Letters

Unesco and Nongovernmental Organizations

I was perturbed by P. H. Abelson's editorial "Unesco's tarnished image" (26 Nov., p. 897). Having just returned from the Unesco General Conference in Nairobi, I would like to offer some comments that diverge from his views.

I readily agree that it is unfortunate that Unesco's noble objectives have been clouded by political overtones. However, this political fallout, which becomes starkly visible only once every 2 years, is not a true reflection of the valuable work that goes on day after day at Unesco *without* political overtones during the periods between General Conferences. The peasant in a less-developed country who gets instructed by television on how to increase his meager harvest or, for that matter, the scientist engaged in a cooperative project developed under Unesco auspices, could not care less about the political rhetoric.

The resolution sponsored by the People's Republic of China that Abelson quotes is nothing new, having been adopted in almost identical, if not harsher form, 2 years ago. Unesco has learned to live with this kind of resolution and will not abandon its support for nongovernmental organizations, most notably the International Council of Scientific Unions, because of it.

There will be moments in any international forum when the actions taken are contrary to our views and interests, as there will be times when things go our way. As children, we could pick up our marbles and go home when things did not go our way. Perhaps we could even get our way in international organizations some years ago by threatening to do so, but many of our old arguments have been worn out. A better approach is to continue to work toward reform and improvement within the system, for how can we otherwise influence the course of events?

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I would like to commend Philip H. Abelson for his editorial of 26 November. I agree wholeheartedly that the time has come when alternative avenues for fostering international science must be explored. Major sectors of the scientific community may consider themselves to be immune to the deterioration of Unesco, but it would be foolhardy to expect free exchange of science and scientists to flourish under auspices which have become so politicized.

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Metabolic Precursors of a Known Human Carcinogen

The National Institute for Occupational Safety and Health (NIOSH) has recently learned that both *N*-phenyl-β-naphthylamine (PBNA) (a widely used rubber antioxidant) and 2-nitronaphthalene (a by-product of α-naphthylamine production) are metabolized to the known carcinogen, β-naphthylamine (BNA). We are writing to highlight the potential problem of the metabolic conversion of materials believed to be relatively innocuous into known human carcinogens.

In a recent study, the B. F. Goodrich Company found 3 to 4 micrograms of BNA in 24-hour samples of urine from two volunteers who had ingested 50 milligrams of PBNA (contamination, 0.7 μg BNA) and from workers (unspecified number) estimated to have inhaled 30 mg PBNA (*1*). These findings indicate that PBNA is at least partially metabolized by the human body to BNA and confirm an earlier study by Shell Nederland (*2*).

An estimated 15,000 workers are at risk of exposure to PBNA during its manufacture and use. The majority of these exposures occur in the rubber industry. PBNA is used as a rubber antioxidant and also as an antioxidant for greases and oils, as a stabilizer during the manufacture of synthetic rubber, and as an intermediate in the synthesis of dyes as well as other antioxidants.

E. I. du Pont de Nemours and Company has informed NIOSH of unpublished studies regarding another compound which can be metabolized to BNA (*3*). 2-Nitronaphthalene (an unmarketed by-product produced during the commercial preparation of α-naphthylamine) is metabolized in laboratory dogs to BNA. However, there are no reports concerning the metabolic fate of 2-nitronaphthalene in man.

The fact that certain substances, as illustrated by PBNA and 2-nitronaphthalene, can be metabolized to known carcinogens lends a new perspective to the control of hazards in the workplace. NIOSH therefore recommends the following.

- More consideration should be given to the assessment of metabolic pathways of chemical agents found in the workplace.
- Materials which can be metabolized by the human body to known carcinogens should be handled in the same manner as carcinogens.
- Industrial hygiene practices should be followed to minimize exposure to PBNA in the workplace. Suggested industrial hygiene practices to minimize exposure to PBNA are available from the Technical Evaluation and Review Branch, NIOSH, upon request.
- Alternative antioxidants to PBNA should be fully evaluated with regard to possible human effects.

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Radiation-Induced Health Effects

Further comment is in order regarding the letters from Bertram Wolfe and Frank von Hippel (29 Oct. 1976, pp. 478 and 479). Wolfe questions the validity of criticisms by the study group of the American Physical Society (APS) of the early drafts of the Rasmussen report (leading to a number of revisions and corrections) and objects to the 11 June

1976 congressional testimony of Panofsky, von Hippel, and Rowe, in which they were critical of the treatment of long-term health effects of reactor accidents in the Rasmussen report. Von Hippel, in his reply to Wolfe, defends his congressional testimony and the improvements made in the Rasmussen report as a result of the APS study. I would like to lend my support to Panofsky, von Hippel, Rowe, and the APS study and to the present practice of taking into account the long-term, somatic, and genetic risks from exposure to ionizing radiation by application of the linear hypothesis.

