

## Pesticide Spraying and Health Effects

I noticed with interest the article "Pesticide Spraying for West Nile Virus Control and Emergency Department Asthma Visits in New York City, 2000" by Karpati et al. (2004). I am a physician who treats hundreds of patients with chronic illness from chemical overexposure. Many of these patients have toxic encephalopathy, reactive airway disease, and other chemically induced organ system damage. When my patients become ill from pesticide spraying, they usually do not head for an emergency room, where they typically experience long waits in an environment containing germicidal residue, scented products, carbonless copy paper, hospital linens with heavy fabric softener, and other exposures. In addition, they have learned from experience that emergency department personnel often do not understand their condition and do not know how to treat it. Thus your survey, while with admirable intent, greatly underestimates the problem of respiratory exacerbation from West Nile virus pesticide use. Many of my patients have experienced severe neurologic and respiratory exacerbations as well as other organ system damage, such as significant increase in liver enzymes, from exposure to residue from pesticide spraying for West Nile virus. In addition, it is my understanding that these pesticides are not effective for controlling adult mosquitoes and that the Centers for Disease Control and Prevention and other authorities recommend larvae control. The extent of exacerbation of illness caused by pesticide use for West Nile virus control is likely greater than the number of cases of West Nile virus.

Persons who are at increased risk for symptom exacerbation from pesticide spraying such as that used for West Nile virus control include individuals with migraines, chronic sinus problems, asthma, reactive airway disease, autoimmune diseases (many of which are exacerbated by pesticide exposure), and conventional allergies (Kipen et al. 1994). There is increased respiratory inflammation with conventional allergies, and pesticides more readily enter the body because the barrier function of the respiratory tract is further compromised. In addition, Karpati et al. (2004) failed to take note of the U.S. Environmental Protection Agency (EPA) final report "Principles of Neurotoxicity Risk Assessment" (U.S. EPA 1994). This document confirmed the lack of a blood-brain barrier between the nose and the brain, so that pesticides readily enter the body through the nose and pass directly to the brain. This report further confirmed the unusual vulnerability of the

brain to neurotoxicants: pesticides are lipophilic and therefore seek out lipid tissue such as the brain, and because the brain has unusually long neurons, repair of damage in the neurons occurs much less readily than in other body cells.

Other groups at increased risk of pesticides are those with chronic obstructive lung disease, toxic encephalopathy, and neural degenerative diseases. Pyrethroid pesticides are significant neurotoxins (Eells et al. 1992; McDaniel and Moser 1993; Tippe 1993; Vijverberg and van den Bercken 1990), and because they are increasingly replacing organophosphates, they now account for a large proportion of the pesticide-induced chronic illness among my patients. Emergency room visits are merely the tip of the iceberg, and patients with many of these disorders usually avoid the emergency room. Thus, the use of emergency rooms is not a sensitive indicator of body damage from pesticides.

In my experience, the use of nebulized glutathione, the major antioxidant and major detoxifying agent of the body (Klaassen et al. 1986), when combined with lipoic acid, helps to improve an individual's ability to detoxify (Packer et al. 1995); lipoic acid reactivates glutathione in lipid- and water-based tissues. Also, nebulized glutathione combined with adequate buffered vitamin C reactivates glutathione in water-based tissues.

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## Pesticides and Health Effects: Karpati et al. Respond

In her letter, Ziem raises the issue of exacerbations of respiratory illness and other health effects of pesticide exposure that we did not measure in our study (Karpati et al. 2004). Our analysis was designed to evaluate only whether a population-level effect on emergency department visits, specifically on asthma and other respiratory illnesses, was evident following pyrethroid pesticide spraying. Similar study designs, despite their limitations, have proven to be sensitive methods of identifying population-level health impacts from exposure to criteria air pollutants from exposure to unusual events, such as smoke from forest fires. Moreover, our analysis did identify adverse population-level health effects of elevated ozone and particulates. As we noted in our discussion, the results of the analysis for pesticide exposure do not rule out the possibility that certain individuals might have been affected by exposure to the agent. Also, our focus was on emergency department visits, which generally signify more serious illness, although in urban neighborhoods even milder illnesses are often treated in such settings. However, if, in fact, certain individuals experienced asthma exacerbations following exposure, we believe our study demonstrates that their number was small enough that it did not result in a population-level increase in emergency department visits for asthma and chronic obstructive pulmonary disease.

