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LETTERS

Helping Physicists Market Themselves

The News article "Young scientists' network shakes up the establishment" by David H. Freeman (1 Oct., p. 24) points out, accurately in my judgment, the severe oversupply of young physics professionals in relation to the availability of jobs among their traditional research employers—universities and government and corporate laboratories. The Young Scientists' Network, some of whose leaders have been angry at the community of established physicists for "misleading" them about this situation, have taken on the American Physical Society (APS) in order, in some way, to redress the "wrongs" done them and to improve the situation for still younger students. This would be a fine idea if the APS were in a position to alter the attitudes and practices of its senior academic members; unfortunately, it isn't.

The best solution to these serious concerns, in my view, is not to limit the input of students to graduate schools, or to reduce the number of such schools (through such methods as professional accreditation), or to constrain the employment options of non-U.S. citizens. Rather, the solution lies with the tenured faculty of the Ph.D.-granting institutions, as individuals, who each should (i) think more broadly about the kinds of jobs their students could do, outside of the traditional, saturated markets; (ii) set an ethos where seeking these broader classes of jobs is not looked down upon as third-class; (iii) reconsider and modify the standards of training that Ph.D. students receive to better prepare them for the alternative careers available today and in the future (such modifications to include interdisciplinary collaborations with engineering, computer science, and biomedical disciplines); (iv) broaden their own research interests and funding sources and actively explore research collaborations to include the research interests of alternative employers of physicists; and (v) become familiar with, and promote among students, conventional, real-world career assessment and job search techniques, so that all students become confident and capable with these critical skills.

Both students and professors are today sadly unaware of how to market themselves effectively to employers. Although the APS has undertaken to bring workshops on career assessment and job searching to interested campuses, on a cost-sharing basis, such a program cannot succeed without its being institutionalized on campuses as a regular part of students' training.

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Programmed Cell Death and AIDS

The Perspective "Apoptosis in AIDS" by Marie-Lise Gougeon and Luc Montagnier (28 May, p. 1269), although interesting, puts forward several controversial suggestions and does not mention some pertinent points.

First, while there is now a good deal of evidence that in vitro activation of mature T cells from human immunodeficiency virus (HIV)–infected individuals by polyclonal stimuli (such as anti-CD3 monoclonal antibodies or calcium ionophore) induces apoptosis in a fraction of $CD4^+$ and $CD8^+$ T cells (*1, 2*), the impression given in the Perspective is that cells from uninfected individuals do not undergo detectable apoptosis under similar conditions. This is not so. We and others routinely detect "background" apoptosis in as many as 10 to 15% of mature T cells from uninfected individuals after activation for 3 days with polyclonal stimuli (*2, 3*). This background apoptosis is large enough to produce the "DNA ladder" typical of apoptosis when DNA from these cultures is assayed on agarose gels. Thus, although HIV may well accelerate and increase apoptotic cell deaths after in vitro activation, this is not an all-or-nothing phenomenon.

Second, Gougeon and Montagnier also suggest that "the time has come to fight this complex disease by combinations of several treatments including antivirals, antibiotics, and anti-apoptotic drugs." Because the role of apoptosis in HIV-induced T cell depletion in vivo remains to be elucidated, this may or may not be true. However, as apoptosis is the normal physiological cell death mechanism (and as such is vital for the control of proliferating cell populations and for the deletion of autoreactive T and B cells), drugs that can inhibit HIV-induced apoptosis may also interfere with apoptotic cell death in many other cell lineages. This possibility warrants at least a passing mention. In addition, apoptosis-inhibiting drugs may be ineffective on syncytium-inducing (SI) HIV