doses, and indeed, a linear term that is barely perceptible in the observable dose range could still dominate at low doses (Kaldor and Day, 1985). For this reason, the EPA and other bodies have based risk assessment on models that assume linearity at low doses, while fully admitting their pragmatic rather than scientific basis. Scientists are all too aware of the complexities involved to take any model of carcinogenesis literally, but generally accept the need for some sort of standardized quantification of the results from animal experiments (Peto et al., 1984). The principle of conservatism also lies behind the choice of the most sensitive animal species or cancer site to indicate human risk.

Cross-species extrapolation may be facilitated in the near future by new developments in biological dosimetry. By using monoclonal antibodies, biochemists can now detect the reaction products of DNA-damaging agents and cellular DNA, and thus potentially have a much closer measurement of the dose received by a target organ (Berlin et al., 1984; Bartsch, Hemminki and O'Neill, 1988). Studies comparing these measurements on different species should shed light on the dose scale that is appropriate across species.

CONCLUSION

Freedman and Zeisel make many points that are true, if not novel, but they seem to assume that other scientists have never considered the problems they address. Risk assessment is a complex process that can probably never be automated, but it plays an essential role in an advanced industrial society.

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Comment

William DuMouchel

1. INTRODUCTION: WHY I EXPECTED TO DISLIKE THIS PAPER

As set out and exemplified more fully in DuMouchel and Harris (1983), I believe that analysis of animal studies can be used to form and improve numerical estimates of cancer risk to humans. Professors Freedman and Zeisel, in their abstract, claim that this is "well beyond the scope of the scientifically possible." This paper, similar in spirit to Freedman and Navidi (1986), seems to deny that statistical modeling can really help much when up against the horrible complexity of real-world problems.

Their confrontational style is designed to provoke reactions of outrage among statistician true believers. Among many examples, overstatements like those in the Introduction "[at first] we felt—along with every other educated person—that DDT caused cancer," and "routine bioassays have little to do with basic research," will probably be pounced upon by other discussants. Not all species extrapolation methods rely solely or naively on Abbott's formula, as the authors seem to imply in Section 2. My personal favorite occurs at the end of the paper "we ... find informal argument more appealing [than statistical modeling] because it brings uncertainties into the

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