Delaney Still the Best Protection
The December 1995 article on pesticides in food (EHP 103:838–843) misstates the basic issues around the Delaney Clause and misses the point. While this federal regulation has its limitations, such as it does not address all food types such as raw fruits and vegetables (and these limitations should be corrected), contrary to what the article says, Delaney offers the best possible protection of public health, including children who are more susceptible and vulnerable, because it says that no amount of a carcinogen in food is acceptable. There can be no better protection than not allowing any carcinogen in food regardless of who eats it. All the alternatives to Delaney incorporate some form of risk assessment which will attempt to define some level of “acceptable risk.” These are the approaches that will suffer many of the problems described in the report, not Delaney. Delaney needs fixing to expand its reach. It should not be replaced by risk assessment approaches that will be more subjective, more vulnerable to assumptions and uncertainties, and ultimately less protective of public health not only for children, but for everyone.

Stephen U. Lester
Citizens Clearinghouse for Hazardous Waste
Falls Church, Virginia

1-Hydroxypyrene as an Indicator of Pyrene Exposure

The article by Øvrebo et al. in the September 1995 issue of EHP (103:838–843) is a valuable contribution to environmental health efforts in eastern Europe and will no doubt serve as a benchmark in the future. We do, however, have concerns about the future correlation between pyrene in air and 1-hydroxypyrene in urine found by Øvrebo et al., which led them to conclude that pyrene in air is not a strong predictor of 1-hydroxypyrene. The basis for collection of polycyclic aromatic hydrocarbons (PAHs) using NIOSH method 5515 (7) assumes PAHs up to fluoranthene are partially retained on the filter substrate and all higher PAHs are 100% retained. Hence, the authors chose to collect only the particulate fraction of workplace and ambient air.

It is interesting to note the absence of method validation data in NIOSH method 5515 but its inclusion in NIOSH method 5506 (2). The difference in these two methods is solely the analytical method of analysis: gas chromatography in 5515 and HPLC in 5506. Inspection of measurement precision in NIOSH 5506 using spiked sampling trains in a laboratory atmosphere indicates PAHs ranging from naphthalene to fluoranthene exhibited significant volatilization and PAHs from benzo[a]anthracene to indeno[1,2,3-cd]pyrene showed no volatilization. The data for pyrene were not determined. Work by Kirton (3) has shown four-ring PAHs incompletely retained on filters with an approximate filtersorent ratio of 50:50 to 15:85 for pyrene and fluoranthene depending on the filter particulate loading (higher loadings mean more filter retention). Sampling coal tar pitch volatiles in a Söderberg potroom of an aluminum smelter, Ny and co-workers (4) found 48% of pyrene in backup XAD tubes. We fully support the use of PAH profiles as the first step in developing biological monitoring programs to address PAH exposure, but consider the effects of incomplete collection of pyrene in the sampling stage to be a majority deficiency in the design of many published studies (5–7). Correspondingly, incomplete atmospheric sampling will lead to overestimates of dermal absorption where this variable is calculated using multiple regression analysis. We would encourage (nay, insist) further sampling in a way which presents loss or nonrecovery of any target compound.

The timing of urine collection of occupational samples by Øvrebo et al. would not allow one to properly determine the level of 1-hydroxypyrene arising from pyrene exposure on the shift where personal measurements were made. The half-life of ingested pyrene was determined by Buckley and Lioy (8) to be 4.4 hr, with the maximum elimination rate occurring at 6.3 hr. Accordingly, samples should be collected 6–7 hr after the work shift. The collection of urine immediately after a single work shift does not allow adequate time for elimination of pyrene assimilated during that shift. This further factor would compound the poor correlation observed between pyrene in air and 1-hydroxypyrene.

1-Hydroxypyrene is the major pyrene metabolite in mammals; however, Grimmer (9) detected another pyrene metabolite in exposed workers: 1,2-dihydroxy-1,2-di-hydroxypyrene. This accounted for around one-fifth to two-fifths of the pyrene metabolites in a study of coke oven workers. Significant interindividual differences in phenanthrene metabolite profiles were also found. This indicates a potential genetic difference in PAH metabolite production, which may affect the levels of 1-hydroxypyrene. Knowledge of the effects of P4501A1 induction and P450 isozymes on pyrene metabolite ratios in chronically exposed humans either through occupation or smoking is needed to clarify the current role of 1-hydroxypyrene as the sole indicator of pyrene and, by default, PAH exposure.

Peter G. Knott
Capral Aluminium Limited
Kurri Kurri, New South Wales, Australia

Peter J. Kirton
BHP Environmental Health Laboratory
Port Kembla, New South Wales, Australia

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Response

In their letter, Knott and Kirton refer to the incomplete collection of pyrene on filter when collecting air samples in polycyclic aromatic hydrocarbon (PAH)-polluted atmospheres. We are aware of this phenomenon, which is discussed in a recent book by Bjøseth and Becher (1). But the correlation coefficient (R²) between pyrene in air and urinary 1-hydroxypyrene is not dependent on how complete the sampling of pyrene is as long as we sample a constant proportion of the pyrene in air. We have studied the
correlation between pyrene collected on filter and pyrene collected on XAD (2). On average, we found 1.6 μg/m³ pyrene on filter and 1.9 μg/m³ on XAD; i.e., 46% on filter and 54% on XAD. This is in good agreement with Ny et al. (3). The correlation between pyrene on filter and the sum of pyrene on filter and pyrene on XAD was 0.69. Since this is less than 1, it is likely that a better correlation between pyrene in air and urinary 1-hydroxypyrene would have been the result of using an XAD backup in our study.

