Letters

Recycled Oil

There are several minor misconceptions in Thomas H. Maugh's excellent article "Rerefined oil: An option that saves oil, minimizes pollution" (Research News, 17 Sept., p. 1108). These concern efforts by the National Bureau of Standards (NBS) to fulfill the Energy Policy and Conservation Act of 1975 (Public Law 94-163).

Maugh states that NBS is to ". . . demonstrate the equivalency of rerefined and virgin lubricating oils. . . ." Actually, the law states that NBS is to ". . . develop test procedures *for determination of* substantial equivalency of re-refined or otherwise processed used oil . . . with new oil for a particular end use [italics ours]." Thus, NBS is to provide test procedures that can be used to determine the equivalency of a particular sample, on a sample-to-sample basis. This is substantially different from demonstrating equivalency, which could be interpreted to mean that, once equivalency is demonstrated, by whatever means, the problem is solved.

Also, Maugh states that NBS is required to ". . . develop simpler ways to measure the quality of lubricating oils." The law does not include that statement, nor any portion of it. While we agree that "simpler ways" would be desirable, such developments are thought to be highly unlikely by experts in the field and would be a side benefit of the NBS program, not a requirement under the law.

Finally, it is important to comment on the comprehensiveness of the law. While Maugh discusses only used oils recycled as engine lubricating oils, the law covers ". . . re-refined or otherwise processed used oil or blend of oil . . . ," which in our judgment includes such end use products as industrial oils, metal-working oils, hydraulic oils, and oils used for fuel, as well as engine (both crankcase and transmission) oils.

Each of these categories of oils has one or more individual sets of specifications, test procedures, and problems. In addition, each category of oil consists of different types [for example, fuel oil has six grades; there are apparently at least 15 different types of hydraulic oils that are widely used; engine oils include crankcase oils (the SA grade requires no performance tests; the SE grade requires costly engine sequence tests) as well as transmission oils]. All petroleum-based oils are covered under the Energy Policy and Conservation Act.

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Nuclear Reactor Accidents: Long-Term Health Effects

The 11 June congressional testimony of Panofsky, von Hippel, and Rowe, as reported by Philip M. Boffey (News and Comment, 25 June, p. 1312), is critical of the treatment of long-term health effects from reactor accidents in the Rasmussen report (*1*). This general criticism was first raised in the American Physical Society (APS) study of reactor safety (*2*), of which Panofsky and von Hippel were major participants. A review of the APS study, however, leads one to question whether its widely quoted results, and the criticisms in the congressional testimony, are valid (*3*).

Table 1. Exposure of an individual to cesium-137 from a nuclear reactor accident [reference accident from (*2*)].

Time after accident (yr)	Highest dose point (60 km)		Population dose midpoint (420 km)	
	Dose rate (rem/yr)	Integrated dose (rems)	Dose rate (rem/yr)	Integrated dose (rems)
0	4	0	0.57	0
1	2.2	2.9	0.31	0.41
2	1.6	4.8	0.23	0.69
3	1.4	6.3	0.20	0.90
4	1.3	7.7	0.19	1.1
5	1.3	9	0.18	1.3
10	1.1	15	0.16	2.1
20	0.81	24	0.12	3.4
30	0.60	31	0.09	4.5
40	0.45	36	0.06	5.1
50	0.33	40	0.05	5.7
70	0.18	45	0.03	6.5
∞	0	52	0	7.4

The APS study considers the long-term health effects of radioactive release from a postulated reference accident with an estimated probability of occurrence of once per 160,000 years of reactor operation. The effects from cesium-137, the major cause of predicted cancer deaths, are treated in detail. In the APS study it is calculated that the long-term population dose of cesium-137 is 70 million man-rems. By use of the linear theory of radiation health effects and (*4*), this figure is equated to 9000 predicted cancer deaths (130 deaths per 10^6 man-rems).

Although the 70 million man-rem figure is large, it represents the integrated radiation dose to a population of 9 million people over a number of decades. The basic question to be answered is, What health effects result from the individual exposures comprising the integrated dose?

