peer Review Panel (4), convened for a rulemaking proceeding under 112(g) of the Clean Air Act, agreed that compounds classified as Group C (possible) carcinogens should not be grouped with “known” and “probable” human carcinogens. The available data simply are not sufficient to justify evaluating or classifying methyl chloride based on a perceived cancer hazard.

Second, when background concentrations from natural sources are removed from the analysis, methyl chloride emissions do not exceed benchmark levels in 100% of the census tracts. Perhaps up to 99% of ambient air concentrations of methyl chloride are due to releases from natural sources, rather than releases from manufacturing and use (5). Although in their Table 2 Woodruff et al. (1) acknowledge that the alleged exceedances for methyl chloride are due almost entirely to background concentrations, rather than man-made sources, they nevertheless purport to identify “HAPs representing the highest potential health risks” with the idea that

Given the almost insignificant amount of methyl chloride emissions from industrial sources, efforts to reduce methyl chloride emissions from industrial sources will not meaningfully reduce ambient concentrations of methyl chloride. Woodruff et al. misleadingly suggest otherwise.

Woodruff et al. (1) also misleadingly suggest that the CEP represents the U.S. EPA’s final analysis. This is not correct. The CEP is an analysis performed by the U.S. EPA that compared modeled ambient air concentrations of HAPs in urban census tracts to chronic health effects benchmarks. HAPs were ranked according to the number of urban census tracts in which the modeled concentration was above the health benchmark. Much of the needed health effects information was previously compiled for the U.S. EPA’s proposed rule making under Section 112(g) of the Clean Air Act Amendments of 1990. In the 112(g) proposal, the U.S. EPA proposed a relative hazard ranking of all HAPs. However, this rule making was never finalized and the U.S. EPA never responded to public comments submitted or to the views expressed by the Scientific Peer Review Panel concerning the inappropriateness of classifying Group C carcinogens with Group A and Group B carcinogens. Because the analysis and conclusions contained in the CEP were never subject to peer review, it is therefore not a reliable source of information, nor does it represent the U.S. EPA’s final analysis of the data.

The editors of EHP have an obligation to ensure that the statements contained in its publications are factually accurate and not misleading. This obligation is paramount, particularly when a paper is drafted by a U.S. EPA staff scientist and therefore has the potential to be mistakenly viewed by readers as an official U.S. EPA position. The misleading statements included in the paper are of particular concern because they have been mistakenly relied upon by the public and other publications. For example, Rachel’s Environment & Health Weekly (6) appears to have relied on the EHP paper as the basis for a statement that

EPA … published a report in 1998 saying that 100% of the outdoor air in the continental U.S. is contaminated with eight cancer-causing industrial chemicals at levels that exceed EPA’s “benchmark” safety standards.

The paper further identified methyl chloride as a “carcinogen” that is “known to cause cancer.” Woodruff et al. (1) is cited as the reference for these misleading statements.

We request that such misleading information not be published again in subsequent papers appearing in EHP and that this letter be published to provide the public with a more accurate presentation of the facts concerning methyl chloride. If you have questions concerning these comments or require further information, please contact me.

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Air Toxic Concentrations: Response

We appreciate Browning’s interest in our paper “Public Health Implications of 1990 Air Toxics Concentrations across the United States” published in the May 1998 issue of EHP (1). In this paper, we compared estimated concentrations of 148 air toxics, derived from dispersion modeling of air toxics emissions, to previously defined benchmarks for cancer and noncancer effects.

As stated in the paper, the goal of the analysis was to provide a relatively comprehensive assessment of the potential public health impacts of air toxics (referred to in the Clean Air Act as “hazardous air pollutants” or HAPs) based on available information. To conduct this analysis, we used emissions data from stationary and mobile sources in an atmospheric dispersion model to estimate 1990 outdoor concentrations of 148 HAPs for every census tract in the contiguous United States. For many HAPs, the estimated concentrations also included a background concentration, which represents the impacts of long-range transport, resuspension of historical emissions, and nonanthropogenic sources, that would be present without local anthropogenic 1990 emissions. Background concentrations were based on measurements taken in locations
remote from the impact of local anthropogenic sources and were applied uniformly to all census tracts.

