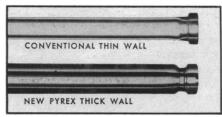


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Letters

Initial Modesty

Authors "who believe that the use of I or we is immodest" were gently chided for their false modesty in the lead editorial, "Passive voice," in *Science* for 22 March 1957 [125, 529 (1957)]. But authors who prefer not to write in the first person are much more honest than those, like the author of the editorial in question, who write in the first person without signing their names.

An editorial, report, or article whose author is identified only by initials has an anomalous status. Does it represent the view or opinion of the editorial board, as competently expressed by one member? Or does the use of initials imply a disclaimer by the board as a whole, so that only the owner of the initials is to be held responsible?

If cryptic initials are used only for internal identification, they should be much less obtrusive—perhaps in 6-point type, or in the form of a code number or of initials run together without periods, even in reverse order. As used, full size, in *Science*, initials usually can be matched up with one of the names in the masthead, so they offer little anonymity. The editorial in question was signed "R.V.O." —presumably Robert V. Ormes, a member of the editorial staff. Was Ormes so ashamed of his editorial that he did not wish his full name attached to it?

(Perhaps he should be ashamed. For an essay on grammatical purity, its own purity leaves something to be desired. I was particularly set on edge by the sentence: "In the editorial office we still see gerunds and participles used in this manner, and it is discouraging." Is the manner discouraging?)

In addition to making an unclaimed orphan out of an editorial or note, the use of initials, rather than an honest name, imposes an unwarranted burden on the poor bibliographer. Forever more, this editorial must be listed as: "R(obert?) V. O(rmes?): Passive Voice," or "R.V.O. (Robert V. Ormes?): Passive Voice." Bibliographers should not be forced to pay thus for an unsure author's false modesty.

Berkeley, California

Arnold Court

Radiation Hazards

In the article entitled, "Mice, men, and fallout" [Science 128, 637 (1958)], M. P. Finkel presents some interesting results regarding the effects of low doses of Sr^{90} on mammalian life expectancy and incidence of certain tumors. However, in discussing these results, the author draws far-reaching conclusions relating to the danger to man (or rather lack of it) from present Sr⁹⁰ fallout. The concluding sentence states, "the present contamination with strontium-90 from fallout is . . . extremely unlikely to induce even one bone tumor or one case of leukemia." I would like to raise the following points with regard to this conclusion.

1) In the data presented, the uncertainty was such that a 7-percent shortening of life span in an experimental group did not represent a statistically significant deviation from the control, nor did a threefold increase in the incidence of osteogenic sarcomas. Yet the above statement refers to effects on the world's population which would amount to a small fraction of 1 percent.

2) No statistically justifiable extrapolation for determination of "threshold doses," or even demonstration that there is a threshold different from zero, seems possible from the data as presented. In fact, these data appear to indicate that the experimental design used is inadequate for this purpose.

3) At the present time, sober and accurate evaluations of the effects of chronic low-level irradiation of human populations, from internal and external radioisotopes, are essential for the formulation of safe and wise national and international policies regarding the testing of nuclear weapons and the development of nuclear power. It is unfortunate that at this time a statement such as that quoted above is published with the implication that it is based on experimental evidence, when actually it appears to be without objective, logical support. Unfounded statements minimizing radiation hazards can be especially harmful if they turn out later to have been false.

A more appropriate conclusion from the data would seem to be that drawn by Austin M. Brues from a discussion of other data relating to carcinogenesis [Science 128, 693 (1958)]—namely, that a linear dose-effect relation is less probable than a nonlinear relation, and that a threshold *might* occur.

Carl Moos

College of Medicine, University of Illinois, Chicago

I should like to comment on the article by Miriam P. Finkel. First of all, it is difficult to tell whether this article should be considered as an editorial or as a strictly scientific paper. If the latter is the case, I should like to strenuously object to the opening paragraph, which in a back-handed kind of way casts disrepute on some of the most eminent scientists of our time who have been concerned with the effects of fallout on human beings.

