Letters

Whistle-Blowing

Daniel E. Koshland, Jr.'s, editorial (29 Apr., p. 585) on whistle-blowing proposes an ugly theory of justice, that the value of a charge of fraud depends on the relative performance records of whistle-blower and accused. In practice, this would confer absolute immunity on important scientists.

"Excesses in whistle-blowing and journalism" are Koshland's own invention. Typically, the whistle-blower wants only to get the offending practice stopped or the offending paper withdrawn. Publicity is not wanted because a reputation for whistle-blowing hurts one's employment prospects. It is only if it becomes plain that the offense is being whitewashed that the whistle-blower, or someone else, may go to the press.

The press thus serves its ancient role as court of last resort. Koshland suggests instead that it carry stories about what kind of a scientist the whistle-blower is and impute motives and character traits, all irrelevant to whether the whistle-blower's charge is true. The press should not do character assassinations in the service of the scientific establishment.

C. W. McCUTCHEN
5213 Acacia Avenue,
Bethesda, MD 20814

Koshland's editorial "Science, journalism, and whistle-blowing" is an excellent statement that is precisely on the mark.

I have been interested in this subject for a number of years and, as the American Bar Association liaison to the National Conference of Lawyers and Scientists (NCLS), have participated in the discussions concerning the NCLS sponsorship of the workshops on "fraud and misconduct in science." Since the initiation of these discussions I have objected to the use of the term "misconduct" on the grounds that the term is so broad and vague that it could include virtually anything that some person disapproves of. I have urged that the title of the workshops should be "fraud and misrepresentation in science" and have argued that this is not merely a matter of verbiage but that it is likely to focus attention on the real problem of the occasional false representation of scientific data in published reports.

While the issue of the proper focus of such discussions has been my principal concern in these matters, I agree entirely with the other points that Koshland makes with respect to the investigations that are appropriate when fraud is charged and the considerations that are relevant in appraising both specific charges and general reports about the prevalence of fraud in science. I hope that Koshland's well-considered editorial will be read by all of those concerned with this subject and will have the influence that it deserves.

LEE LOEVINGER
Hogan & Hartson,
555 Thirteenth Street, NW,
Washington, DC 20004-1109

Chinese Diet Study

While the National Cancer Institute and the Chinese government provided the major sources of funding for the project on diet and health in China (Research News, 1 Apr., p. 27), I also wish to call attention to important additional sources of funding from the American Institute for Cancer Research, the U.S. Food and Drug Administration, the U.K. Imperial Cancer Research Fund, and several private U.S. companies.

T. COLIN CAMPBELL
Division of Nutritional Sciences,
Cornell University,
Ithaca, NY 14853–4401

Etiology of AIDS

In the first paragraph of William Booth's News & Comment briefing of 15 April (p. 279), I was described as an ally of Peter Duesberg and characterized as particularly unacquainted with the AIDS literature. I welcome this opportunity to reply. In my formal presentation I explicitly stated my disagreement with Duesberg's categorical denial of a role for HIV in the origin of AIDS, although I commended him for raising a serious discussion of the subject. In Booth's passing shot at my knowledge of the AIDS literature, I am afraid he mistakes my critical questioning for ignorance. Although I have not worked with the retrovirus known as HIV (human immunodeficiency virus), or with the AIDS complex of diseases, I do have some credentials for having been invited. I initiated modern work on retroviruses in cell culture some 35 years ago (*1*) and thoroughly studied their epidemiology after developing the laboratory tools for doing so (*2*). I reversed my initial refusal to attend when I sensed the meeting was designed to discredit my colleague Duesberg. He insisted I come despite our points of disagreement because he knew I was not committed to either side of the dispute, which says something about his attitude.

From what I learned at the meeting and its aftermath I have concluded that my objections to Duesberg's major thesis may have been based, paradoxically enough, on my inadequate knowledge of all the nuances in the field. Let me summarize my interpretation of his position and try to explain my own modified position. Duesberg points out that AIDS patients and individuals in the high-risk groups (multipartner male homosexuals and intravenous drug abusers) not only have a high incidence of HIV infection, but have a similarly high incidence of infection with cytomegalovirus, Epstein-Barr virus, herpes virus, and hepatitis B virus (*3*). At least one of these, cytomegalovirus, produces the same deficiency of T4 (CD4) helper cells as is seen in AIDS (*4*). Patients also have a high incidence of gonorrhea, syphilis, and other sexually transmitted diseases (*5*). Since these agents are, for the most part, more readily transmitted than HIV, the latter is likely to be a good indicator for the presence of one or more of the other agents. The well-known difficulty of HIV transmission also explains why it is less prevalent than the other agents in low-risk groups, which gives it the *appearance* of specificity. If we assume that AIDS is a consequence of multiple infections and associated practices (see below), the indicator status of HIV explains why there is a strong relation between its presence and manifestations of AIDS, although it may not be directly causal.