The estimated concentrations were compared to previously defined benchmarks for cancer and noncancer effects (2). For this analysis, a HAP was considered to be a potential human carcinogen if it was classified by the U.S. EPA (3) as Group A (known), B (probable), or C (possible), or by the International Agency for Research on Cancer (IARC) as Group 1 (known), 2A (probable), or 2B (possible). The description of the IARC categorization for carcinogens is found in the preamble of each IARC Monograph (4). This is consistent with the prescribed risk-based standards for risks resulting from exposures to known, probable, and possible carcinogens in the Clean Air Act Amendments of 1990 [section 112(f)]. The benchmark concentration for carcinogens was set equal to a concentration associated with a one-in-a-million cancer risk for lifetime exposure. We then assessed the number of exceedances, or census tracts with estimated concentrations greater than the one-in-a-million benchmark, for each HAP.

The initial assessment of the carcinogenicity of methyl chloride was reported in a document prepared by the U.S. EPA Office of Research and Development (5). In this document methyl chloride was classified as a group C carcinogen (possible human carcinogen) on the basis of kidney tumors found in mice exposed via inhalation. Therefore, we considered methyl chloride to be a possible human carcinogen on the basis of the U.S. EPA classification. The Section 112(g) technical support document (6) referred to by Browning did not classify any HAPs as carcinogens, but rather adopted existing agency assessments for use in its hazard ranking. The procedures for adopting assessments for the Section 112(g) document were peer reviewed by an external expert panel, but this panel did not engage in further review of individual pollutant assessments that had already been through various forms of external and internal peer review. The analysis of Caldwell et al. (2) referenced in our paper built on and extended the principles used in the Section 112(g) document (6) to assemble hazard information on air toxics. One of these principles was to use existing reviewed toxicologic data. Although it was beyond the scope of our paper (1) to review the toxicologic data for each HAP, the general assessment procedures, as well as the specific methyl chloride weight-of-evidence classification and benchmark concentration, were presented by Caldwell et al. (2). Although the U.S. EPA classification of methyl chloride differs from that of IARC, the tiering approach adopted by Caldwell et al. considered the U.S. EPA classifications first and then used IARC assessments for pollutants lacking a U.S. EPA classification.

Browning correctly quotes the "Results" of our paper (1): methyl chloride was one of several pollutants that had modeled concentrations exceeding the benchmark concentrations for cancer in 100% of the census tracts.

Immediately after this statement, we explained that this result was due to the fact that the estimated background concentrations (applied to every census tract) alone were greater than the benchmark concentrations for these pollutants. We further explored the results for these pollutants by considering the number of exceedances when background is disregarded. Table 2 in our paper (1) clearly displayed our finding that when the background concentration was disregarded, estimated 1990 methyl chloride concentrations exceeded the cancer benchmark in about 110 (out of 60,000) census tracts in the contiguous United States. This information is all clearly presented in the same paragraph that contains the statement quoted by Browning.

Our main objective in conducting this analysis was to estimate concentrations experienced in ambient air, regardless of source, to help define the potential scope of impacts on public health. As we stated in the paper,

Future regulatory and scientific activities can begin to focus on these pollutants to address and further evaluate their public health significance.

In our paper (1), we did not recommend any specific course of action for methyl chloride or any other pollutant.

We agree that greater confidence should be placed in results for pollutants classified as known and probable human carcinogens than for those classified as possible human carcinogens. However, as we have stated in our work, we believe it is important to include as much information about the potential hazards of as many HAPs as possible. To do otherwise would be to initially assume that there is no risk and would not reflect prudent public health practice. As we stated in our paper (1), it is appropriate to follow up with further research to investigate these relationships more closely.

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