Aside from this, I particularly wish to criticize some of the scientific conclusions. The type of effect that one is looking for with respect to the action of fallout on man is such that it has been predicted that several tens of thousands



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of individuals may develop tumors or leukemia. If the entire population of the world is considered, then only one individual in a few hundred thousand might be expected to show this damage, if the magnitude of the effect is what has been predicted. I do not see, therefore, how the figures given in Table 1 of Finkel's article are adequate to enable one to draw the conclusion that there will be no effect of the above-mentioned magnitude. At the lowest level of radiation used (group number 12 of Table 1), it seems to me, the number of animals used should have been approximately 200,000 instead of 150 in order to establish an effect of the magnitude we are seeking. Even with 200,000 animals it might be expected that only one mouse would develop a tumor as the aftermath of the radiation, and therefore the number utilized should be many times greater than 200,000 to establish a statistical significance of the effect at the low levels. In view of this I think the final sentence in the conclusion is extremely unwarranted and is not in accord with an objective scientific appraisal of the data presented.

Although the author points out that there are considerable differences to be expected in the response to radiation of a mouse and of man, I think this point should have been further stressed, particularly in view of the conclusion in the final sentence. One very obvious great difference is the fact that the mouse cells are exposed to the radiation for a period of not more than approximately 2 years, whereas human cells may be exposed to the radiation for a period of 60 to 70 years, with much more far-reaching consequences possibly accruing in the latter case.

I feel that the great publicity given to the article in question in the newspapers has given perhaps an erroneous viewpoint to many laymen who are not familiar with some of the imponderables involved.

JAY S. ROTH

Department of Biological Chemistry, Hahnemann Medical College and Hospital, Philadelphia, Pennsylvania

The article by Miriam P. Finkel of Argonne National Laboratory propounds very sweeping conclusions on the lack of danger from small doses of ionizing radiations, and particularly from strontium-90 fallout. An examination of the assumptions upon which these conclusions rest is called for. The chief of these is that the main danger of radiations in man's environment lies in their effects on the individuals exposed. The author states (page 637): "At lower levels [of radiation], tumor induction and shortening of life are the major signs of damage." This leads her to restrict her discussion of the evidence as follows: "The most useful criteria of radiation damage to the mammalian organism as a whole are decrease in life span and increase in incidence of certain tumors." Although she states that "these studies are concerned with the effects upon the exposed generation only," the article shows no sign whatever that the author is aware of the fundamental distinction between somatic and germinal radiation damage.

The undoubted fact that high-energy radiations induce mutations in germ cells would seem to be sufficient reason for at least using caution in discussing a question of such importance as the radiation damage to human populations. The data presented have obviously no bearing on the problem of direct proportionality between the radiation exposure and the number of germinal mutations induced. The question of linearity of response of somatic cells to radiation is treated in a paper by A. M. Brues, from the Argonne Laboratory, in the issue of Science following that in which Finkel's paper appeared [128, 693 (1958)]. The conclusion reached is that there is no evidence of linear relationship between carcinogenesis and the dosage of carcinogen, and that this makes a mutational origin of cancer doubtful. Whether or not this conclusion is accepted, the evidence for it is set forth clearly and examined critically. The same cannot be said for Finkel's presentation, which arbitrarily excludes from consideration the genetic radiation damage.

