A Negative Exposure

"U.S. women cannot transmit AIDS to their male sex partners, but they can transmit the virus. Most of these men will remain HIV negative."

October 1, 2001 By Joseph Sonnabend, MD

Many in the AIDS establishment deny the long-established epidemiological fact that female-to-male transmission is of negligible risk in the U.S. They often do so by pointing to the devastating explosion of heterosexual AIDS in Africa as proof that HIV is as easily spread from woman to man as from man to woman. But after two decades there is still very little heterosexually transmitted AIDS here in the U.S. The essential difference between the two epidemics is, I believe, in the sexual transmission of AIDS from women to men -- it is extremely inefficient here but much more efficient in Africa. This difference has never been adequately explained. I can propose a testable explanation for these starkly contrasting epidemiological characteristics.

It has long been rather dogmatically asserted that infection with HIV is *always* followed by sufficient viral replication to elicit an antibody response. Thus, after a "window period" of seronegativity lasting up to 12 weeks, seroconversion ensues, and the individual tests positive on the antibody test. There is no doubt that this sequence of rapid seroconversion often occurs and is often associated with disease progression. But there is no basis to justify the assertion that this course of events is inevitable.

It is just as likely -- albeit virtual heresy to propose -- that infection in some individuals is followed by viral replication so limited that it cannot elicit an HIV-antibody response. What it can induce is a so-called cell-mediated immune response. This leaves the viral DNA in a latent state. In such a case, an individual would test negative on the antibody test while showing the presence of HIV by a gene-detection technique such as a PCR test. Such tests, however impractical on a large scale, might reveal that more people carry the HIV genome than are HIV-antibody positive.

So there are two responses to infection: rapid seroconversion and prolonged latent infection in HIV negative individuals. What determines the responses? Two key factors: the size of the infecting inoculum and the presence of signals known to activate HIV DNA, which produces HIV RNA and then the virus' replication. Such signals are common to many infections caused by viruses, bacteria, protozoa and other microorganisms.

Cell-mediated immune responses also play a role in determining the course of infection. These immune cells may destroy HIV-infected cells, and so keep HIV negative an individual "infected" with a small inoculum and free of activating signals. There is ample evidence that these so-called

exposed seronegatives (ESNs) not only exist but also show both anti-HIV cell-mediated immune responses and HIV DNA. It is even possible that any burst of HIV production will prime and strengthen these responses.

As for the size of the infecting inoculum, the male/female differences are obvious: Women are infected (presumably repeatedly) with a relatively large inoculum (in semen), as are some homosexual men. But male sex partners of women with HIV are infected with a relatively small inoculum, possibly leading to a silent infection -- no HIV antibodies but some cell-mediated responses. In this sense it can be said that U.S. women cannot transmit AIDS to their male sex partners, but they can transmit HIV. On the whole, in the absence of any infection-related activating signals, these men will remain negative and healthy. While this hypothesis is testable, the idea of “HIV negative infection” with HIV is needlessly but likely too terrifying not only to ESNs themselves but to everyone who depends on the “safe” no-gray zone between HIV positive and negative.

The crucial difference in Africa is the high prevalence of STDs, TB, malaria and other infections -- due to widespread poverty, lack of hygiene and health care -- that supply signals known to activate HIV. Some infections are also immunosuppressive and, by blunting cell-mediated immune responses, help convert a latent to an active HIV infection. Sexual transmission of HIV is also facilitated by a high viral burden -- another consequence of the activating effect of frequent infections.

One crucial implication of all this science? These associated endemic infections are the primary force driving the African epidemic. That’s why it is absolutely appropriate for the United Nations to devote a large part of its Global AIDS Fund to basic public health measures in Africa that will help control these infections.

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