Letters

Just Awards?

The article about the "Molecule of the Year" award (20 Dec., p. 1706) prompts me to write with a concern I have had ever since the award was announced. The concept of recognizing a major discovery each year is commendable. But the name "Molecule of the Year," while clever, seems biased and unrepresentative of the broadly based science that AAAS and *Science* aspire to represent.

No one denies that some of the most exciting research being done today is in the disciplines of molecular biology and biochemistry. Nor do I have any argument with the selection of buckminsterfullerene for this year's award. But surely the prime science results of each year cannot be expected always to lie in the areas of molecular biology and biochemistry. What if the most exciting discovery were a new concept in elementary particle physics, or a new class of objects in astrophysics? What if cold fusion had turned out to be correct? Would these still be called "Molecule of the Year"?

I get the message that my kind of science is second-class and is not likely to produce an award-winning discovery. To me it seems analogous to writing in the masculine gender and then being told that masculine really is neuter and thus includes everyone. The editor of *Science* may believe that molecular science really includes all science, but the net result seems biased to me.

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Response: Morrison is correct—we should consider all areas of science for special recognition, and we have done so by awarding "Molecule of the Year" to the polymerase chain reaction, a method, in 1989; to diamonds, an engineered material, in 1990; and to the buckyball, a molecule, in 1991. Our award runners-up have been selected from areas ranging from cosmology to social science. We meant the name "Molecule of the Year" to be symbolic of scientific discovery in general. However, we are open-minded and invite readers to suggest a more appropriate name for this award.

—DANIEL E. KOSHLAND, JR.

Florida Dentist Case: Research Affiliation and Ethics

We wish to clear up some misconceptions and errors that were contained in the News & Comment article "The case of the Florida dentist" (24 Jan., p. 392) by Joseph Palca.

Although Palca notes that one of us (L.G.A.) has no financial connection to the case, the clear implication in the caption of the photo accompanying the article (p. 393) is that Lionel Resnick, Stanley Weiss, and one of us (L.G.A.) are all part of the "defense team of experts." In fact, neither of us is part of any defense team, and we began our investigation independently in August 1990. We first contacted the Florida AIDS office in October 1990 and corresponded with the Centers for Disease Control (CDC) shortly thereafter. At the suggestion of the CDC we wrote directly to Gerald Myers at Los Alamos National Laboratory in December 1990, offering to collaborate on a statistical analysis of the sequence data. After 4 months, Myers declined our offer by phone. It is therefore incorrect to state that one of us (L.G.A.) was "recruited" by Resnick for help on this project. Only after our analysis was complete and a manuscript was nearly completed did we learn of a financial connection between Resnick and Weiss and CIGNA Dental Health of Florida. Neither of us has had any connection to CIGNA or any legal group involved in the case, nor do we have any direct or indirect financial interest in the case. Our research is funded solely by our institutions, the National Science Foundation (L.G.A.) and the National Institutes of Health (R.W.D.).

We are also particularly concerned about the implication that our use of the Freedom of Information Act to obtain data from the CDC was possibly unethical. It is standard practice, particularly in the field of molecular phylogenetics, for any data used in a publication to be included in the paper, submitted to the recognized international sequence databases, or be made available on request. The CDC had published three reports in *Morbidity and Mortality Weekly Report* that included statements about the similarities of sequences as well as a phylogenetic tree based on those sequences, although the data on which these analyses were based were not included. These published reports were being discussed widely and were being cited as the basis for public policy. We neither requested nor obtained any data that had not already been the basis of published work.

We are confident that we have in no way behaved unethically and that our critique of the CDC's analyses stands entirely on its own merit. We have no interest in promoting any particular result; we only feel that this case deserves careful consideration by the scientific community and that the data and analyses should be subject to peer review.

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Biopesticides and Resistance

The letter by M. K. Harris (6 Sept., p. 1075) and the Research News article by Ann Gibbons (1 Nov., p. 646) about the development of resistance in insects to insecticides and to transgenic plants that express the *Bacillus thuringiensis* (*Bt*) gene give the mistaken impression that little has been accomplished in this discipline and that *Bt* research is breaking new, totally unexplored ground. We believe the vast literature on conventional pesticide resistance management, as well as ongoing research with engineered plants, was not adequately addressed in these statements.

Experiments that have been done to test deployment strategies for transgenic cotton include those by one of us (D.A. and coworkers) in which two *Bt* gene constructions were introduced into 56 different plant populations that had three unrelated mechanisms of resistance to lepidopterous insects. This is making it possible to assess the benefit of pyramiding different types of genetic resistance (*1*). Several insect management strategies are being tested (*2*), including high-dose expression of *Bt* proteins in transgenic cottons, the creation of "refugia" (refuges provided for sensitive insects within a population so they will not be exposed to *Bt*), tissue-specific or inducible expression, or both, of foreign gene products such as *Bt* (to reduce overall insect population exposure), mixed seed or mosaic plantings, insertion of combinations of two or more foreign genes into the plant genome by using *Bt* or other proteins that affect Lepidoptera, and cultivation of *Bt* transgenic cottons by using certain agronomic practices to reduce exposure time. Our research has not shown that *Bt* can be deployed without resistance problems, but assertions that *Bt* transgenic cotton (and other plants) will not work are premature.

