

# Rethinking

## AIDS

VOLUME 1, NUMBER 1

JUNE 1992

### It's time to re-evaluate the HIV-AIDS hypothesis

In April 1984, U.S. Health and Human Services Secretary Margaret Heckler announced to the world at a press conference that an American government scientist had discovered the cause of AIDS. This claim, made in the absence of the usual scrutiny and debate that is provided by refereed publication, was nonetheless received as fact by the general scientific community, and without further investigation a vast research program was launched. Based on the proposition that a newly identified retrovirus, termed HIV, is responsible for the apparently irreversible destruction of T-helper cells characteristic of AIDS patients, this program has until now been unsuccessful at providing either a vaccine or a cure, and has resulted in public health policies that are of questionable value in preventing the spread of AIDS.

Since 1987, data contradicting a single-virus etiology of AIDS have been accumulating. As a result, a loosely affiliated worldwide network of scientists — The Group for the Scientific Reappraisal of the HIV-AIDS Hypothesis — was formed in an attempt to bring about an impartial investigation of the question that was inadequately considered in 1984: Is HIV really the cause of AIDS?

As an explanation for the origin of AIDS, the HIV hypothesis is implausible because:

#### 1. It contradicts a number of established principles of virology and immunology:

—Retroviruses do not typically kill their host cells. On the contrary, they depend on continued replication of the host for their own survival (Weiss et al., *Mol. Biol. of RNA Tumor Viruses*, 1985, Cold Spring Harbor Press, NY). It is therefore improbable that a retrovirus would have evolved that kills its only natural host with an efficiency close to 100%, and yet is horizontally transmitted as inefficiently as HIV. It is even more unlikely that two such viruses (HIV-1 and HIV-2), which differ by almost 50% in their nucleotide sequence (GeneBank), would have simultaneously evolved.

—Pathogenic viruses typically cause disease as a consequence of infecting, replicating in, and killing more cells than a host can regenerate or spare. Complete proviral HIV has only been demonstrated in one in several thousand peripheral

blood mononuclear cells (in both asymptomatic and symptomatic persons), and HIV RNA is detected in 1 in 10,000 to 1 in 100,000 such cells (Simmonds et al., *J. Virol.* 64:864, 1990). Yet 5% of the body's T-cells are regenerated in the two days it takes HIV to establish an infection (Guyton, *Textbook of Medical Physiology*, 1987, WB Saunders, Phil.).

—Viruses typically cause disease shortly after infection, before the immune systems of their hosts can respond. There is no other example of a viral pathogen which causes primary disease only after long and unpredictable latent periods, only in the presence of neutralizing antibodies, and in the virtual absence of gene expression, as HIV is said to do.

#### 2. It is at variance with a growing body of empirical observations:

—Antiviral immunity to HIV is sufficient to keep infectious virions in cell-free serum below the limits of detection until the final stages of AIDS when B-cell immunity is lost and HIV (along with all other chronically latent viruses) is sometimes reactivated (Ho et al., *NEJM* 321:1621, 1989).

—Extensive studies of HIV gene structure and function have neither identified any specific determinants of pathogenicity, nor shown it to be significantly different from many other retroviruses, which are not said to cause degenerative diseases (Duesberg, *PNAS* 86:755, 1989).

—Many chimpanzees have been successfully infected with HIV, yet all have remained disease-free until now, up to 7 years later (Weiss and Jaffe, *Nature* 345:659, 1990).

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*RETHINKING AIDS is an occasional publication of the Group for the Scientific Reappraisal of the HIV/AIDS Hypothesis. A. James Trabulse, Publisher, 2040 Polk Street, Suite 321, San Francisco, CA 94109, fax 415-775-1984.*

## Re-evaluating the HIV-AIDS hypothesis (continued)

—HIV is said to be a sexually transmitted virus, yet it is barely detectable in the semen of AIDS patients (Van Voorhis et al., *Fertil. and Steril.* 55:588, 1991).

—The number of HIV carriers in the U.S. has remained constant at one million since 1985, when widespread antibody testing was introduced (Institute of Medicine, *Confronting AIDS*, 1986, Nat. Acad. Press, Wash. DC, and Vermond, J. *NIH Res.* 3:77, 1991), yet new viruses spread exponentially in a susceptible population (Freeman, Burrows *Textbook of Microbiology*, 1979, WB Saunders, Phil.).

—AIDS has remained confined to the same risk groups since it was first identified as a new disease syndrome, and there are many fewer cases than predicted. Forecasts of the spread of AIDS continue to be falsified, with one notable exception, that is the model published by Bregman and Langmuir—Farr's Law Applied to AIDS Projections (*JAMA*, 263:1522, 1990). This model predicted a crest in the AIDS epidemic in 1988-1989, with a subsequent decline to an endemic level. Data from the July, 1991 public domain diskette compiled by the Centers for Disease Control tends to confirm such a crest in 1989-1990.

—Approximately 75% of American hemophiliacs have been infected with HIV for more than 7 years (*Confronting AIDS*, op. cit.). According to the HIV hypothesis at least 50% should have died of AIDS by now, yet mortality among hemophiliacs has not increased (Koerper, In: *AIDS Pathogenesis and Treatment*, Levy (Ed.), 1989, Marcel Dekker, NY) and only 2% of HIV-positive hemophiliacs develop AIDS indicator-diseases annually (CDC, *HIV/AIDS Surveillance*, 1986-1991, US Dept. of Health and Human Services, Atlanta, GA).

—The same diseases are found in similar frequencies in HIV positive and HIV negative intravenous drug users, and the overall mortality in the two groups is the same (Stoneburner et al., *Science* 242:916, 1988).

Despite these and many other inconsistencies, the HIV-AIDS hypothesis remains the sole basis for public health policies that are aimed at controlling the spread of AIDS by advocating (1) "safe-sex" practices, (2) the use of "clean" needles to inject toxic, unsterile drugs, and (3) the long-term administration of potent metabolic poisons, like AZT, which are

Future issues of *Rethinking AIDS* will be devoted to analyses of the variances between HIV-theory and AIDS-reality, an examination of the various estimates of the number of HIV-infected cells and the correlation between these numbers and disease progression, a consideration of the meaning and implications of the growing consensus that HIV-pathogenesis may depend on chemical or infectious "cofactors," the addressing of unanswered questions about the natural history of HIV and the proposal of controlled epidemiological and other studies designed to critically test the HIV-AIDS hypothesis. The editors welcome contributions reflecting all facets of the debate. We ask only that they be concise and appropriately referenced.

claimed to prolong the lives of HIV-infected persons; and for research programs directed almost exclusively at developing pharmaceuticals designed to interfere with HIV replication.

It is in the interests of formulating an approach to the prevention and cure of AIDS consistent with what we really know that we call for a re-evaluation of the evidence for and against the HIV-AIDS hypothesis. It is the obligation of scientists to ask the most unpleasant and difficult questions of even the most cherished theories, especially when the answers may prevent possibly needless suffering and loss of life.

—The Editors

The Group for the Scientific Reappraisal of the HIV/AIDS Hypothesis came into existence as a result of our efforts to get the following four sentence letter published in a number of prominent scientific journals. All have refused to do so.

"It is widely believed by the general public that a retrovirus called HIV causes the group of diseases called AIDS. Many biomedical scientists now question this hypothesis. We propose that a thorough reappraisal of the existing evidence for and against this hypothesis be conducted by a suitable independent group. We further propose that critical epidemiological studies be devised and undertaken."

### TO THE EDITORIAL OFFICE

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