Environmental Tobacco Smoke

I read with interest the article from Bermudez et al., "Environmental Tobacco Smoke Is Just as Damaging to DNA as Mainstream Smoke" (EHP 102: 870–874). Environmental tobacco smoke (ETS) is a complex mixture of chemicals resulting from dilution in a confined environment of tobacco smoke. ETS has three forms: 1) sidestream smoke (SS) is produced by a cigarette during the puff intervals, 2) mainstream smoke (MS) is released by the smoker after smoke inhalation, and 3) residual mainstream smoke (RMS), which is a minimal proportion, slowly seeps from the mouth end of a cigarette during the puff intervals. Thus, ETS cannot be identified by one of these components alone. Furthermore, SS or MS determinations cannot be used as a predictor of the concentration of compounds in the ambient air because the composition and the chemical nature of ETS changes dramatically as it ages and is diluted in the environment (the same can be said regarding the prediction of ETS's effects in terms of public health). Therefore, I was rather surprised to read that ETS is equivalent to sidestream smoke (see the Introduction and Material and Methods), so the particulate matter trapped on a Cambridge filter is equivalent to ETS "tar." This statement is obviously untrue and deliberately disregards the evidence that ETS is a dilute system compared to MS and/or SS.

The in vitro tests used to monitor the adverse effects of SS-derived tar trapped on a Cambridge filter consisted of 1) rat alveolar macrophages for the measurement of the electron spin resonance (ESR) to detect the presence of a persistent radical after incubation with the tar solution, 2) isolated rat thymocytes incubated with the tar solution that were then submitted to fluorescence analysis of DNA unwinding to determine DNA damage. Both these assays gave positive results in terms of an effect of the test material employed. After having obtained these results, Bermudez et al. concluded: "to our knowledge, this is the first report of the DNA nicking capability of tar from ETS" (p. 873). I cannot agree for at least two reasons: tar was collected from SS and not from ETS, and a genotoxic effect of SS tar has been known for a while (1,2).

In the article, Bermudez et al. cite work by Hammond et al. (3) indicating macromolecular adduction in people exposed to ETS. Hammond et al. examined the relationship between quantitative measurements of 4-amino-1-phenyl-1,2-5,4 benzoquinone (4-ABP–H) in nonsmoking, pregnant women. Surprisingly, only one blood sample was collected at delivery, and a relationship was found between women exposed to ETS (monitored during the third trimester of pregnancy by a questionnaire and by wearing a monitor which sampled nicotine by passive diffusion to a filter treated with sodium bisulfate) and the level of 4-ABP-Hb adducts found at the time of delivery. The conclusion of these authors was that the increase in the levels of 4-ABP–Hb was not dramatic and that the public health significance was unclear. Bermudez et al. failed mention a number of studies aimed at detecting increased levels of DNA and hemoglobin adducts in people exposed to ETS, all with questionable or frankly negative outcomes (4–10).

I would suggest repeating the alveolar macrophage study using cells obtained by the same technique (bronchoalveolar lavage) from rats exposed to a real ETS environment, controlling certain parameters: particle concentration, particle size, and carbon monoxide. This would produce much more meaningful information. Alternatively, repeat the alveolar macrophage study using trapped particulate matter carried by persons exposed to an ETS environment and compare it with the material trapped by the filters obtained from devices carried in a smoke-free environment. Phillips et al. (11) were able to prove that, in a confined environment where smoking was permitted, only a median of 2.5% of the particulate matter trapped by portable monitors was of ETS origin.

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Assessing Chemicals for Estrogenic/Hormone-Disrupting Properties: Lessons from Carcinogenicity Assessment

Recent articles and letters in EHP (1–9) have highlighted the growing interest in chemicals that have the potential to mimic estrogens or in other ways disrupt endocrine hormone balances. The specific concerns were listed succinctly in the Wingspread consensus statement of 1991 (10). Any such “new” area of toxicology poses particular problems for those charged with assessing the safety of industrial or other environmental chemicals—all chemicals concomitantly come under suspicion, but the screening assays necessary to assess this toxic potential are usually only in the early stages of development. As a consequence, assay method development and chemical evaluations proceed in parallel, with many potential misaligns along the way. Thus, at this moment, chemical companies and commercial testing laboratories around the world face an apparent toxicological problem of undefined dimensions, but in the absence of agreed techniques by which to assess or solve it. In this situation, valuable parallels are already evident between estrogenicity testing and carcinogenicity prediction.

The field of environmental carcinogenesis was underpinned from the start by data on approximately 50 discrete chemical or environmental exposure situations in which a firm link between chemical exposure and the induction of cancer in humans was established. This reference point of stability is missing with environmental estrogens. In its place are a range of suspected associations with reduced human sperm counts and increases in the incidences of human testicular, prostate, or breast cancer. Thus, the reality or otherwise of a human problem will have to be evaluated concurrently with the development of meth-