Agricultural scientists have dealt with insect and pathogen adaptation to conventionally bred resistant cultivars for nearly a century. The public rarely notices the loss of resistance genes to pest adaptation unless a highly visible crisis such as the 1970 Southern corn leaf blight epidemic occurs. Biotechnology has focused on the problems of pest adaptation, but funded research initiatives or incentive programs for good management practices have not materialized in legislation. Commitment from public institutions has been minimal for the development of sustainable approaches to conventional pest resistance breeding, and the analogous situation with transgenic plants has generated interest, but no additional resources. Without action by public agencies, engineered plants may be developed

and used in ways that rapidly lead to insect resistance, but we do not think the fate of engineered plants can be predetermined at present.

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1. D. W. Altman, in *Gene Conservation and Exploitation: 20th Stadler Genetics Symposium*, J. P. Gustafson, P. Raven, R. Appels, Eds. (Plenum, New York, in press).
2. F. Gould, *Am. Sci.* **79**, 496 (1991).

As a participant in a National Audubon Society workshop on insect resistance to *Bacillus thuringiensis* (*Bt*), I came away with a significantly different view from that expressed by your reporter Ann Gibbons. As the representative of the Monsanto Company, I described our development of insect-resistant transgenic crop plants expressing *Bt* genes; I did not express a "worry" that the cotton bollworm will become resistant to *Bt* soon after we commercialize our *Bt* cotton plants. I described several of our recommended strategies for resistance management that we believe will ensure that this does not happen. I believe there was a general consensus in the workshop that (i) *Bt*-containing crop plants will have great benefits in agriculture; (ii) with proper management *Bt*-containing crops will have a long and useful life in agricultural insect control; and (iii) we are well along the way to developing resistance management programs for *Bt* crops that will work and can be implemented by farmers.

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Response: For the record, Fischhoff told me that he was "concerned" that farmers would manage Monsanto's *Bt* cotton plants properly to prevent resistance. In fact, that was the focus of the meeting, convened by the National Audubon Society because they are "concerned that the use of delta-endotoxin products by farmers will be short-lived if insect resistance develops quickly." I also noted that "researchers have designed strategies to prevent—or at least slow down—the onset of resistance," and described several of those strategies, although there was disagreement about how effective they would be.—ANN GIBBONS

Cancer and the Environment

The otherwise excellent article by Brian E. Henderson *et al.* "Toward the primary prevention of cancer" (22 Nov., p. 1131) disregards opportunities for primary prevention of cancer in the workplace and in the general environment. They state that "[t]he widespread public perception that environmental pollution is a major cancer hazard is incorrect" and that occupational factors "are not likely to account for more than 4% of cancers in the United States. The actual percentage may be substantially lower."

These statements, while correct, show a disregard for workplace and environmental pollution that is inconsistent with the attention given by Henderson *et al.* to other primary preventable causes of cancer. Workplace-induced cancers are fully preventable. Henderson *et al.* devote much discussion to tumors such as those of the endometrium (3% of total cancer) and ovary (2% of total cancer), detailing factors such as diet and hormone use, which are smaller targets for primary prevention than are occupational cancers. Similarly, radon—even at the lower range of the estimates of exposure—while not nearly as important as tobacco, still produces more preventable lung cancer deaths than many of the carcinogens discussed by Henderson *et al.* Radon is not mentioned in the paper. Henderson *et al.* mention only diagnostic x-rays as a cause of leukemia. Yet benzene, a proven cause of human leukemia, is widely distributed in the air and water. Preventable workplace exposure to benzene still continues. The authors note that ultraviolet light (UV) is a cause of malignant melanoma, yet do not mention the role of environmental pollution in the thinning of the ozone layer. This preventable event will result in an increase in UV exposure and thus a higher incidence of all types of cancer.

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Response: We agree with Goldstein that every effort should be made to prevent cancers that are induced in the workplace. We did not mean to "disregard" the contribution of occupational exposure to cancer occurrence but, with the possible exception of asbestos-induced lung cancer, we are unaware of any particular occupational exposure that would be considered a *major* cause in the general population of any of the specific cancers that we discussed.

We agree with Goldstein that ozone depletion may have an effect on cancer incidence, not just on melanoma occurrence but also on other skin cancer. However, ozone depletion is a relatively recent phenomenon and is still mainly confined to polar regions, so any measurable increase of cancer would occur in the future (*1*). Even so, such an increase may be preventable through lifestyle changes aimed at avoiding severe sun exposure, particularly during childhood, which is true for all forms of skin cancer.

Radon is a known cause of human lung cancer in occupational settings, and it has been estimated that indoor radon exposure might result in as many as 16,000 lung cancer deaths annually in the United States (mainly in smokers) (*2*). However, numerous assumptions, regarding both exposure assessment and dose-response curves, must be made in order to derive such a conclusion, and no convincing epidemiologic study has yet been performed. This is an important issue that is being actively studied.

To our knowledge, no estimates have been made of the contribution of current ambient benzene exposure to the risk of acquiring leukemia in the general population (occupational benzene exposure is an established cause of myelogenous leukemia).

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1. T. M. Mack and B. Floderus, *Cancer Causes Control* **2**, 401 (1991).
2. A. V. Nero, *Cancer Prev.* **89**, 1 (December 1989).

Erratum: In the article "Benefits and costs of HIV testing" by D. E. Bloom and S. Glied (28 June, p. 1798), the Americans with Disabilities Act of 1990 should have been described as Public Law 101-336.

Erratum: In the Research News article "Is nitric oxide the 'retrograde messenger'?" by Marcia Barinaga (29 Nov., p. 1296), in the illustration on page 1297, the word "citroline" should have read "citrulline".