The timing of urinary sampling is an important issue. However, it is impossible to select a perfect sampling time for biological monitoring. Even if you sample for 8 hr and start at a time point equal to the half-life of 1-hydroxypyrene after the start of work, some of the collected urine will contain 1-hydroxypyrene from the previous day’s exposure, and day-to-day variation may be significant. Jongeneelen (4) has studied samples collected after and before shift and found a correlation between pyrene in the air and urinary 1-hydroxypyrene both when collected after shift and before the shift the next day. The American Conference of Industrial Hygienists (ACGIH) has established biological exposure indices (BEI) for several organic compounds (but not pyrene) and suggested sampling times. The ACGIH suggested end of shift and prior to next shift as the time when sampling time is critical (3). Compromises have to be made in practical biomonitoring, but for validation of methods, 24-hr sampling of urine may be important.

We agree that 1-hydroxypyrene is the main metabolite and that other metabolites are important, but urinary 1-hydroxypyrene is a marker for PAH exposure and does not represent the total exposure. A large proportion of PAHs are excreted in feces. In a recent study of voluntary ingestion and dermal application of pyrene, less than 4.5% (ingestion) and 0.2% (dermal) of the dose was recovered in a 48-hr collection period (6). The study of the influence of genetic factors and lifestyle factors is important in validating biomarkers like urinary 1-hydroxypyrene. We are currently conducting such studies. But PAH uptake is also influenced by particle size of the PAH. The new filter cassettes called IOM (7) show that construction of a filter orifice can have a great impact on the fraction samples and may be more important than AD backup, which mostly gives a constant loss that can be corrected for.

There is still need for more validation studies of the biomarker 1-hydroxypyrene. To sum up, we would like to cite from implementation of the BEI (5): “Biological monitoring should be considered complementary to air monitoring. It should be conducted when it offers an advantage over the use of air monitoring alone.”

Steinar Øvrebø
Per Einar Fjeldstad
Elin Hegland Kure
Aage Haugen
National Institute of Occupational Health
Oslo, Norway
Ewa Grzybowska
Mieczyslaw Chorazy Institute of Oncology
Gliwice, Poland

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Radiation and Childhood Cancer

Ostensibly, the impetus for Wakeford’s review of reported associations of childhood cancers with radiation exposures (EHP 103:1018–1025) was to refute our judgment that cancer risk coefficients for low-dose exposures of populations, as officially adopted by national and international radiation commissions, have been substantially underestimated (EHP 102:656–667). We based our judgment on documented inconsistencies and omissions in the literature, as well as on discrepancies between official predictions and observed health effects among various groups, including nuclear workers and residents of radioactively contaminated areas. We reviewed a wide range of studies under low-dose exposure conditions, especially those that found radiogenic cancer risk coefficients inconsistent with official estimates for protracted low-dose exposures. These significant discrepancies between observed and expected values contradict the model assumptions made when low-dose, low-rate risks were extrapolated from primarily one-flash, high-dose effects (1,2).

Wakeford ignores these inconsistencies by neither refuting nor discussing them. His substantive criticism of our contribution is limited to just two of our reported findings, which we can accept. Yet, this in no way affects our stated conclusions:

1) We had overlooked a downward revision of the prenatal exposure risk of 20 fatal childhood cancers per 10⁴ person-cGy (3) to 12.7 cancer deaths (and 17 nonfatal cancers) per 10⁴ person-cGy, as derived from the Oxford Survey of Childhood Cancers (OSCC) (4), generally recognized as the most extensive database on childhood cancers. The main body of Wakeford’s paper, however, is a presentation of alternative radiogenic risk estimates, emphasizing those that are closer to the generally accepted norm; i.e., those derived from the A-bomb survivor data. This brief communication is not the appropriate place for a detailed evaluation of Wakeford’s selection of data. Yet, a crucial omission from his review is the evidence we referenced in our paper showing that, as a consequence of significant selection effects among A-bomb survivors, prenatal exposure risks as derived from A-bomb data are intrinsically incompatible with those based on X-ray exposures of general populations (such as the OSCC). Recently released data on early radiation injuries among the LSS survivor cohort by the Radiation Effects Research Foundation, Hiroshima, strongly confirm Stewart’s earlier conclusions about the effects of selection (5). From now on, evaluations of risk from prenatal exposures to X-rays, low-dose gamma rays, or internal radioisotopes will have to stand on their own reliability in methodology, since agreement or disagreement with the A-bomb data appears to have become irrelevant.

2) Regarding the “Gardner hypothesis” (postulating a genetic component for leukemia in young people through preconceptual parental exposure), we reviewed several findings, supportive and unsupportive. In view of the unknown contribution of internal radioisotopes to the health hazards in some of the conflicting findings, as well as confounding clustering effects of leukemia with infectious epidemics in rural areas (6), we consider the discussion as undecided at this time. In contrast to this more cautious approach, Wakeford, without presenting a plausible counter-hypothesis for either the