The APS study group should be commended for the improvements it brought about in the Rasmussen report and for indicating the failure of the early drafts of the report to take into proper account the long-term risks to the population and the seriousness of land contamination from radioactive contaminants—especially cesium-137. I agree with von Hippel that the final Rasmussen report was remiss in not including cancer and genetic deaths as one of the consequences of a reactor accident when making comparisons with other events, such as meteorite impacts and dam failures. I am strongly in favor of a nuclear power industry, but I want it to be reasonably safe; I fear the Rasmussen report, in underestimating the radiation risks by a factor of 1000 in these comparisons, does not make the job easier for some of us who would like to convince the public that the risks of a nuclear power plant are real but that there is good reason to believe they are considerably less than those of a fossil fuel plant.

There are many conclusions of the Rasmussen report that raise the eyebrows of someone like myself who has been in the nuclear energy business for over 40 years. Thus I would like to repeat the quotation taken by von Hippel from the APS study that "based on our experience with problems of this nature involving very low probabilities, we do not have confidence in the presently calculated absolute values of probabilities. . . ." I often recall my argument with friends in the Atomic Energy Commission shortly before the United States launched a space rocket which, together with its 17,000 curies of plutonium-238, was incinerated in the upper atmosphere over the Indian Ocean in April 1964. My friends had tried unsuccessfully to convince me before the launch that the probability of something like this happening was of the order of 10^{-7}; if so, we were very unlucky.

I would like to believe the absolute values of the low-risk numbers in the Rasmussen report, but in view of such things as overpressure in pressurized water reactors, the common mode failure aspects of the Brown's Ferry incident, and the low quality of some reactor health physics programs, I am forced to believe the risks may be larger than indicated by the Rasmussen report; we should stop speculating on the absolute magnitude of these risks and reduce them until they are as low as is reasonably achievable.

Wolfe attempts to depreciate the use of the linear hypothesis as it is used, for example, in reports of the Environmental Protection Agency and the BEIR report (*1*) of the National Academy of Sciences. However, there are many studies of human populations which provide strong evidence of an increased cancer incidence resulting from exposures to ionizing radiations equal to or less than those we accept as the maximum permissible exposure levels for radiation workers. I would like to give the following three examples.

Mondan *et al.* (*2*) examined the records

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of 11,000 migrants into Israel to whom x-rays had been administered in order to control ringworm. They found a very high risk of thyroid carcinoma (6.1×10^{-6} carcinomas per year per rad administered); the mean dose was only 6.5 rads.

Stewart and co-workers (*3*) have studied thousands of children who received in utero radiation doses of 0.3 to 0.8 rad and found mortality from leukemia and other forms of cancer is 50 percent higher on the average among these exposed children than among the unexposed controls.

Bross (*4*), at the Congressional Seminar on Low-Level Ionizing Radiation, pointed out that there are groups in the population with a very high susceptibility to radiation damage. His studies showed that children with diseases such as asthma, hives, eczema, allergy, pneumonia, dysentery, or rheumatic fever have a 5000 percent greater risk of developing leukemia as a result of exposure to x-rays than do children not so exposed. (One of my objections to the Rasmussen report is that it treats averages and does not recognize the nonhomogeneity of the human population; subgroups—children, old people, persons with respiratory diseases—should be given special consideration in evaluating environmental risks.)

Wolfe gives the impression that many experts in the radiation protection business believe there is a large factor of safety when applying the linear theory of radiation risk. However, this is not the opinion of some knowledgeable scientists. For example, the BEIR report (*1*, p. 90) states, "Because a linear extrapolation model has been used in these calculations, the number of cancer deaths attributable to any dose other than 0.1 rem/y can be estimated by simple multiplication; however, it must be borne in mind that the foregoing estimate of mortality from radiation exposure (at 0.1 rem/y) may be too high, or too low, for a variety of reasons. . . ." An ICRP (International Commission on Radiological Protection) committee report (*5*) states, "It is recognized that factors involved in tissue response to high doses of radiation might lead to either a decrease or an increase of the response/dose ratio obtaining at low doses and dose rates." It is fortunate that the BEIR committee, the ICRP committee, and others have taken a neutral position on this issue, because so much evidence is accumulating to support the use of the linear hypothesis and to confirm its applicability to many types of radiation-induced malignancies. In fact, there is strong theoretical and experimental evidence (*6*) that the linear hypothesis, in many cases, and especially for high LET radiation, for example, from neutrons and alpha particles underestimates the risk.