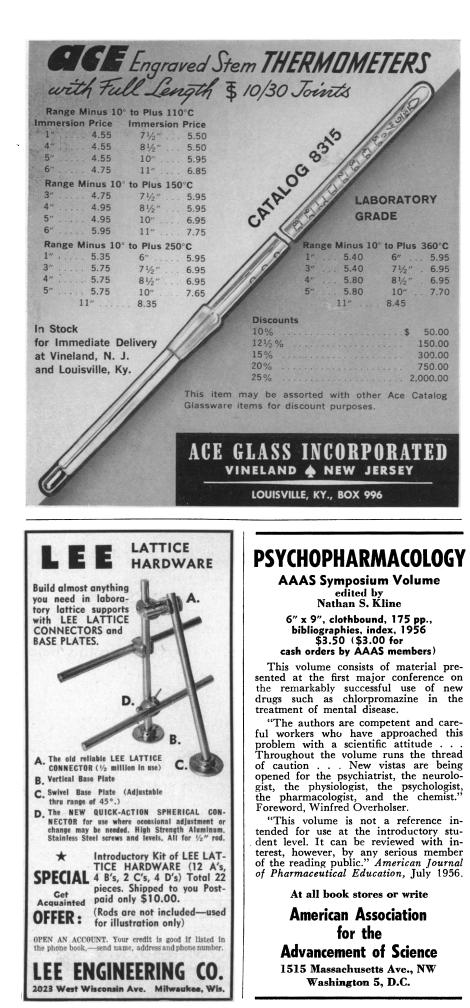
The neglect of elementary methods of critical examination of evidence leads us to doubt not only Finkel's main conclusion that "the present contamination with strontium-90 from fallout is so very much lower than any of these levels that it is extremely unlikely to induce even one bone tumor or one case of leukemia" but also the rationale on which the work was based. Surely understanding of the effects of radiation on populations of organisms, including man, is not likely to be advanced by willful neglect of one of the well-established effects of radiation.

L. C. Dunn T. Dobzhansky

Department of Zoology, Columbia University, New York, New York

Moos' comments are most pertinent to the complicated problem of the potential danger from very low doses of radiation. Since the major assumptions upon which the usual estimations of the human hazard have been based are not supported by animal experimentation, there is no reason to believe that straight lines drawn from the effects of moderate doses to zero effect at zero dose have any meaning. My conclusions have been based upon alternative methods of assessing the human hazard.

The objection is raised by Moos in point 1 that, in spite of the statistical uncertainties of the values at low levels, statements referring to large populations (Continued on page 1580)



(Continued from page 1534)

are made. On the contrary, my conclusions are not based on the results at these low levels. They stem from the extrapolation of tumor data from mice through cats and dogs to man, and from comparisons of radium and strontium-90 toxicity in mouse and man.

In point 2 Moos objects that the experimental design is inadequate to demonstrate a threshold dose. That is certainly true. However, the design is adequate for the intended purposes of the experiment—namely, to examine the effects of a range of doses and to investigate the shape of the dose-response curve.

One consistent difficulty in assessing the fallout situation is exemplified in point 3 of Moos's letter. The first sentence is one with which any intelligent person could agree whole-heartedly. The second sentence is a consequence of the charged, emotional approach so often apparent in discussions of the hazards of fallout. This attitude has unconsciously influenced many interpretations of radiobiological data. We need honest, objective, unemotional evaluations of the experimental results, which can then be applied to problems of world-wide contamination. It is very important that concern over these problems not be permitted to distort the appraisal of the experimental results.

Moos has suggested that I temper my conclusions. None of the animal data have produced linear dose-response curves. The obvious conclusion is not "that a linear dose-effect relation is less probable than a nonlinear relation" but that the relationship probably is not linear. Regarding the evidence for a threshold, I agree that the only justified conclusion at this time is that a threshold might exist. I so stated in my article.

Roth's reaction to the opening paragraph of "Mice, men, and fallout" beautifully illustrates one of the primary reasons for that article's having been written. Too many of us expect the distinguished authority in some specialized field to be an unquestioned authority in all fields.

It has been objected that not enough animals were used to predict events that might happen one time in a few hundred thousand. This is certainly true. If Roth will reread the third paragraph of the article in question, he will find that my objective was not to test such frequencies but to examine the two major assumptions upon which the previous predictions of damage from fallout have been based. The 960 mice provided doseresponse curves with characteristics contrary to these two assumptions. That is, they are not linear, and they suggest that a minimum dose must be exceeded before the response is manifest. Consequently, extrapolations along straight lines from effects at moderate or high doses to no effect at no dose are unwarranted. Our best information, based on experimentation rather than speculation, is that ". . . the present contamination with strontium-90 from fallout . . . is extremely unlikely to induce even one bone tumor or one case of leukemia."