In our analysis we evaluated only respiratory complications of pesticide spraying to control West Nile virus, and we did not purport to measure possible neurotoxic or other nonrespiratory effects. Also, we did not evaluate the efficacy of pesticide spraying for mosquito control or its cost-benefit ratio with regard to pesticide-related health effects.

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## “Epidemiology of Health Effects of Radiofrequency Exposure”

In a recently published review (Kundi et al. 2004) on mobile phone use and cancer, we concluded that

Epidemiological studies that approached reasonable latencies [time period between first exposure and diagnosis] consistently observed elevated risk for the development of neoplastic diseases.

This assessment is distinctly different from the main message of the review from the International Commission for Non-Ionizing Radiation Protection (ICNIRP; Ahlbom et al. 2004). The authors stated that

Results of these studies to date give no consistent or convincing evidence of a causal relation between RF [radiofrequency field] exposure and any adverse health effect.

Although the use of subjective terms is sometimes unavoidable in the context of risk assessment (e.g., to evaluate sufficiency of evidence), the decision whether or not it evidence is “convincing” should be left to the reader. Furthermore, what constitutes consistent evidence or the lack of it is unclear when the scope is as broad as the authors implied in their reference to a “causal relation between RF exposure and any adverse health effect.”

This review of epidemiologic evidence addressed the issue of causation without any consideration of the concept of causation in epidemiology, and it failed in its essential task to assess the possible association between exposure to RF and health. Concerning cancer, Moolgavkar and Luebeck (2003) have shown that agents that increase the growth rate of preneoplastic cells may have a distinctly greater impact on cancer incidence than agents that induce malignant transformation. However, this holds only for agents that act for prolonged periods of time. Regarding the natural history of cancer, a noticeable effect at the population level will only occur many years (and possibly decades) after first contact with the promoting agent. Although Ahlbom et al. (2004) pointed to the insufficient latencies in epidemiologic studies, they did not draw the straightforward conclusion—to assess the relationship between the latencies covered in the studies and their outcome.

Although there is agreement between Ahlbom et al. (2004) and us (Kundi 2004;

Kundi et al. 2004) that epidemiologic studies of RF/microwave exposure generally have deficiencies concerning exposure assessment, we must not ignore that the consequence of exposure misclassification is predominantly a bias of risk estimates towards the zero hypothesis.

Another aspect that has contributed to, in our view, the inappropriate assessment of evidence is their view about the end points of the investigations. Among malignancies studied so far, the most heterogeneous group are brain tumors that comprise benign as well as malignant neoplasms with grossly different cellular origin, growth behavior, and fate. Until now no risk factor for brain tumors has firmly been established except ionizing radiation for meningioma and meningeal sarcoma and less consistently for other brain tumors. Regarding brain tumors of high malignancy, little is known about induction periods and the steps necessary to reach the final invasive state; however, case reports of glioma after sellar irradiation (Simmons and Laws 1998) suggest an average induction period of about 10 years. Therefore, because exposure started too late for an effect during initiation and because proliferation is too fast for an effect on growth rate, brain tumors of highest malignancy must be studied very thoroughly in relation to latency, which was not the case for most of the studies published so far. Disregarding these conditions will strongly dilute any possible effect.

Except for insufficient latency, other sources of possible bias were mentioned by Ahlbom et al. (2004), but again without consideration of the consequences on risk indicators. Ahlbom et al. (2004) stated that

Several of these studies did not follow workers after they left the job of interest (Garland et al. 1990; Grayson 1996; Szmigielski 1996), with the potential for bias if individuals left employment because of health problems that subsequently turned out to be due to cancer....

The presence of this bias in these studies would have reduced the power in the case of no relation between exposure and the likelihood of leaving employment due to early signs of the target disease, or it would have led to a bias of risk estimates in the direction determined by the sign of the correlation between exposure and leaving service. It is quite likely that this correlation is positive because early signs of brain tumors will create problems in radio operators and also in personnel operating and maintaining radar equipment. Hence, the consequence of the bias is either reduction in the precision or inflation of risk estimates.

