gamma radiation these quanta are sufficient to remove electrons from atoms, leaving positively charged ions (hence the term "ionizing" radiation). The end product of such an event in living tissue can be strand breakage or base damage in the cell's DNA. Such an event *could* result in a mutation leading to a cancer. It is important to note that, because of the quantum nature of radiation, there is no dose of ionizing radiation (other than zero) below which these events cannot occur, and the dose-response curve for their production must be linear. However, although such a mechanism for radiation carcinogenesis is plausible—even likely if we consider the relationship between mutagenesis and carcinogenesis (*1*)—it is not based on solid experimental data. However, there is a wealth of data which indicates that most basic cellular effects of radiation (for example, chromosome aberrations, deletion mutations, and cell killing) are produced by the interaction of two "sublesions" which occur close together in a cell in both space and time (*2*). Although this means that the dose-response curve for cellular effect will be quadric (that is, related to the square of the dose), it can be shown rigorously that the dose response curve at low doses must be linear without a threshold, purely on the basis of the physical deposition of energy at the microscopic level (*3*). Another way of arriving at the same conclusion, again based purely on the physics of radiation interaction, derives from the fact that all low LET (linear energy transfer) radiations (such as x- and gamma rays) have a high LET, or densely ionizing, component. This component affects cells exactly as does pure high LET radiation (*4*). Because the dose-response curve for cancer induction by high LET radiation is invariably linear, without a threshold (*5*), it follows that the low dose portion of the low LET radiation dose-response curve must also be linear, with no threshold. The extent of the linear portion of the curve remains to be established firmly, but the evidence, both from a variety of endpoints in mammalian cells (such as chromosome aberrations and inheritable mutations) and from studies of cancer induction in humans by radiation, indicates that the linear portion is dominant to approximately 100 rads (*6*). Since much of our knowledge of carcinogenesis by radiation comes from doses of around 100 rads, this means that, for all practical purposes, linear extrapolation from data obtained at these doses will be a good way of estimating effects at very low doses.

Wolfe's point that radiation effects in the general public from nuclear power (or from any other source of radiation contributing to public exposure) cannot be detected statistically is probably correct. One reason for this is that radiation-induced cancers or genetic changes are no different from cancers that are not radiation-induced or from genetic effects and hence are indistinguishable from them. A second reason is that the huge number of such effects constitutes a large statistical background. For example, more than 300,000 people will die of cancer this year in the United States.

If we had no way of distinguishing death by murder from death by natural causes, the death rate from murder could increase manyfold before it become noticeable as an increase in the mortality from all causes. Such is the problem with cancers induced by radiation or by any other carcinogen in our environment. It is important not to equate "undetectable" with "insignificant."

MARTIN BROWN
Division of Radiobiology Research, Department of Radiology, Stanford University, Stanford, California 94305

References

1. B. N. Ames, *Science* **191**, 241 (1976).
2. A. M. Kellerer and H. H. Rossi, *Curr. Top. Radiat. Res. Q.* **8**, 85 (1972).
3. A. M. Kellerer, in *Biology of Radiation Carcinogenesis*, J. M. Yuhas, R. W. Tennant, J. D. Regan, Eds. (Raven, New York, 1976), pp. 1–12.
4. W. T. Tobleman and A. Cole, *Radiat. Res.* **60**, 355 (1974).
5. C. W. Mays, H. Spiess, G. N. Taylor, R. D. Lloyd, W. S. S. Jee, S. S. McFarland, D. H. Taysum, T. W. Brammer, D. Brammer, T. A. Pollard, in *Health Effects of Plutonium and Radium*, W. S. S. Jee, Ed. (J. W. Press, Salt Lake City, Utah, 1976), pp. 343–362.
6. J. M. Brown, *Health Phys.* **31**, 231 (1976).

Page Charges: Shifting the Burden

Windsor's professedly preposterous notion (Letters, 24 Dec. 1976, p. 1377) that authors of papers should pay higher dues because they generate most of a scientific society's expenses leads indirectly into the most important aspect of the discussion of page charges.

That learned societies charge for publication of the scholarly work of their members shows just how thoroughly they have lost sight of what they were organized for in the first place—to disseminate knowledge. The Postal Service law is to the point. Learned societies should discontinue page charges and distribute the burden among all the members, subscribers, and advertisers, who are the primary beneficiaries.

MARSHALL C. JOHNSTON
Department of Botany, University of Texas, Austin 78712