Exposures to individuals are not specifically presented in the APS study but can be derived from the report. Table 1 shows the radiation exposure at the highest dose point considered in the APS study, 60 kilometers from the site of the accident. It also shows the point, 420 km from the accident, where the integrated population dose (70 million man-rems) divides into two equal parts, half incurred inside the 420-km radius and half outside. Thus, half of the integrated population dose (35 million man-rems) is due to individual radiation exposures less than those in the last two columns of the table.

The figures shown in Table 1 are noteworthy for their relatively small values. Even the annual dose rates at the highest dose point are below the occupational exposure limits set by the National Council on Radiological Protection and Measurement (NCRP) (*5*, *6*). The NCRP limits are such that "it is impossible to find any evidence of injuries either directly or by statistical means for people working within and living within such limits" (*7*). The difference between this statement and the figure in the APS study of 9000 deaths results from the use in that study of the linear theory of radiation effects, which attempts to extrapolate data from high-dose, high-dose rate exposures to

low exposure environments where effects, if any, are so small that they cannot be directly observed (*8*).

In examining radiation risks the NCRP states that the linear theory 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" (*6;* see also *7, 9*). Indeed, reservations about the use of the linear theory are contained within the body of (*4*), and its use is not justified either in (*4*) or in the APS study on scientific grounds but rather on "pragmatic grounds," since there "is no means at present" of predicting effects in the low-dose region of interest.

Thus, the results in the APS study and the Rasmussen report and the relevant comments in the congressional testimony should be viewed within the context of our knowledge of low-level radiation effects. The only positive statement one can make about the effects on people of low-level radiation is that, if there are any, they are so small that they are masked by other environmental factors to which we are subjected in normal life. This, of course, is why our knowledge is limited.

The above points are applicable beyond considerations in the Rasmussen report and appear particularly relevant to predictions of deaths being made for dose rates hundreds of times less than those discussed above. The linear theory, as it is being used in some quarters, allows one to obtain newsworthy figures by taking negligibly small and meaningless numbers and multiplying them by hundreds of millions or billions of people and infinite periods of time (*10*). The linear theory makes it possible for the Environmental Protection Agency (EPA) to issue unsupportable press releases claiming that standards which would reduce population exposures by one-half millirem per year would save 1000 lives (*11*). At these low levels it is not even plain that a beneficial effect is precluded (*12, 13*). Indeed, we have extensive experience with one form of radiation (*13*) which produces cancer at high exposures, and one could be concerned about an epidemic of rickets if the EPA treated solar radiation in a manner consistent with its statements on nuclear radiation.

The needless risk of life is too high a price to pay for any activity, and the establishment of prudent and cautious radiation standards should be supported by all. But unrealistic risk estimates may increase overall loss of human life by encouraging substitution of more hazardous activities, by the allocation of resources which might more effectively be used elsewhere, and by depriving society of important benefits it might otherwise have.

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References and Notes

1. Nuclear Regulatory Commission, *Reactor Safety Study* [WASH 1400 (NUREG 75/014), Washington, D.C., 1975]; available from the National Technical Information Service, Springfield, Va.
2. American Physical Society, Study Group on Light Water Reactor Safety, *Rev. Mod. Phys.* **47** (Suppl. 1), (1975).
3. In view of the following remarks, I should note that, throughout, the authors of the APS study have taken evident pains to present their results in a balanced and responsible manner.
4. Committee on Biological Effects of Ionizing Radiations, *The Effects on Populations of Exposure to Low Levels of Ionizing Radiation* (National Academy of Sciences–National Research Council, Washington, D.C., 1972).
5. National Council on Radiological Protection and Measurement, *Basic Radiation Criteria* (NCRP Report No. 39, Washington, D.C., 1971); available from NCRP Publications, Post Office Box 30175, Washington, D.C.
6. ———, *Review of the Current State of Radiation Protection Philosophy* (NCRP Report No. 43, Washington, D.C., 1975); available from NCRP Publications, Post Office Box 30175, Washington, D.C.
7. L. S. Taylor, "Testimony Before the Environmental Protection Agency" (Washington, D.C., 8 March 1976).
8. In simplest form, the linear theory states in effect that, if 100 aspirin tablets taken at one time will kill a person, then one tablet taken by each of 100 people will kill one of them.
9. R. D. Evans, "Comments for the EPA hearings on March 8–10, 1976" (Washington, D.C., 8 March 1976).
10. D. Comey, *Bull. At. Sci.* **31**, 43 (Sept. 1975); New England Coalition on Nuclear Pollution, *Fed. Reg.* (16 January 1975); R. O. Pohl, *Nuclear Energy: Health Impact of Carbon 14* (Laboratory of Atomic and Solid State Physics, Cornell University, Ithaca, N.Y., 1975).
11. Environmental Protection Agency, "Environmental news press release" (Washington, D.C., 23 May 1975).
12. N. A. Frigerio, K. F. Eckerman, R. S. Stowe, *Carcinogenic Hazard from Low-Level, Low Rate Radiation* (ANL/ES-26, Argonne National Laboratory, Argonne, Ill., 1973), available from National Technical Information Service, Springfield, Va.; F. J. Jankowski, *Nucl. Saf.* **7**, 12 (1965).
13. R. J. Wurtman, *Sci. Am.* **223**, 68 (July 1975).

