Letters

The Cause of AIDS

Peter Duesberg (Policy Forum, 29 July, p. 515) cites the prospective study of AIDS, in men infected with human immunodeficiency virus (HIV) carried out here at San Francisco General Hospital (*1*) as evidence that "discrepancies between the epidemiologies of HIV antibody and AIDS indicate that neither HIV nor antibody to it is sufficient to cause AIDS." His point appears to be that our study, the only study of homosexual men cited, shows rates of progression to AIDS in HIV antibody-positive homosexual men that are different from those observed in hemophiliacs and in the population as a whole. This is the opposite of the conclusion we reached. We reported a progression rate of AIDS of 22% after an estimated 6 years of infection and predicted a progression rate of 49 to 52% after 9 years of infection; we observed that these results were consistent with the other principal studies of both homosexual men and hemophiliacs. We found no evidence of "factors defined by lifestyle, health, gender, and country of residence" that might affect progression to AIDS, other than age.

Our study is also consistent with progression rates in the country as a whole, if one assumes, as we do, that the total number of HIV antibody-positive people in the United States is 500,000 to 700,000 (*2*).

Duesberg is the latest and most successful in a long line of "contrarians" in AIDS, including the proponents of immune overload and the believers in mosquito transmission. His campaign has had the useful effect of reminding us how little we understand the pathology of HIV infection. However, his interpretation of what the epidemiology of AIDS tells us is the opposite of that of most epidemiologists. The consensus of epidemiologists is increasingly that most persons with antibody to HIV will progress to clinical AIDS.

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Duesberg's position flies in the face of a large and convincing body of epidemiologic data. We believe that the popularity which Duesberg's views have been accorded by segments of the lay public has its roots in denial—a well-known psychological defense mechanism which allows an individual to cope, especially in times of stress. People may deny the gravity of a traumatic situation either "By saying something to the effect that it cannot be so, or by trying to invalidate something intolerable by deliberately ignoring its existence" (*1*).

Recent studies (*2*) have suggested that the vast majority of the approximately 1 million Americans infected with HIV will die of AIDS or other HIV-related pathology. Denial has taken different forms as both the epidemic and the scientific understanding of it have progressed. In 1985 14% of gay men in San Francisco who were surveyed denied that their clearly unsafe behaviors put them at risk of infection (*3*). With the widespread availability of antibody testing, much of this population has learned of their own infection, or of the infection of former lovers or close friends. Faced with irrefutable results of what has become one of the most reliable testing systems in modern medicine, the easiest path to comfortable denial may now lie in a seemingly scientific argument that either (i) presence of antibody to HIV does not mean that an active or transmissible infection exists or (ii) even if such an infection does exist, it has no grave prognosis.

Denial can allow a person to continue to function in the face of overwhelming stress and, with time, to make use of other less radical defenses (*4*). Thus, it is not harmful unless it prevents a person from making more appropriate adjustments. Unfortunately, denial can have deadly consequences in the face of an epidemic such as AIDS.

It is unfortunate that seemingly scientific arguments supporting such deadly denial have been widely disseminated to a vulnerable population. We hope their presentation and refutation in the *Science* Policy Forum will allow the debate to progress to a discussion of the management of denial in susceptible populations and of how to minimize its adverse effects on the health of the public and the spread of the epidemic.

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In response to the letters of Moss *et al.* and Trachtenberg and Winter and to the statements made by Blattner, Gallo, and Temin in the Policy Forum of 29 July, I submit the following: The Centers for Disease Control (CDC) reports American AIDS cases on the basis of two different criteria. (i) the presence in the patient of antibody to a latent retrovirus, termed HIV, currently confirmed in about 40% of U.S. AIDS patients (*1*) and (ii) the inclusion of the patient in an AIDS-risk group, confirmed in about 95% of all AIDS cases (*2*). Most researchers think that AIDS is caused by HIV. However, AIDS occurs only an average of 8 years after infection (*3, 4*). During AIDS and in the preceding years, HIV is neutralized by the same antiviral immunity that is diagnosed with the "AIDS test." Only 1 of every 10,000 T cells lost during AIDS is infected with HIV. But while the virus remains idle, 95% of its hosts who develop AIDS take drugs, an activity that is often associated with malnutrition, are promiscuous male homosexuals with frequent parasitic infections and drug use (*5*), or receive regular transfusions for hemophilia, each for an average of 8 years (*2*). Indeed, these specific AIDS risks typically define subsequent AIDS diseases, for example, Kaposi's sarcoma exclusively in homosexuals and mostly pneumocystis in injective drug users (*6*).

