hormone profile in men and I accordingly predicted (12) that exposure to dioxin would be associated with the production of excess female offspring. This hypothesis has since been confirmed (13).

To summarize, there are a number of forms of male exposure which are associated with significantly low offspring sex ratios. These have been replicated in the case of DBCP and non-ionizing radiation. In regard to DBCP, dioxin, and methyltestosterone, the known hormonal effects of the chemicals are consistent with my hypothesis that the testosterone/gonadotropin ratio in mammalian parents at the time of conception is causally and positively related to the offspring sex ratio (proportion male).

I suggest that offspring sex ratio is a useful non-invasive alternative to sperm counts and hormone assays as a monitor of male reproductive hazard.

THE AUTHORS REPLY

We thank Dr. James for his observations regarding our study (1). As noted in our article (2), the consistent finding of a male deficit for births fathered by men who did not use protective equipment is impressive. The reason we did not tabulate the summary measure for "any chemical exposure" with and without use of protective equipment was that the heterogeneity of exposures associated with such diverse chemical applications was thought to be less informative than the results for specific types of chemical applications. However, in response to Dr. James's inquiry, we did analyze "any chemical" with and without use of protective equipment. As noted, in making his calculation based on published results, the assumption was made that overlap based on multiple types of chemical application was similar for those who did and did not use protective equipment. For reasons that are not at all clear, there were actually more men who had applied pesticides for multiple purposes among those who used protective equipment (1,184 actual births vs. 2,370 estimated by summing across types of chemical application, for a ratio of 0.4995) than among those who did not use protective equipment (370 births vs. a sum across categories of 682, for a ratio of 0.5425). This small disparity leads to a much smaller male deficit than that estimated by Dr. James, with 181 males and 189 females born to men who did not use protective equipment, for a risk ratio of 0.97 (95 percent confidence interval 0.86–1.10).

Nevertheless, the examination of sex ratio for each type of chemical application among men who did and did not use protective equipment is more likely to be valid, though less precise, than the result for the aggregate of chemical applications. We agree that at least this component of our results lends very modest support to the hypothesis that male chemical exposures may reduce the proportion of male offspring. Other data cited by Dr. James (1) make this case much more strongly than our results.

REFERENCES


William H. James
The Galton Laboratory
University College London
Wolfson House
4 Stephenson Way
London NW1 2HE
England

REFERENCES


David A. Savitz
Kathryn M. Curtis
Tye Arbuckle
Department of Epidemiology
School of Public Health
University of North Carolina
Chapel Hill, NC 77599-7400

Diane Kaczor
Carolina Population Center
University of North Carolina
Chapel Hill, NC

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