

## Delaney Still the Best Protection

The December 1995 article on pesticides in food (*EHP* 103:1082) 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.

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## 1-Hydroxypyrene as an Indicator of Pyrene Exposure

The article by Øvrebø 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 poor correlation between pyrene in air and 1-hydroxypyrene in urine found by Øvrebø 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 (1) 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 benz[*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 filter:sorbent 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 major 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) future sampling in a way which presents loss or nonrecovery of any target compound.

The timing of urine collection of occupational samples by Øvrebø 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 Liou (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-dihydroxypyrene. 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.

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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 Bjørseth and Becher (1). But the correlation coefficient ( $R^2$ ) 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