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Sex ratio of offspring of residents of a highly polluted housing site

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Sex ratio of offspring of residents of a highly polluted housing site

Fertmann et al (1) have described the reproductive features of residents in a housing site built on a contaminant sludge layer with high concentrations of arsenic, heavy metals, dioxins, and furans. The site has now been reportedly abandoned by most families. The authors noted a significantly high sex ratio (proportion male) at birth, and they request suggestions on how best to pursue research on this matter. I should like to respond to that request.

This high sex ratio is in contrast to the significantly low offspring sex ratios of men exposed to a variety of chemicals, illnesses, and occupations. I have adduced evidence for low offspring sex ratios among men exposed to the nematocide DBCP (1,2-dibromo-3-chloropropane) (2), borates (3), and dioxin (4) and in men suffering from multiple sclerosis (2) or non-Hodgkin's lymphoma (2) and in men who experience deleterious occupational exposures such as in professional diving (2), professional driving (4), carbon-setting (4) and work in high-voltage installations (5). Thus many forms of adverse exposure are associated for men with the production of daughters.

There is now very strong evidence that the hormone levels of both parents around the time of conception partially control the sexes of mammalian (including human) offspring (2). In conformity with this hypothesis, many of the aforementioned forms of exposure to men have been documented in association with low testosterone or high gonadotrophin levels. However much less is known about the effect on the offspring sex ratio of such adverse *maternal* exposures. Women with multiple sclerosis reportedly have an excess of sons (6), and this excess may be due to the fact that stress has an opposite effect on the androgen levels of men and women since stress is known to lower men's androgen levels but is thought to raise women's levels (7). If this assumption is correct, then one can suspect that the excess of male births in the data of Fertmann et al (1) represent an adverse effect on the mothers rather than (or more than) on the fathers. This may be true, for instance, if the female residents had spent more of their time each day engaged in domestic tasks on or around the contaminated site and if the men had mainly been elsewhere (in employment or otherwise) during the day. It may also explain the cohort effect on sex ratio, that is, the fact that the most recently born children (whose mothers had ex hypothesi been exposed longer or to higher levels of pollution) had a significantly higher sex ratio than the

children who were 9—16 years of age at the time of registration.

To test these points, I suggest that the following research be considered:

1. Assays should be made of subjects' hormone levels (namely, of androgens, estrogens, gonadotropins and progesterone). Ex hypothesi, the ex-resident mothers should have (or have had) high levels of androgens or estrogens or both and possibly low levels of gonadotropins or progesterone or both. It is difficult to predict how the contaminants may have affected the ex-resident fathers. Dioxin is known to lower the testosterone: gonadotropin ratio of men (8) — but the result should be excess daughters instead of sons. However, I have no knowledge of the hormonal effects of the other contaminants (arsenic and heavy metals) on men.
2. Because some of the endocrine-disrupting effects of the contaminants may be expected to be reversible, it is particularly important that the hormone levels of any remaining residents of the site be assayed.
3. It would be interesting to know whether there are any systematic differences in the hormone levels and residual contaminant levels between the parents of sons and the parents of daughters in the settlement. Any such differences could then be proposed as the causes of the sex ratio differences.

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