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## Epidemiology of Radiofrequency Exposure: Ahlbom et al. Respond

We thank Kundi for his comments on our review of the epidemiologic literature on health effects of radiofrequency exposure (Ahlbom et al. 2004). He points out, quite correctly, that our assessment of the literature differs from the one he and colleagues have made in a previous review (Kundi et al. 2004). We do, however, stand by our judgment that the literature we reviewed offers little support for a causal relation between radiofrequency exposure and disease risk. Although quality of research varies, most of the studies we reviewed were methodologically limited and more rigorous studies are needed. It is obviously impossible to tell what more sophisticated research along the lines suggested by Kundi, and also by ourselves, will reveal in the future. We certainly agree that consideration of latency and various types of bias is important and, indeed, we did consider these at some length in our review.

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## Listing Occupational Carcinogens

The review by Siemiatycki et al. (2004) is extremely valuable, and I am sure I will refer to it often in the future. However, I would like clarification on the risk classification of some chemicals. In the text the authors state that some chemicals, such as glass wool, were downgraded in risk between 1987 and 2002, from “possible human carcinogen” (group 2B) classification, to unclassifiable (group 3). This contradicts Table 5 (Siemiatycki et al. 2004), where the chemicals are listed as “possible human carcinogens” and the authors cited the 2002 volumes of the *IARC* (International Agency for Research on Cancer) *Monographs*; this gives the impression that this is the most up-to-date classification.

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## David Rall and the National Toxicology Program

Concerning the 25 year history and milestones of the National Toxicology Program (NTP), McGovern (2004) failed to acknowledge the huge conceptual and leadership contributions provided by David P. Rall (1926–1999) in the creation, development, and continuing achievements of the NTP (Huff 2000). Of course, Rall was the individual most responsible for conceiving, nurturing, and establishing the National Institute of Environmental Health Sciences (NIEHS) as a world-recognized leader in environmental health sciences. The NTP, a natural and independent partner of the NIEHS, was the innovative idea of Rall, who with a few other like-minded collegial giants in the fields of public and occupational health, recognized the need to better coordinate the disparate and often redundant toxicology and health hazard identification activities in the Department of Health and Human Services.

Among others, Congressman David Obey (Wisconsin) was convinced by Rall and key colleagues that a coordinated national program was needed to better promote the health of the American people by protecting them from exposures to hazardous chemicals in the workplace, environment, and home. Obey was instrumental in getting Congress to endorse this need for a national program and, after Rall's untimely death, encouraged Congress to name the main structure housing NIEHS/NTP in Research Triangle Park, North Carolina, as the David P. Rall Building.

Rall was supported in this innovative and monumental NTP-forming effort by several other distinguished pioneers in environmental health research and public health, including Eula Bingham, then director of the Occupational Safety and Health Administration; Joseph Califano, then Secretary of the Department of Health, Education, and Welfare (renamed the Department of Health and Human Services); Donald Fredrickson, then director of the National Institutes of Health; Donald Kennedy, then director of the Food and Drug Administration; Cesare Maltoni, then director of the Bologna Centre for the Prevention and Detection of Tumours and Oncological Research; Norton Nelson, then director of the Institute of Environmental Medicine (renamed the Nelson Institute of Environmental Medicine) New York University School of Medicine; Irving Selikoff, then director of the Mt. Sinai Medical Center in New York City (renamed Mount Sinai-Irving J. Selikoff Clinical Center for Occupational and Environmental Medicine); Lorenzo Tomatis, then chief of

the Unit of Chemical Carcinogenesis, International Agency for Research on Cancer (later director of IARC); and Arthur Upton, then director of the National Cancer Institute.

Also, during that NTP-formative era, Rall helped establish the 1978 Public Law that initiated the innovative *Report on Carcinogens* (Huff 1998). Now in its eleventh edition, the RoC documents 246 chemicals, groups of chemicals, or mixtures known or anticipated to cause cancer in humans. Much earlier, in 1972, Rall originated the pivotally directed and most frequently referenced environmental journal, *Environmental Health Perspectives*. Of course, during his illustrious career as physician, assistant surgeon general, scientist, and staunch public health advocate, Rall made many more and varied contributions to basic sciences and public health (Hinson 2000; Huff 2000, 2002; Rall 2000).

