LETTERS

Cell Contamination: Relevance to Radiation Experiments

I would like to respond to William J. Broad's article "The case of the unmentioned malignancy" (News and Comment, 12 Dec. 1980, p. 1229). I worked for and with Paul Todd from 1969 to 1978 and was responsible for much of the tissue culture operation in the laboratory during that time. I feel a disparaging shadow has been unjustly cast over Todd's professional reputation by Broad's article.

The following points should be noted.

1) Furcinitti and Todd (*1*) measured cell *death* at low gamma ray doses, *not* genetic mutations or transformation from the "normal" to the "cancerous" state. Broad's reference to the *New Scientist* headline (*2*) that "Low radiation doses do cause cancer" is that magazine's premature conclusion. Furcinitti and Todd never made that statement in their report; they merely suggested that their findings "should stimulate a more intense experimental investigation of the applicability of the linear hypothesis to human mutagenesis and carcinogenesis" (*1*). They regretted the *New Scientist*'s misleading statements in *Science* (*3*), a fact not mentioned in Broad's article.

2) Our laboratory always operated under the premise that "normal" (for cultured mammalian cells) meant that they were diploid and had a finite capacity for cell multiplication (*4*). T-1 cells have been passaged hundreds of times and were known to be very heteroploid (*5–7*). Even if T-1 had been derived from normal tissue, only the most naïve tissue culturist would feel they were representative of "normal" tissue.

3) With regard to the credibility of the data obtained with T-1, it is important radiobiologically to use one cell line so that nuclear cross-sectional areas, repair capacities, and so forth are fairly consistent from experiment to experiment and from laboratory to laboratory. T-1 cells fill that need. Nelson-Rees *et al.* have shown that the various T-1 lines throughout the world are karyotypically similar with a "likelihood of a clonal derivation of all T-1 cultures" (*8*). The use of an established line of cells technically and financially facilitates radiobiological research in its early stages. The use of "normal" diploid fibroblasts like WI-38 would require significantly larger expenditures for dishes, serum, media, and labor and thereby restrict the amount of data obtained.

What do data obtained with T-1 cells have to do with the real world? Probably no less than if the same data were obtained with any other cell line. We showed how similar cell lines can vary radiobiologically under similar conditions (*6*), and Nelson-Rees and Flandermeyer state that "It is, no doubt true that the different bona fide strains of HeLa perform in different ways and exhibit many distinct characteristics; the same is true of cultures of HeLa that are known by different designations, but are de facto HeLa strains themselves" (*9*).

Had "normal" WI-38 been the cell line of choice among radiobiologists, the low plating efficiency, technical demands, and the progressive senescence of the cell line would add almost as many complications to data interpretation and extrapolation as we have now with T-1 cells. It is a very long jump from log-phase, partially or fully dedifferentiated cells in a dish to the mostly stationary-phase and differentiated cells in a human body. It is even risky to assume that what is true for cell line A is also true for cell line B, as the quote above (*9*) states.

4) Our first knowledge of the possible HeLa origin of T-1 cells came when I submitted a sample to the American Type Culture Collection (ATCC) with the hope that it might be an established human cell line of epithelial-like morphology that was *not* suspected of being HeLa contaminated. Several, if not all, of the ATCC holdings in that category at that time had HeLa markers (*10*). I was informed that the ATCC found the isozyme G6PD(A), one copy of HeLa marker chromosome No. 2, and two copies each of No. 3 and No. 4 (*7, 11*). With the ubiquity of HeLa markers in the ATCC lines, we could not exclude the possibilities of an assay artifact or the inadvertent finding that many established human lines develop these markers. As we never considered T-1 "normal" in any way and none of our data was immediately affected, we did not pursue it further. I openly described the foregoing to the point of stating that "it appears likely that our T-1 cells are HeLa cells" (*7*). Todd tried in vain to publish the findings, uncertain as they were, and this is mentioned in Broad's article. There was never any cover-up.

5) Nelson-Rees *et al.* state that "If a human cell, whether normal or tumor, sufficed in the protocol, the conclusions drawn remain sound" (*8*). T-1 cells are as good as any other established cell line for the colony-formation experiments in question. The conclusions drawn by Furcinitti and Todd (*1, 3*) are considerably more conservative than the headline in the *New Scientist* (*2*) and Broad's article might lead one to believe.

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References

1. P. S. Furcinitti and P. Todd, *Science* **206**, 475 (1979).
2. *New Sci.* **84**, 439 (1979).
3. P. S. Furcinitti and P. Todd, *Science* **210**, 806 (1980).
4. L. Hayflick and P. S. Moorhead, *Exp. Cell Res.* **25**, 585 (1961).
5. P. W. Todd, *Univ. Calif. Radiat. Lab. Rep. UCRL-11614* (1964).
6. C. B. Schroy and P. Todd, *Mutat. Res.* **33**, 347 (1975).
7. C. B. Schroy, thesis, Pennsylvania State University (1978).
8. W. A. Nelson-Rees, R. R. Flandermeyer, D. W. Daniels, *Science* **209**, 720 (1980).
9. W. A. Nelson-Rees and R. R. Flandermeyer, *ibid.* **191**, 96 (1976).
10. S. Grimwade, *Nature (London)* **259**, 172 (1976); K. S. Lavappa *et al.*, *ibid.*, p. 211.
11. R. J. Hay, personal communication.

Creationism in Toronto

In a recent summary of the Toronto AAAS meeting (News and Comment, 23 Jan., p. 368), Nicholas Wade faulted the session "Views of the Universe: Science versus Tradition" for not including "creationists" as speakers. In doing so, he missed an important point that was made in several contexts during the session.

We considered including a defender of a specific "creationist" point of view, and immediately ran up against the question of *which* such point of view to include. Some people believe firmly the universe was created in the recent past, others in the remote past, and yet others that there was never a specific creation ("steady-state" or "cyclic" cosmologies); there is similarly a wide (and mutually exclusive) variety of accounts of the beginnings of mankind (*1*). It was impractical to include representatives of each, and yet no defender of one would defend any other. Thus, one of the speakers (Milton K. Munitz) addressed the nature of a number of creation traditions.

Another speaker (William V. Mayer) discussed at length one particular "creationist movement" active in the United States and Canada. This is the one referred to by Wade as though it were the only such movement differing with the corpus of science. But the discoveries of science have conflicted with tradition along a number of lines, both today and in the past, in different cultures. The