In view of this, an unbiased investigator could propose three working hypotheses for AIDS: (i) AIDS is caused only by HIV. The 8-year latency, the more than 20 AIDS diseases (*1*), and the severe loss of mostly uninfected T cells would all be due to the many mysterious properties encoded in the tiny 9-kilobase genome of HIV (*7, 8*) (ii) AIDS diseases are caused only by AIDS risks. The specific combinations of these risks would generate specific diseases. The average of eight asymptomatic years would be the thresholds of pathogenicity. (iii) AIDS diseases are caused by AIDS risks and HIV together. To find a correct hypothesis, one must know the incidence of AIDS diseases in controlled cohorts of risk-takers or non-risk-takers, with and without antibody to HIV. But such studies are not available.

The study conducted by Moss *et al.* on the "progression to AIDS" in a cohort of antibody-positive homosexuals from San Francisco is not representative of the popula-

tion "as a whole." Their conclusion that there is "no evidence of 'factors defined by lifestyle, health . . .' that might affect progression to AIDS, other than age" is not convincing unless antibody-positive heterosexual controls of the same age are included. There is also no "consensus of epidemiologists" on this claim, as two studies postulate such factors in the generation of AIDS (*3, 9*).

Similarly misleading is the suggestion of Blattner, Gallo, and Temin that the conversion to AIDS by a single antibody-positive health care worker [reported anonymously (*10*) without data on gender, latent period, or symptoms] proves that HIV causes AIDS. This one case falls into the ~5% window of all American AIDS cases that have no verifiable AIDS risks (*2*), while 95% of the 2586 health care workers with AIDS fall into the conventional risk groups (*10*). Moreover, despite much greater exposure to HIV (*10, 11*), the AIDS risk of health care workers is exactly the same as that of the general population (*2, 10*). Even the sexual distribution of AIDS cases is the same as that of the general population, namely, 92% of these cases are male, although 75% of the health care workers are female (*10*). Thus, unless the percentage of health care workers with AIDS who do not belong to the known risk groups exceeds that of the rest of the population and reflects their sexual distribution, such isolated cases are statistically irrelevant.

Trachtenberg and Winter are concerned that my "seemingly scientific" views serve as a psychological narcotic, allowing a "vulnerable population" to deny their fate. This concern is warranted only if their own, presumably more scientific, arguments that infection with HIV is necessary and sufficient to cause AIDS are correct. However, since HIV is nearly undetectable in AIDS and the pathology is not understood, the consensus of the "HIV establishment" that HIV causes AIDS appears to be "deadly" denial of biochemical alternatives. Since all other viral and microbial pathogens are biochemically very active in or on more cells than the host can spare or regenerate when they cause a fatal disease, I favor hypotheses that offer biochemically tangible bases for AIDS, namely drugs, acute infections (*5*), and malnutrition (*12*). If latent viruses or microbes were pathogenic at the level of activity of HIV, most of us would have pneumocystis (80 to 100%) (*13*), cytomegalovirus disease (50%), mononucleosis from Epstein-Barr virus (50 to 100%), and herpes (25 to 50%) (*14*) all at once, because the respective pathogens are latent, immunosuppressed passengers in the U.S. population at the percentages indicated.

In view of all of the above, it is "unfortunate" that the toxic AZT therapy of symptomatic and even asymptomatic antibody carriers designed to inhibit DNA synthesis of latent HIV (*15*) and the psychologically toxic AIDS test "have been widely disseminated to a vulnerable population."

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