In case the reader is confused as to the nature of the statements with which Wolfe is taking issue, I would like to begin with the following two points of clarification.

1) The contribution of the American Physical Society light water reactor safety study (*1*) with respect to the population radiation doses from cesium-137 was simply to point out that most of the radiation dose from this long-lived radionuclide (30-year half-life) was inadvertently not included in the doses calculated in the Nuclear Regulatory Commission's *Draft Reactor Safety Study* (more commonly known as "the Rasmussen report"). As a result the cumulative population doses calculated there were low by a factor of approximately 25. This correction was accepted in the final Rasmussen report (*2*).

2) The point which I made in my congressional testimony that is relevant to Wolfe's letter was that, in the final Rasmussen report, long-term effects, such as cancer deaths, were not included in the comparisons made between the consequences of reactor accidents and those of other events, such as meteorite impacts and dam failures. This omission produced misleading results, since the numbers of cancer deaths and genetic defects calculated in the Rasmussen report were about 1000 times greater than the number of early fatalities shown in the comparisons (*3*).

In addition, there are three points raised in Wolfe's letter on which I would like to comment.

1) He describes the APS study as having considered the long-term consequences of "a postulated reference accident with an estimated probability of occurrence of once per 160,000 years of reactor operation." The unwary reader might assume from this statement that the APS group estimated the probability as being of that magnitude, when, in fact, that estimate was made in the Rasmussen report. Indeed, it is stated in the APS study that, "based on our experience with problems of this nature involving very low probabilities, we do not now have confidence in the presently calculated absolute values of the probabilities. . . ." This is why we were so interested in checking the claim in the draft Rasmussen report that "the possible consequences of potential reactor accidents are predicted to be no larger, and in many cases much smaller, than those of non-nuclear accidents." We found in our partial review of that report major errors in the calculations of cancer deaths, genetic defects, and the natural duration of radioactive land contamination. As a result of our review, the numbers in each of these cases were revised upward approximately tenfold in the final Rasmussen report.

2) As Wolfe observes, the risk of an individual dying from cancer as a result of a radiation dose of a few tens of rems is relatively small—less than 1 percent. Large numbers of cancer deaths (on the order of 10,000 for the Rasmussen group's reference accident, along with a similar number of genetic defects and perhaps ten times as many thyroid abnormalities) are projected when these relatively small individual risks are added up for an exposed population of millions. Even after an accident, most of the can-

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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2. Nuclear Regulatory Commission, *Reactor Safety Study* [WASH 1400 (NUREG 75/014), Washington, D.C., 1975]; available from the National Technical Information Service, Springfield, Va.
3. For a more general review of the usefulness of the final Rasmussen report for policy-making purposes, see F. von Hippel, *Bull. At. Sci.*, in press.
4. B. Modan, D. Baidatz, H. Mart, R. Steinitz, S. G. Levin, *Lancet* **1974-I**, 277 (1974).
5. J. M. Yuhas, *Radiat. Res.* **60**, 321 (1974).
6. K. Z. Morgan, *Am. Ind. Hyg. Assoc. J.* **36**, 567 (1975).
7. J. Martin Brown, *Health Phys.* **31**, 231 (1976).

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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