can at least partially neutralize HIV *in vitro*. Kennedy is optimistic that these results indicate good possibilities for a vaccine.

It appears that anti-idiotype antibodies are also able to modulate the immune response against tumors. Dorothee Herlyn (Wistar Institute, Philadelphia, PA) reported the results of preliminary clinical trials (held in West Germany) on colon carcinoma patients who also have metastatic tumors in various organs. In this case the Ab1 is monoclonal 17-1A, which is specific to a non-circulating cell surface antigen associated with human colon carcinoma.

Patients treated with Ab1 raised anti-idiotypic antibodies against Ab1. The Ab2s mimicked the antigen *in vitro*. When the doctors removed B cells from patients who were seropositive for anti-idiotype antibody and stimulated them with Ab2 *in vitro*, the B cells produced Ab3 with the same binding specificity for carcinoma cells as the Ab1. When patients were treated with Ab2s, they produced Ab3s, which Herlyn says were specifically induced by the Ab2s. One patient treated with Ab2 had numerous liver metastases; nine months later, they are gone. Herlyn postulates that the autologous Ab3 anti-id response may induce cytotoxic T lymphocytes to lyse tumor cells.

In principle, anti-idiotypic vaccines should also work for parasitic infections. David Sacks (National Institutes of Health, Bethesda, MD) says that the carbohydrate epitopes on the surfaces of parasitic protozoa and helminths are highly immunogenic. Responses against these carbohydrates seem to be especially important in inducing immunity. Sacks has focused on studying *Trypanosoma cruzii* (the cause of Chagas' disease) and *Leishmania*. He says that, in the vertebrate host, it is quite clear that these parasites' surface carbohydrates are critical in initiating infection by interacting with receptors on the target cell. (*Leishmania*, for instance, infects and lives in macrophages: It interacts with receptors for mannose, fucose, lectins, and C3.)

Sacks says that anti-ids can induce anti-parasite-carbohydrate responses in a number of species, including mice, guinea pigs, and rabbits. This could indicate that the anti-ids are detecting an interspecies cross-reactive idiotype. Sacks sees great promise for anti-ids: mice immunized with *Leishmania* anti-idiotype antibody had superlative immunity—"as good if not better than we have ever seen with the antigen."

—Jennifer Van Brunt

COMMENTARY

SENSE IN A REIGN OF TERROR

Perhaps the most insidious aspect of the human immunodeficiency virus is that it has already infected the human psyche more pervasively than it has the blood. In the United States, AIDS has done more to change the social and cultural climate than any other event in recent history. For every person harboring an HIV provirus, thousands harbor the *fear* of infection. Given this penetrance, the responsibility we have as scientists becomes acute. We must not only develop effective vaccines and treatments, but we are asked to do so in an increasingly hysterical atmosphere. We therefore need to be particularly vigilant in assuring that epidemiological data regarding the spread of the AIDS virus are not misused to further alarm an already frightened public.

With what we know about the epidemiology of AIDS in the United States, we can make accurate predictions only for well-defined risk groups, and *not* the general population. If we fail to make this explicit—and we too often do—we allow our estimates to be used in ways which manipulate people's extreme fear, and are thus finally counterproductive to efforts aimed at stopping what is rather suddenly the most serious epidemic of the century. A public scared by statistics may demand premature approval and application of vaccines and nostrums.

Moreover, we must direct attention to the clearly recognized common features of the various risk groups. Failure to do so only adds further mystery to a virus that is surely mysterious enough. What seems clear is that HIV spreads most easily, and is most virulent, in an immunocompromised population—one subject to frequent immune stimulation, with a high incidence of persistent viral infections. These characteristics mark the risk groups in developed countries; they are also strikingly widespread in the general population of many African countries, where AIDS is truly epidemic and represents a threat still not sufficiently appreciated.

Recent serologic studies of homosexual men in the United States and heterosexual men in Zaire showed that, in both groups, the prevalence of hepatitis B, cytomegalovirus, and the Epstein-Barr virus was between 90 and 100 percent. AIDS patients from the two countries showed the same pattern—nearly complete infection with these agents (Fauci, *PNAS* **83**:9278-9283, December 1986).

Another *PNAS* paper adds molecular biological support to the idea that these viruses may be cofactors in the infection's spread and productivity (Gendelman et al. *PNAS* **83**:9759-9763, see also *Bio/Technology* **4**:940-941, November 1986). When cells in tissue culture were simultaneously transfected with two plasmids—one containing a chloramphenicol acyltransferase (*cat*) gene under the control of the HIV long terminal repeat, the other containing *trans*-activation sequences derived from a variety of DNA viruses—the researchers found that *cat* expression was induced by genes from JC virus, BK virus, lymphotropic papovavirus, bovine papilloma virus, type 1 herpes simplex, and varicella-zoster virus.

The role of immune challenge in AIDS disease progression has been frequently discussed, and the epidemiological and molecular evidence cited above lend additional credence to the view that activation of infected lymphocytes is a key step in HIV's proliferation and cytotoxicity. Discussion of this question is sure to be invigorated by a Medical Intelligence report in the March 12, 1987 issue of *The New England Journal of Medicine* (**316**:673-676). Robert Redfield and co-workers at the Walter Reed Army Medical Center (Washington, D.C.) describe the case of a patient with HIV infection and subclinical T-cell deficiency who developed AIDS and disseminated vaccinia after receiving a series of immunizations upon entering military service.

The public health implications of these considerations are clear. In educating people about the essential risk factors in AIDS, it is important to emphasize that it is the immunocompromised state, and not only life-style related habits, that permits the virus to spread so effectively. And when counseling antibody-positive, but asymptomatic individuals, we need to stress the role of immune stimulation in disease progression.

—Harvey Bialy