SCIENCE

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LETTERS

AIDS Research Priorities

The "comprehensive research plan that sets the scientific priorities" to deal with the AIDS plague, as presented by William E. Paul, the Director of the Office of AIDS Research at the National Institutes of Health (NIH) (Policy Forum, 3 Feb., p. 633), is heartbreakingly inadequate. Passing the buck to yet another committee is not what we need. Paul is asking for a year for the committee to get themselves up to some sort of par. No doubt they will only put out another report that will take another year to get read, another year to get peer reviewed, another year to get funded, and another year to start operating. I could form a committee of experts and have a report in 3 weeks. Paul's plan seems mired in typical government bureaucracy. In no way that I can see does it embody the kind of urgency that Congress or Senator Edward Kennedy (D–MA) had in mind when they set up this office. William Haseltine has predicted 1 billion infections shortly after the arrival of the new century. Does not the specter of 1 billion sick people scare *anyone* enough to stop with the commissions and the committees and roll up their sleeves and get down to work?

To me a plan means strategy, priorities, direction, goals, results. Any corporation director would know exactly what I'm talking about. Find the weak spots, hire the best people to fill these spots, demand results for the funds expended, or fire the laggards and refill the positions. No progress is accomplished without an efficient system, without specific goals. Where are the goals and vision in Paul's plan?

Drugs and combinations of drugs can be tested on macaque monkeys speedily and answers made available in months, not the years expensive trials on humans require. "There is already an active program of primate research," Paul tells us, before discussing the need for cooperation. But if those involved aren't cooperating with one another, how can it be an active program? An active program works and goes forward, and everyone knows about it. That monkey research is not accelerated and coordinated and that it takes 2 years just to requisition and obtain a monkey in the NIH bureaucracy are paramount issues that Paul and his panel must deal with. What do we have an Office of AIDS Research for (as well as an AIDS czarina), if not to eliminate red tape for things like this? Even more to the point, why doesn't Paul come down out of the ivory tower, take some of the millions that Congress has given him, and just go out and buy some monkeys on the open market and get to work?

Paul's plan trumpets "investigator-initiated research proposals" as if a new religion has arrived. But the process each newly annointed "thoughtful" scientist must go through to get funding is just the same as that old time-gobbler, one "fully determined by the procedure of peer review as part of the entire competitive process." Competition and peer review are part of the very system that can reward those adept at grantsmanship and punish those who are brilliant in the lab.

If the new Congress is intent on seeing bigger bangs for their bucks, then it's time NIH face the fact that if they aren't delivering them, their salaries, their budgets, and their billions may soon be diverted directly to those places of independent research that deliver much greater value for the money expended. AIDS activists long ago discovered what the new Republican Congress is just beginning to sense: not only has there never been a cure for any major illness to come out of NIH, but, for $14 billion each and every year, the taxpayers are getting rotten value for their money.

*Larry Kramer**
2 Fifth Avenue, New York, NY 10011, USA

*Co-founder, Gay Men's Health Crisis. Founder, ACT UP. Author, *The Normal Heart* (Plume, New York, 1985); *Reports from the Holocaust: The Story of an AIDS Activist* (St. Martin's, New York, 1995).

Paul's assertion that a turning point has been reached in ordering priorities for AIDS research is especially welcome at a time of increasing criticism of established-discipline only approaches to HIV/AIDS.

The NIH policy evaluation panel, in developing recommendations for future directions, has unprecedented opportunities. First, they cannot only invite, but actively solicit, new ideas and alternative lines of inquiry. We suggest a review of idiopathic $CD4^+$ lymphocytopenia, autoimmune disorders, and endogenous retrovirus activation. Since these and other new approaches would be expected more often in investigator-initiated proposals than in requests for applications, placing a high priority on the former will advance the goals of the plan. Second, they can weigh advice and testimony from research-

ers in commercial settings as well as from those representing government and academia. Industry-based researchers have a substantial record of aggressively pursuing and developing HIV/AIDS science and technologies. Third, they can accelerate research communications by encouraging publication of discoveries and results on electronic information (Internet) highways—keeping in mind requirements for a peer review system and its evolution.

We join others in applauding the policy plans and suggest the changes will do much to attract imaginative people and to support productive lines of inquiry. That, in turn, will drive advances in HIV/AIDS science and medicine. Build it and they will come.

Howard B. Urnovitz
Roy W. Stevens
Calypte Biomedical Corporation,
1440 Fourth Street,
Berkeley, CA 94710, USA

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AIDS: Modeling Epidemic Control

We would like to comment on the report by Sally Blower and Angela McLean "Prophylactic vaccines, risk behavior change, and the probability of eradicating [human immunodeficiency virus] HIV in San Francisco" (2 Sept., p. 1451). They use a mathematical model of disease transmission to explore the possibility of eliminating (*1*, *2*) HIV in that city. They conclude that, for certain parameter values, and under the model's assumptions, elimination of the virus would be unlikely or impossible, even with 100% vaccination coverage. There are, however, key factors Blower and McLean do not include in this study that make the potential use of a prophylactic HIV vaccine attractive.

First, the only efficacy characteristic included in the model is the reduction of susceptibility to infection. The most important effect of many vaccines, however, is to prevent or reduce clinical disease or to reduce shedding of the disease agent in already infected persons. For example, oral polio vaccine may not always prevent infection by wild polio virus, but it prevents paralytic disease and reduces intestinal and nasopharyngeal viral shedding of wild polio virus (*3*). Inactivated polio vaccine has a similar, but less dramatic effect on reducing viral shedding (*3*). In the case of chicken pox, many vaccinated persons become infected, but symptoms, when they occur, are mild (*4*). A further example is the unexpected success of *Haemophilus influenzae* type b vaccine, which decreases transmission by reducing nasopharyngeal carriage of the virus in infected, vaccinated persons (*5*). This is beneficial to unvaccinated younger children and infants. HIV viremia is generally high during the primary infection period (for example, the first 3 to 12 weeks of infection) and then drops precipitously (*6*). In the HIV pandemic, there is evidence that transmission often occurs during this period (*7*). Reduced infectiousness decreases the intensity of an epidemic in proportion to the reduction of viral shedding and the fraction of the target population that is vaccinated. Including this relation in the model could reduce to a plausible percentage the vaccination coverage that would be required to eliminate HIV.

Second, Blower and McLean imply that reduction of HIV incidence could be a secondary goal if elimination of the virus proves to be impossible. Reduction of incidence and overall morbidity is more often the *primary* goal of a vaccination program than is elimination, and we believe that HIV vaccines should be judged on this basis. A vaccine against primary chicken pox, for example, would not eliminate varicella-zoster virus because of the reservoir of latent virus that reactivates to produce shingles (*2*, p. 22).