Roth calls attention to the very short life span of the mouse contrasted to that of man. This difference, along with the great dissimilarity in size, is the main obstacle to transferring mouse data directly to man. He will note, in the tentative extrapolations given in Fig. 5 of the article, that both of these factors have been taken into account.

Apparently Dunn and Dobzhansky feel that my article should have encompassed all of radiobiology. On the contrary, it was deliberately limited to one small aspect of this subject-namely, considerations of the methods that have been used and that can be used to predict the consequences to exposed individuals of low levels of radiation. A discussion of inheritable damage was not pertinent, and I expressly stated that the exposed generation only would be considered. If one announces that he is going to investigate the effects of temperature upon mitosis, for example, should he be accused of "willful neglect" if he does not include the effects of temperature upon gene mutation? I have had no experimental experience with radiation genetics, and it would be presumptive for me to pose as an authority on that subject. I am confident that the geneticists themselves will eventually be able to tell us whether the linear relationship between gene mutation and exposure holds at doses lower than 25 roentgens.

Dunn and Dobzhansky say that I have assumed "that the main danger of radiations in man's environment lies in their effects on the individuals exposed." There is no basis in my article for this statement. The sentences they quoted were not intended to justify the omission of a discussion of genetic consequences, as they suggest. These quotations merely describe the kind of changes that are most apparent and most easily measured in exposed animals.

I agree wholeheartedly that the data I presented have no bearing on the problem of radiation exposure and germinal mutations. It also was not my purpose to discuss somatic mutations or possible mechanisms of carcinogenesis. Why should a reader be surprised that these subjects were not covered? I also did not include any mention of the effect of radiation upon the sexual behavior of *Paramecium*.

It is difficult to understand how two distinguished scientists could so misread my paper that they should accuse me not only of ignorance of the distinction between somatic and germinal radiation damage but also of "neglect of elementary methods of critical examination of evidence." My main thesis was that most



predictions of the effect of fallout on tumors and life shortening have been based on very scanty evidence and unsupported assumptions. I proposed alternative methods of prediction that use information from animal experiments as well as available human data. I am forced regretfully to conclude that the fallout problem elicits such an emotional response that many otherwise sagacious and objective scientists lose their ability to read accurately and think clearly.

MIRIAM P. FINKEL Argonne National Laboratory, Lemont, Illinois

Forthcoming Events

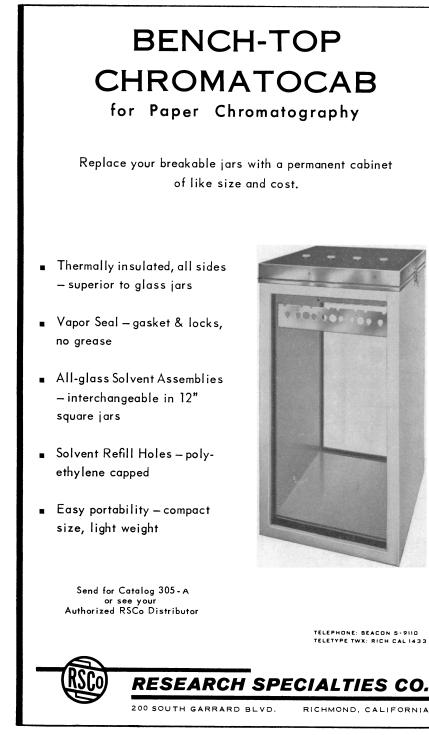
January

12-14. Reliability and Quality Control, 5th natl. symp., Philadelphia, Pa. (W. T. Sumerlin, Philco Corp., 4700 Wissahickon Ave., Philadelphia 44.)

