Letters

Radiosensitivity of Human Cells in vitro

William J. Broad's article "The case of the unmentioned malignancy" (News and Comment, 12 Dec. 1980, p. 1229), misleads *Science* readers with regard to three major issues.

1) Concerning the relevance of our findings (*1*) on cell survival to other biological effects of ionizing radiation, we did not see the *New Scientist* article "Low radiation doses do cause cancer" (*2*) before publication and were thus unable to correct its misleading title and statements in it. Since the fundamental mechanisms involved in cell killing, carcinogenesis, and mutagenesis, both in vitro and in vivo, are unknown, it is a non sequitur to claim that the lack of a threshold dose for killing in vitro implies the lack of a threshold dose for any other effect in vivo or in vitro. We were careful to avoid any application of our findings to other biological effects in our original report and specifically warned against such applications in our reply to comments on our report (*3*). Radiation protection standards are based on cancer induction in human populations and genetic effects in experimental organisms, not on cell survival (*4*).

2) We do not dispute that the cells used in our research, human T-1 cells, do indeed have an abnormal number of chromosomes, HeLa markers, and enzymes, as well as their own unique markers. However, Stevenson, quoted several times by Broad, has commented concerning the claim that human T-1 cells are HeLa cells: "Attribution of HeLa origins to transformed cell lines with marker chromosomes and the type A isozyme of glucose 6-phosphate dehydrogenase is not unlike Berenson's work on the authorship of old Italian paintings: educated guesses that may not stand the test of time" (*5*). Regardless of whether or not T-1 cells are HeLa cells, no one has proved that T-1 cells are malignant in the sense of the scientific definition of malignancy. It is rather naïve to assume that if one wishes to study cancer one should study HeLa cells; HeLa cells have been widely used in mammalian-cell molecular biology, and most of the general principles of cell cycle progression, DNA synthesis, and RNA synthesis were established in HeLa cells without consideration of their possible malignant origin.

3) The crux of the problem is whether the shape of the dose-response curve for cell survival in vitro after gamma irradiation is dependent on the cell type used. Since Broad's article does not present the scientific evidence pertinent to this question, we summarize a small portion of it here. The first in vitro survival curve was measured by Puck and Marcus (*6*) using HeLa S3 cells. The second related report by Puck *et al.* (*7*) described a series of experiments to ascertain if HeLa cells were abnormally sensitive to x-rays in comparison to other long-term cell lines and normal human diploid fibroblast cells. Puck, commenting on this work, states that "virtually all mammalian cells tested yielded similar survival curves, almost all of which had an initial shoulder and exhibited mean lethal dose values clustering around 100 rads. Cells taken from supposedly highly sensitive tissues, such as bone marrow, displayed virtually the same radiosensitivity as those taken from any other part of the body when tested by this *in vitro* method" (*8*). Since Puck's pioneering experiments, refined variations on this theme have been reported, but more recent research by other investigators using human diploid fibroblasts from normal tissues and human tumor cell strains shows unequivocally that normal human cells are as sensitive if not more sensitive than tumor cell lines. In the case of normal human diploid fibroblasts, survival curves do have different initial slopes, but the curves are linear on a semilogarithmic plot with no trace of a threshold dose (*9*).

It appears that *Science*'s reporters could become more familiar with widely known facts when writing about a specific issue in a scientific specialty.

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Evolutionary Confusion?

Although I am not in sympathy with the substance of Roger Lewin's article (Research News, 21 Nov. 1980, p. 883) about the macroevolution conference, he is hardly to blame for the confusion that surrounds the subject, or for writing about those few aspects that, thanks to their repetition at the conference, were comprehensible. Futuyma *et al.* (Letters, 20 Feb., p. 770) label his effort as "advocacy" journalism, which they deem inappropriate for *Science* in the belief that the "scientific community and the public at large" are apt to mistake it for "news" and thereby be misled into "misunderstanding of a particular set of issues and, more generally, of the way science actually works." The readers of *Science* surely are not so naïve, but if they are they deserve their fate. That Lewin exposed only a small part of the currently confused state of evolutionary theory can hardly be held against him on the pretext of advocacy, which, if anything, characterizes the field of evolutionary theory as a whole, from its beginnings to the present day. I regret only that he did not expose more, in which case the pretexts of reaction would doubtless have been more numerous, perhaps more histrionic, but at least more interesting.

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Erratum: In the report "Regulation of cutaneous previtamin D_3 photosynthesis in man: Skin pigment is not an essential regulator" by M. F. Holick *et al.* (6 Feb., p. 590), a word was transposed in the sentence beginning on p. 592, column 1, line 5. The sentence should have read: "Loomis suggested that skin color regulated the transmission of solar ultraviolet radiation so that vitamin-D_3 photosynthesis would be relatively constant, . . ."

Erratum: In the report by E. T. Walters, T. J. Carew, and E. R. Kandel (30 Jan., p. 504), labels (B) and (C) of Fig. 1 were inadvertently reversed. Figure 1B should be "Siphon withdrawal," and Fig. 1C should be "Inking."