David P. Rall, a dedicated physician and scientist, is among a relatively small group of exceptional public servants who have had a deep and lasting positive impact on human health by showing us how to understand and improve the environment in which we live, work, and play. To many of us who joined with him to make a safer environment, we clearly recognize the void he has left.

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## Potential Selection Biases

In a recent article we reported an association [adjusted odds ratios (ORs) in the range of 2–3] between the concentrations of specific phthalates in dust from children's bedrooms and doctor-diagnosed disease among children (Bornehag et al. 2004). This study has been

accused of "selection bias problems" by the American Chemistry Council Phthalate Esters Panel (American Chemistry Council 2004) and the European Council for Plasticisers and Intermediates (2004).

A major criticism of the study is based on the assumption that families with allergic members change their flooring from carpets to a hard floor [e.g., polyvinyl chloride (PVC)]. If this is true, our findings would be biased (i.e., families with allergic members would have relatively more PVC flooring and would thus be exposed to higher concentrations of phthalates). However, very few families in Sweden today have wall-to-wall carpets; in total, 1% of the homes in this study reported having such carpets, two cases and two controls. Given that Swedish homes already have hard floors, there is no obvious reason why allergic families would change from one hard floor (e.g., wood) to another hard floor (e.g., PVC).

Still, there is some bias in the study. The case-control study in question is based on a cross-sectional baseline survey. The baseline questionnaire showed that there were no indications of selection bias among cases concerning self-reported flooring materials in the home (Bornehag CG, Sundell J, Sigsgaard T, Janson S, unpublished data). However, among controls, a main difference was a significant overrepresentation of wood flooring among participating families compared with nonparticipating families. Furthermore, on the basis of inspectors' observations, parents sometimes misclassified the type of flooring material in their home. Quite often parents classified PVC as linoleum or cork.

Presuming the misclassification of linoleum and cork is similar among participating and nonparticipating families, then the distribution of flooring materials can be recalculated. The distribution of PVC flooring after such a recalculation becomes 59.8% (including cases), 61.2% (nonparticipating cases), 51.1% (included controls), and 59.5% (nonparticipating controls). Consequently, cases report PVC flooring slightly more often than controls. However, regarding hard flooring, we found no bias between the groups because 99% of the families had hard floors.

Even if PVC-flooring is associated with phthalates in dust, it is not simply a proxy for phthalate exposure. Several observations support such a statement. First, our study indicated that there are other significant indoor sources for phthalates because, in the absence of PVC flooring, the dust concentrations of plasticizers are still significant (Bornehag CG, Sundell J, Lundgren B, Weschler CJ, Sigsgaard T, Hagerhed-Engman L, unpublished data). Second, the association between doctor-diagnosed disease and the concentration of specific phthalates in dust was much stronger than the association between disease and PVC flooring

(Bornehag et al. 2004). Third, the correlation between the concentration of different phthalates was rather weak ( $r < 0.35$ ) (Bornehag et al. 2004). There are still other observations that indicate that the reported association is not due to selection bias. Fourth, an underrepresentation of about 9% of PVC flooring among controls cannot explain reported ORs in the range of 2–3. (However, the earlier reported association between PVC flooring and case status, OR 1.59, is overestimated because there is selection bias regarding PVC flooring among controls.) Fifth, when we included in the analyses only buildings with PVC in the child's bedroom, the association between butyl benzyl phthalate (BBzP) in dust and rhinitis and eczema remained (Bornehag et al. 2004). In such a restricted analysis, the potential selection bias has been eliminated. Finally, we found different associations for different phthalates: di(2-ethylhexyl phthalate (DEHP) was associated with asthma; BBzP was associated with rhinitis and eczema; and DnBP was not associated with asthma or allergies.

In summary, for the reasons stated above, we judge that our reported association between phthalates in dust and asthma/allergic symptoms among children is not a consequence of either selection bias or active avoidance of specific flooring materials because of allergic disease in the family.

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