18-31. Bahamas Serendipity Session, Nassau, Bahamas. (B. L. Frank, 1290 Pine Ave., W. Montreal, Canada.)

20-22. American Mathematical Soc., annual winter, Philadelphia, Pa. (E. G. Begle, Leet Oliver Hall, Yale Univ., New Haven, Conn.)

21-22. American Group Psychotherapy Assoc., 3rd annual institute, New York,



N.Y. (C. Beukenkamp, Public Relations Chairman, 993 Park Ave., New York 28, N.Y.)

22-23. Mathematical Assoc. of America, 42nd annual, Philadelphia, Pa. (H. M. Gehman, MAA, Univ. of Buffalo, Buffalo 14, N.Y.)

23-24. American Group Psychotherapy Assoc., 16th annual conf., New York, N.Y. (C. Beukenkamp, Public Relations Chairman, 993 Park Ave., New York 28.)

23-24. Reproductive Physiology and Protein Nutrition, 15th annual conf. on protein metabolism, New Brunswick, N.J. (J. H. Leathem, Rutgers Univ., New Brunswick, N.J.)

24-29. American Acad. of Orthopedic Surgeons, Chicago, Ill. (C. L. Compere, 720 N. Michigan Ave., Chicago, Ill.)

26-29. American Meteorological Soc., New York, N.Y. (K. C. Spengler, AMS, 3 Joy St., Boston 8, Mass.)

26-29. American Soc. of Heating and Air Conditioning Engineers, 65th annual, Philadelphia, Pa. (W. M. Vidulich, ASHACE, 62 Worth St., New York 13.)

26-29. Institute of the Aeronautical Sciences, 27th annual, New York, N.Y. (IAS, 2 E. 64 St., New York 21.)

26-30. Writing and Publication in Industry, conf. and workshops, Brooklyn 1, N.Y. (T. L. Donahue, Writing and Publication Conf., Polytechnic Inst. of Brooklyn, 333 Jay St., Brooklyn 1.)

27-30. Society of Plastics Engineers, Inc., 15th annual tech. conf., New York, N.Y. (L. A. Bernhard, SPE, 65 Prospect St., Stamford, Conn.)

28-29. Nuclear Fuel Elements, 1st intern. symp., New York, N.Y. (H. H. Hausner, 1st Intern. Symp. on Nuclear Fuel Elements, 730 Fifth Ave., New York 19.)

28-31. American Physical Soc., annual, New York, N.Y. (E. R. Fitzgerald, Dept. of Physics, Pennsylvania State Univ., University Park.)

29-31. Western Soc. for Clinical Research, 12th annual, Carmel-by-the-Sea, Calif. (W. N. Valentine, Office of the Secretary, Univ. of California Medical Center, Department of Medicine, Los Angeles 24.)

February

1-6. American Inst. of Electrical Engineers, winter general, New York N.Y. (N. S. Hibshman, 33 W. 39 St., New York 18.)

3-5. Reinforced Plastics Conf., 14th, Chicago, Ill. (Soc. of Plastics Industry, Inc., 250 Park Ave., New York 17.)

6-7. American College of Radiology, Chicago, Ill. (W. C. Stronach, 20 N. Wacker Dr., Chicago 6.)

9-11. American Acad. of Allergy, Chicago, Ill. (B. Rose, Royal Victoria Hospital, Montreal, P.Q., Canada.)

9-11. Nature of Coal, symp., Bihar, India. (Director, Central Fuel Research Inst., P. O. Fuel Research Inst., Dhanbad District, Bihar.)

11-13. American Acad. of Occupational Medicine, Boston, Mass. (L. Blaney, 1608 Walnut St., Philadelphia, Pa.)

12-13. Solid State Circuits Conf., Philadelphia, Pa. (A. B. Stern, General Electric Co., Bldg. 3, Syracuse, N.Y.)