TIMES AND TRENDS

Research in Immunology, and prior to that, the Annales de l’Institut Pasteur, have traditionally offered a significant part of the journal space to scientific exchanges and discussions, as, for example, the Forums. The journal is open to many types of exchange, and various forms can be adopted. Here we open our columns to Dr. P. Duesberg and Dr. L. Montagnier, who have agreed to discuss the matter of whether HIV is the causative agent of AIDS. Below is Dr. P. Duesberg’s starting contribution. We shall later publish that of Dr. L. Montagnier, and invite other contributions on this topic. In particular, we hope that the question of autoimmune disease in AIDS will be further discussed. Contributions should be sent to the Editor-in-Chief, and publication will be very rapid.

The Editors

AIDS: NON-INFECTIONOUS DEFICIENCIES
ACQUIRED BY DRUG CONSUMPTION AND OTHER RISK FACTORS

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The first 5 AIDS cases, described in 1981, were all male homosexual drug users with pneumocystis pneumonia and acute cytomegalovirus infections (Gottlieb et al., 1981). Since then, over 100,000 cases of about 25 conventional diseases including predominantly pneumonia, Kaposi’s sarcoma, lymphoma, dementia, tuberculosis, candidiasis and diarrhea have been recorded as AIDS diseases in the US (CDC, 1990). Over 90% occurred in two major risk groups (CDC, 1990), namely in 20- to 40-year old intravenous drug users and male homosexuals who also consume drugs (Gottlieb et al., 1981; Laupland and Wilson, 1986; CDC, 1987; Darrow et al., 1987; Haverkos and Dougherty, 1988; Rappoport, 1988; Adams, 1989; Chaisson et al., 1989; Weiss, S., 1989; Friedman-Kien et al., 1990). Because acquired immune deficiency is thought to be the common cause of these vastly disparate diseases, they were grouped together as the AIDS syndrome.

There are two competing hypotheses as to what causes AIDS, the virus-AIDS and the risk-AIDS hypothesis.
The virus-AIDS hypothesis, currently held by most medical scientists, in particular virologists, proposes that the retrovirus HIV (human immunodeficiency virus) causes AIDS by killing billions of T cells but only on average 8 to 10 years after infection (Duesberg, 1989a).

a) The virus-AIDS hypothesis is not compatible with orthodox viral pathology. It is paradoxical that:

i) all other pathogenic viruses, HIV hardly infects any cell when it is claimed to be pathogenic (Duesberg, 1987, 1988, 1989a). During AIDS, <1 in 500 lymphocytes contain a latent HIV provirus and only 1 in 10,000 to 100,000 contain an active virus - just as in millions of virus of asymptomatic carriers (Duesberg, 1987, 1989a, 1989b; Schmittman et al., 1989). Therefore, it is difficult to explain a loss of billions of T cells said to be the hallmark of AIDS (Institute of Medicine, Natl. Acad. Sci., 1986, 1988; CDC, 1987, 1990; Duesberg, 1989a). Like the rest of us, even viruses need to do something to get something done.

b) In view of the claims that in vivo HIV kills T cells directly (Baltimore and Feinberg, 1989; Ho et al., 1989) over 99% of the few T cells that ever become infected survive infection to become "reservoirs" of latent HIV (Schmittman et al., 1989).

c) In view of recent claims that viraemia is necessary for AIDS (Baltimore and Feinberg, 1989; Coombs et al., 1989; Ho et al., 1989), the number of infected cells remains the only proof of infection (Duesberg, 1989a).

d) AIDS diseases - by definition (Institute of Medicine, Natl. Acad. Sci., 1986; Coffin et al., 1986) - only occur after the onset of antiviral immunity, a "positive" AIDS test (Duesberg, 1987, 1988, 1989a). All other virus-caused diseases primarily occur before antiviral immunity when they are biochemically most active (Evans, 1989).