I agree with von Hippel that, in an accident, "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." Wolfe, on the other hand, states, "At these low levels it is not even plain that a beneficial effect is precluded." My only response to persons who make this claim is that I wish all who really believe this would install a small cesium-137 source in the ceiling of each room of their homes so that over a period of years we could obtain some convincing proof that this is a bad assumption.

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Bertram Wolfe comments on the linear theory of radiation health effects used to predict the number of cancer deaths and genetic effects from a given dose of ionizing radiation. Wolfe's main points are (i) that there are no scientific grounds for acceptance of the linear theory of radiation effects and (ii) if there are these effects at low doses, "they are so small that they are masked by other environmental factors," and hence (one is led to conclude) of little significance. The first statement is incorrect. The second, although true, is misleading, as the same statement could be made about other causes of death (such as murder by handguns) which we do regard as of some significance.

The scientific basis for the linear theory of radiation induction of cancer rests on our knowledge of how radiation interacts with biological material at the molecular level. Like all forms of electromagnetic radiation, ionizing radiation transfers energy to the material through which it passes in "quanta" or discrete packages of a fixed size. With x- and

gamma radiation these quanta are sufficient to remove electrons from atoms, leaving positively charged ions (hence the term "ionizing" radiation). The end product of such an event in living tissue can be strand breakage or base damage in the cell's DNA. Such an event *could* result in a mutation leading to a cancer. It is important to note that, because of the quantum nature of radiation, there is no dose of ionizing radiation (other than zero) below which these events cannot occur, and the dose-response curve for their production must be linear. However, although such a mechanism for radiation carcinogenesis is plausible—even likely if we consider the relationship between mutagenesis and carcinogenesis (*1*)—it is not based on solid experimental data. However, there is a wealth of data which indicates that most basic cellular effects of radiation (for example, chromosome aberrations, deletion mutations, and cell killing) are produced by the interaction of two "sublesions" which occur close together in a cell in both space and time (*2*). Although this means that the dose-response curve for cellular effect will be quadric (that is, related to the square of the dose), it can be shown rigorously that the dose response curve at low doses must be linear without a threshold, purely on the basis of the physical deposition of energy at the microscopic level (*3*). Another way of arriving at the same conclusion, again based purely on the physics of radiation interaction, derives from the fact that all low LET (linear energy transfer) radiations (such as x- and gamma rays) have a high LET, or densely ionizing, component. This component affects cells exactly as does pure high LET radiation (*4*). Because the dose-response curve for cancer induction by high LET radiation is invariably linear, without a threshold (*5*), it follows that the low dose portion of the low LET radiation dose-response curve must also be linear, with no threshold. The extent of the linear portion of the curve remains to be established firmly, but the evidence, both from a variety of endpoints in mammalian cells (such as chromosome aberrations and inheritable mutations) and from studies of cancer induction in humans by radiation, indicates that the linear portion is dominant to approximately 100 rads (*6*). Since much of our knowledge of carcinogenesis by radiation comes from doses of around 100 rads, this means that, for all practical purposes, linear extrapolation from data obtained at these doses will be a good way of estimating effects at very low doses.

Wolfe's point that radiation effects in the general public from nuclear power (or from any other source of radiation contributing to public exposure) cannot be detected statistically is probably correct. One reason for this is that radiation-induced cancers or genetic changes are no different from cancers that are not radiation-induced or from genetic effects and hence are indistinguishable from them. A second reason is that the huge number of such effects constitutes a large statistical background. For example, more than 300,000 people will die of cancer this year in the United States.

If we had no way of distinguishing death by murder from death by natural causes, the death rate from murder could increase manyfold before it become noticeable as an increase in the mortality from all causes. Such is the problem with cancers induced by radiation or by any other carcinogen in our environment. It is important not to equate "undetectable" with "insignificant."

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Page Charges: Shifting the Burden

Windsor's professedly preposterous notion (Letters, 24 Dec. 1976, p. 1377) that authors of papers should pay higher dues because they generate most of a scientific society's expenses leads indirectly into the most important aspect of the discussion of page charges.

That learned societies charge for publication of the scholarly work of their members shows just how thoroughly they have lost sight of what they were organized for in the first place—to disseminate knowledge. The Postal Service law is to the point. Learned societies should discontinue page charges and distribute the burden among all the members, subscribers, and advertisers, who are the primary beneficiaries.

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