Another major consideration in questioning a unitary cause of AIDS is the complexity of the syndrome. I counted some 20 diseases subsumed under this category, some with several subclasses (*6*). In the light of this growing list, the imminent addition of new disease entities is likely. None of the diseases is new, but they certainly occur more frequently and more severely than before in the high-risk groups. Both major high-risk groups have increased dramatically in number, in local concentration, and in the intensity of their activity with the advent of gay liberation (*7*) and the well-publicized drug epidemic. In many cases the jusitification for diagnosing the disparate diseases as AIDS is the presence of HIV antibody, which amounts to circular reasoning in supporting HIV causation. The other side of the circular coin is the rejection of AIDS diagnosis in the absence of HIV antibody.

A major feature of AIDS is a lowered T4 helper lymphocyte count, but this is a common feature of other severe diseases and is probably determined more by the state of the host, including hormonal status, than by direct infection of this set of lymphocytes. Indeed, the remoteness of the latter possibility in AIDS has been repeatedly emphasized by Duesberg in light of the tiny fraction

(less than 1 in 10,000) of actively infected lymphocytes and the low pathogenicity of HIV for the few cells that are infected.

Although the case for HIV causation of AIDS is largely based on guilt by epidemiological association, that same epidemiology raises serious questions about the rigor of the association. The rate of onset of clinical AIDS in the high-risk groups is 5% per year (*8*). The Public Health Service estimates that there are 1 to 1.5 million HIV-positive individuals in the country (*5*). If they were at equal risk of developing the disease as the HIV-positive individuals of the high-risk groups, there should be 50,000 to 75,000 new cases per year, but the estimated number for 1986 was 16,000 (*5*). The three- to fivefold shortfall of cases implies that the probability of developing the disease in HIV-positive individuals varies greatly with the behavior of those individuals. It is not inconceivable that the probability approaches zero in heterosexuals with pure HIV infection who do not abuse drugs, as Duesberg has surmised.

Duesberg has implicated what is euphemistically called life-style in the onset of the AIDS complex of diseases. This category includes drug abuse, repeated receptive anal intercourse, and excessive use of antibiotics to treat repeated bouts of gonorrhea, frequently on a prophylactic basis (*9*). The latent period of more than 5 years between HIV infection and overt expression of AIDS symptoms could be an illusion created by the need for chronic or repeated exposure to an array of microorganisms and the cumulative effects of practices that have been shown to be immunosuppressive (*9, 10*).

Duesberg has been condemned as irresponsible because his views allegedly encourage unsafe practices such as sex without condoms and the intravenous use of contaminated needles. In fact, his view of HIV as an indicator for infection by other agents would logically call for even greater sanitary precautions. Indeed, his detractors might well be tarred with the same brush for discounting the role of immunosuppressive practices and for unwarranted reductionism of such a complex syndrome.

I cannot rule out some role for HIV, in concert with other factors, in the development of the complex morbidity we call AIDS, although its effect on the immune system might be indirect. There is, however, a clear need to widen the scope of epidemiological investigation to include the diverse factors mentioned above. Given the statistical problems inherent in AIDS, the citing of anecdotal cases among infants and partners of hemophiliacs—without documenting numbers and distinguishing between HIV infection and AIDS symptoms—as was done at the 9 April Washington forum, borders on the demagogic. In all fairness to present, and particularly to potential, victims of this scourge, we ought to proceed to a balanced, nonconfrontational consideration of the true nature of the problem and of all the factors involved. Where lives are at stake, the enemy should not be the other side in a scientific dispute, but the disease itself.

HARRY RUBIN
Department of Molecular Biology, University of California, Berkeley, CA 94720

REFERENCES

1. H. Rubin, *Virology* **1**, 445 (1955); H. Temin and H. Rubin, *ibid.* **6**, 669 (1958).
2. H. Rubin *et al.*, *ibid.* **17**, 143 (1962).
3. P. Duesberg, *Cancer Res.* **47**, 1199 (1987).
4. W. P. Carney, *et al.*, *J. Immunol.* **126**, 2114 (1981).
5. Institute of Medicine, *Confronting AIDS* (National Academy Press, Washington, DC, 1986).
6. *J. Am. Med. Assoc.* **258**, 1143 (1987).
7. F. Fitzgerald, *Cities on a Hill* (Simon & Schuster, New York, 1986).
8. R. Detels, paper presented at a forum sponsored by the American Foundation for AIDS Research, Washington, DC, 9 April 1988.
9. H. L. Coulter, *AIDS and Syphilis: The Hidden Link* (North Atlantic Books, Berkeley, CA, 1987).
10. A. G. Gilman, L. S. Goodman, A. Gilman, *The Pharmacological Basis of Therapeutics* (Macmillan, New York, ed. 6, 1980).