e) AIDS diseases only occur after long, unpredictable latent periods, averaging 8 to 10 years with a minimum of 2 years in adults (Duesberg, 1989a), while all other viruses including pathogenic retroviruses cause primary disease within weeks after infection. Such long latent periods are particularly paradoxical if one argues that antiviral immunity is essential for pathogenicity (Duesberg, 1989a).

f) The same virus would take an average of 2 years to cause diseases in children and an average of 8 to 10 years in adults (Institute of Medicine, Natl. Acad. Sci., 1988; Duesberg, 1989a).

g) The same virus is proposed to cause neoplastic diseases such as Kaposi's sarcomas and AIDS lymphomas as well as neurogenic diseases such as T-cell death or AIDS dementia (Duesberg, 1989a). This, in particular, is implausible, since either Kaposi's cells or neurons are ever infected by HIV (Duesberg, 1989a). Indeed, neurons cannot be infected by HIV because retroviruses depend on mitosis to initiate infection (Rubin and Tejin, 1988), and neurons don't divide.

h) Immune deficiency should cause neoplasms such as Kaposi's sarcoma and lymphoma (Duesberg, 1989a). T-cell-deficient humans or animals such as nude mice have no more tumours than immune competent counterparts (Kilien, 1982; Sharkey and Fogh, 1984).

i) The most common AIDS disease in the US is pneumocystis pneumonia while the most common AIDS diseases in Africa are slum disease, diarrhoea and fever, although Pneumocystis carinii is ubiquitous in all humans, including Africans (Duesberg, 1989a).

j) AIDS diseases would occur in the US to over 90% in males, but would be equally distributed between the sexes if they were caused by a sexually transmitted virus introduced into each country about 10-20 years ago (Duesberg, 1989a).

k) Among all risk groups in the US, HIV would cause Kaposi's sarcoma almost exclusively in homosexuals (Duesberg, 1988, 1989a; Beral et al., 1990). It is also incompatible with viral etiology that Kaposi's sarcomas in American homosexuals with HIV are much more frequent than those without (Friedman-Kien et al., 1990).

The virus-AIDS hypothesis is unpreserved, because 1) HIV as the hypothetical cause of AIDS fails to meet many classical criteria of viral pathology. 2) HIV has not been shown to have any unconventional properties to resolve the above paradox (Duesberg, 1989a), and 3) there are no controlled epidemiological studies showing a role for HIV in AIDS. The view that HIV is not sufficient or even not proven to be necessary for AIDS (Duesberg, 1987, 1988, 1989a) has been voiced by a number of scientists including Sylly (1988); Eigen (1989); Evans (1989); Duesberg (1989a); Gilbert (1988); Liversidge, 1989; Griffin (1988); Haas (1989); Holub (1988); Root-Bernstein (1990); Rubin (1989); Schwartz (1989); Sonnabend (1989); Stewart (1989); Weiss (1989); and at least one non-scientist, Fauci's sister Denise (Fauci, 1989); Duesberg (1989a). Moreover, Friedman-Kien et al. (1990) have now called HIV into question as a cause of Kaposi's sarcoma, which used to be the hallmark of AIDS in the early 80's (Beral et al., 1990) for the same reasons I have cited above and previously (Duesberg, 1989a).

The risk-AIDS hypothesis suggests that AIDS is caused primarily by non-infectious agents. These include psychoactive drugs, overmedication with antibiotics (Rappoport,
1988; Rubin, 1988a; Adams, 1989; Sonnabend, 1989) and above all AZT, a chain terminator of DNA synthesis administered to treat HIV infection since 1987 (Duesberg, 1989a; Farber, 1989; Lauritzen, 1989). Consumption of psychoactive drugs is often associated with traditional causes for immune deficiency such as protein malnutrition and parasitic infections (Seligman et al., 1984) and AZT is directly immunosuppressive because it is designed to kill lymphocytes (Duesberg, 1989a; Farber, 1989; Lauritzen, 1989). This hypothesis is compatible with views stated by Root-Bernstein (1990); Rubin (1988a,b); Sonnabend (1989); and Stewart (1989). The risk hypothesis explains:

a) why AIDS is limited to risk groups rather than random in the population, as would be expected from an infectious agent.

