cer deaths and genetic defects in such a large population would not be due to the associated radiation doses, although a small percentage of a very large number of these afflictions probably would be.

All these points were made in the APS study. They are important in helping to put the possible consequences of a reactor accident into perspective. But it is important that this process of gaining perspective not be carried to the point where it is concluded that "the solution to pollution is dilution." We must be concerned about reactor safety even if most of the victims of an accident would not know the original cause of their affliction.

3) Wolfe quotes the National Council on Radiological Protection and Measurement as stating that the linear hypothesis (by which observed effects of high doses of radiation are extrapolated to low doses by assuming that the probability of cancer induction is linearly proportional to the dose) has "such a high probability of overestimating the actual risk as to be of only marginal value, if any, for purposes of *realistic* risk-benefit evaluations." In fact, the situation is much more complicated and uncertain than this quote would seem to imply. In some cases, as in the induction of human thyroid tumors where effects have been observed from very low doses, the linear hypothesis works quite well (*4*). In some animal experiments, on the other hand, it appears to overestimate the hazard (*5*). In still other cases, it may underestimate the hazard (*6*). Overall, for estimating human radiation carcinogenesis by beta and gamma rays (the types of radiation of greatest concern in radiation accidents), it would appear that the linear approximation is not unreasonable (*7*).

It is interesting to note in this connection the experience of the Rasmussen group, which, contrary to Wolfe's implication, abandoned the linear hypothesis in their final report and used "central estimate" dose effect relationships for estimating the incidence of each type of cancer fatality downwind from a reactor accident. The numbers of cancer fatalities which they calculated with these assumptions were only about a factor of 2 lower than those which they would have gotten using the linear hypothesis—well within any reasonable uncertainty that would be assigned to such calculations.

What is the "bottom line" on all this? I agree with Wolfe that we shouldn't become so obsessed with certain risks, such as reactor accidents, that we become blinded to other, potentially more serious, risks. On the other hand, in the case of reactor safety at least, I would prefer that the industry offer better-designed safety systems (as the APS study suggested in the case of emergency core cooling systems and reactor containment buildings) rather than the choice many participants in the current debate seem to prefer: "Today's reactors—take them or leave them."

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Cell Line Identification

The report by Ferrone *et al.* (2 July, p. 53) indicating the presence of the fourth component of complement (C4) on human lymphoid cells was of interest to us. At the Roswell Park Memorial Institute in Buffalo, New York, approximately 1000 human cell lines with the prefix RPMI have been established; Ferrone *et al.* specify two RPMI lymphoid cell lines, RPMI 1788 and RPMI 1301, in their report. The RPMI 1301 cell line is not in the established records and does not fit into the coding system.

These investigators, as well as others, have not thoroughly characterized or referenced the cell lines they are using and thereby have added confusing information to the literature. Nelson-Rees (9 Jan., p. 96) has summarized some of the problems associated with cell line identification; the solutions are difficult and errors have occurred in many laboratories, including our own.

Hundreds of investigators have been given RPMI cell lines without charge. We have recommended that such cell lines not be passed on to other investigators without proper historical information, including type of tissue and date of origin, special characteristics, and maintenance of a stock culture in a cell bank. This kind of information would minimize confusion of such lines. Scientists who are using cell lines from this laboratory may wish to send a cell sample back to us in order to ensure that the cells are properly labeled and without significant aberrations.

Last, we think that the use of a cell line by an investigator does not warrant including the cell line originator as a coauthor (despite the ephemeral glory of being widely cited), but accurate identification is necessary.

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The cell line 1301 was obtained from Berge Hampar at the National Institutes of Health 3 years ago. Due to an error in our laboratory, the cell line became labeled as RPMI 1301 instead of 1301. It is not true, however, that we do not characterize our cell lines. We routinely characterize the cell lines and reanalyze them at 6-month intervals for their histocompatibility antigenic profile and for expression of receptors for the third complement component (C3), receptors for monkey red blood cells (MRBC), and receptors for sheep red blood cells treated with 2-aminoethylisothiouronium bromide (AET-SRBC). The cell line 1301 does not express any major HLA specificity as determined by a quantitative microabsorption technique or receptors for C3, MRBC, or AET-SRBC as detected by rosette formation. The cell line RPMI 1788 expressed the HLA antigens A2, A10, B7, and B14, C3 receptors, and MRBC receptors, but not AET-SRBC receptors. We have previously published our characterization of these cell lines (*1*).

Thus while we have erred in our labeling of cell line 1301, we have thoroughly characterized this line and others in use in our laboratory. We completely agree with Moore and Woods that the literature is full of confusing information and thank them for pointing out our error.

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