b) Why natural vaccination against HIV (a "positive AIDS test") does not protect against AIDS.

c) The long and unpredictable latent periods between HIV infection and AIDS, averaging 8 to 10 years in adults, as the individual reaches a pathogenic threshold of AIDS risks. The shorter latent periods in children averaging about 2 years would reflect prenatal and postnatal risk factors, as 90% of babies with AIDS are either born to mothers who are drug addicts or prostitutes or both, or are haemophiliacs (Duesberg, 1988; CDC, 1990).

d) The enormous diversity and risk-group-specificity of AIDS diseases in terms of the diversity of risk factors. For example, the incidence of Kaposi's sarcoma exclusively in homosexuals correlates and declines directly with their group-specific use of nitrite inhalants (Lauritzen and Wilson, 1986; Haverkos and Dougherty, 1988; Rappoport, 1988; Duesberg, 1989a; Beral et al., 1990) regardless of the presence of HIV (Friedman-Kien et al., 1990). The nearly complete absence between the major AIDS diseases in the US and Africa could be explained as a consequence of drug consumption in the US and of malnutrition and conventional parasitic infections in Africa.

e) The paradox as to why HIV, unless an innocent bystander, can afford to infect actively < 1 in 10,000 and latently < 1 in 500 susceptible lymphocytes even in those dying from AIDS (Duesberg, 1989a; Coombs et al., 1989; Ho et al., 1989; Schmitt et al., 1989)

f) The recent emergence of AIDS diseases in the US as a function of the enormous increase during the last 10 to 20 years in the consumption of psychoactive drugs (NNICCR, 1978-1988; Rappoport, 1988; AZT, 1989). For cocaine consumption alone has increased 3-fold during the last 10 years in the US (NNICCR, 1978-1988). Indeed, about a third of the American AIDS patients are confirmed intravenous drug users (CDC, 1990). In addition many, perhaps most, of the male homosexuals with AIDS appear to have used various psychoactive drugs (Gottlieb et al., 1981; Lauritzen and Wilson, 1986; Darrow et al., 1987; Haverkos and Dougherty, 1988; Rappoport, 1988; CDC, 1990; Adams, 1989; Friedman-Kien et al., 1990). Together, these two risk groups represent 90% of the US AIDS cases (CDC, 1990). Moreover, since 1987, 20,000 antibody-positive symptomatic and asymptomatic persons in the US (Marx, 1989) and 50,000 worldwide (Deer, 1989) have been treated with the DNA chain terminator AZT, which is directly immunosuppressive (Duesberg, 1989a). Thus, the use of drugs appears to be a common denominator of most AIDS cases in the US.

g) The preferential occurrence in the US of HIV in AIDS risk groups can also be reconciled with the risk hypothesis. Since HIV is not widespread in the US and also extremely hard to transmit due to its chronic latency (Duesberg, 1989a), only those who have intimate contacts with many others are likely to be infected, as for example promiscuous homosexuals, drug addicts sharing needles or practicing prostitution, and haemophiliacs. Thus HIV would serve as an outstanding, but not an absolute surrogate marker for AIDS risks (Darrow et al., 1987; Duesberg, 1989a; Weiss, S., 1989).

It is concluded that in the US and probably in Europe, AIDS is a collection of non-AIDS diseases of which about 90% are acquired by drug consumption associated with malnutrition and parasitic infections and other specific risks, such as chronic transfusions for treatment of haemophilia. The 10% of AIDS cases occurring outside the risk groups in the US (CDC, 1990) are consequences of the clinical definition of AIDS, namely conventional diseases occurring in persons accidentally infected by HIV. The African AIDS epidemic would also appear to be the product of the newly detectable, dormant HIV in wild animals endemic in South Africa and in New Guinea. For example, about 10-20% of the 30 million wild and domestic animals infected with HIV, since they more convincingly explain the heterosexual distribution in Africa of HIV, and would thus explain the heterosexual disease in New Guinea and in South Africa.

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