BACK

HIV Infection—the cause or effect of acquired immune deficiency?

Bradford Hill developed “nine viewpoints [that] can bring indisputable evidence for or against a cause and effect hypothesis”,[1]. They include strength of association, dose-response relationship, biological plausibility and temporality, that is, the logical necessity for cause to precede effect. Although these criteria have been the subject of some controversy over the years,[2] it is generally accepted that temporality, although not sufficient, is absolutely necessary to prove causation. According to the HIV infection hypothesis of AIDS, acquired immune deficiency (AID = low numbers of T4 cells) follows HIV infection.

However,

1. In 1985 Montagnier stated "This [clinical AID] syndrome occurs in a minority of infected persons, who generally have in common a past of antigenic stimulation and of immune depression before LAV [HIV] infection".[3] That is, Montagnier recognised that in the AIDS risk groups AID appears before HIV infection.
   More recently, Montagnier seems to be an apologist of our long standing hypothesis[4] that both HIV expression, that is, detection of the phenomena which are considered to prove HIV infection as well as AID, are the result of oxidation induced by the many oxidative agents and conditions to which the patients belonging to the AIDS risk groups are exposed. Although Montagnier has never published his view about the oxidative agents proposed by us, he accepts one of them—in Africans the cause of oxidation and thus the decrease in T4 cells is malnutrition.[5]
2. In a prospective study published in 2003, researchers of the Amsterdam Cohort study, analysed "CD4 and CD8 T cell activation marker expression in 102 individuals with known seroconversion data, before and after seroconversion. They concluded “This study demonstrated for the first time that low preseroconversion numbers of CD4 T cells and increased levels of immune activation were associated with an increased risk to develop AIDS after seroconversion...In conclusion, our data

show that chronic immune activation and the size of the CD4 T cell pool are critical factors in HIV-1 pathogenesis, even when measured before seroconversion".[6].

In the MultiCenter AIDS Cohort Study seropositive gay men were divided into two groups: Group A, who developed AIDS and group B, who did not develop AIDS five years post seroconversion. Unlike other sexually transmitted diseases, the authors found that: "receptive anal intercourse both before and after seroconversion with different partners was reported more frequently by men with AIDS...When group A was stratified by development of AIDS within 30 months and within 30-60 months and compared to matched controls, a greater proportion of high-risk sexual activities postseroconversion was noted in the group progressing to AIDS most rapidly. The differences were statistically significant 12 and 24 months post seroconversion". It was concluded "These data then suggest that greater sexual activity following establishment of HIV-1 infection leads to exposure to promoters or co-factors that augment (or determine) the rate of progression to AIDS".[7] Hence, HIV experts have published evidence showing there are non-HIV factors which act both pre and post seroconversion that induce AID and “augment (or *determine*) the rate of progression to AIDS" (emphasis ours).

3. In a study of IV drug users in New York it was shown that "The relative risk for seroconversion among subjects with one or more CD4 count <500 cells/uL compared with HIV-negative subjects with all counts >500 cells/uL was 4.53".[8]
4. A similar study in Italy showed that "low number of T4 cells was the highest risk factor for HIV infection".[9]
5. Numerous reports from many well known researchers of AIDS in haemophiliacs have shown that T4 cell depletion precedes "HIV infection".[10-13]
6. The same has been reported in recipients of “Transfusions of Blood-Derived Products”.[14]
7. One of the principal major signs of the Bangui AIDS definition is loss of body weight. However, in a study of Rwandan women, over a 24 months period it was reported that nutritional status assessed by loss of body weight "was a significant predictor of eventual HIV seroconversion…In addition to those findings for measured weight loss during follow-up, reported weight loss

before enrolment was also a risk factor for subsequent seroconversion".[15] That is, weight loss preceded HIV seroconversion by many months or even years.

8. In a recent publication involving over 22,000 patients it was shown "that the virological response after starting HAART has improved steadily since 1996....In 1995-96, 58% [of patients] achieved HIV-1 RNA of 500 copies per ml or less by 6 months compared with 83% in 2002-03....However, there was no corresponding decrease in the rates of AIDS, or death, up to 1 year of follow-up. Conversely, there was some evidence for an increase in the rate of AIDS in the most recent period".[16]
   The discrepancy between "virological exposure and the apparently worsening rates of clinical progression", means that either: (i) the clinical benefits resulting from the virological control are annulled by drug toxicity or (ii) HIV is not the cause of AIDS.

The above evidence from all the main AIDS risk groups shows that, contrary to the HIV infection theory of AIDS, HIV infection follows, does not precede AID. Thus HIV can be its effect but not its cause.

REFERENCES

1. Doll R. Sir Austin Bradford Hill and the progress of medical science. Brit Med J 1991;305:1521-1526
2. Hofler M. The Bradford Hill considerations on causality: a counterfactual perspective. Emerging Themes in Epidemiology 2005;2:11.
3. Montagnier L. Lymphadenopathy-Associated Virus: From Molecular Biology to Pathogenicity. Ann Int Med 1985;103:689-693
4. Papadopulos-Eleopulos E. Reappraisal of AIDS: Is the oxidation caused by the risk factors the primary cause? Med Hypotheses 1988;25:151-1625. Montagnier L. Apports de la recherche dans la lutte contra le Sida en Afrique. In: Pietteur M, editor. Le sida en Afrique. Belgique: Collection Resurgence; 2004. p. 224.
6. Hazenberg MD, Otto SA, van Benthem BH, et al. Persistent immune activation in HIV-1 infection is associated with progression to AIDS. AIDS 2003;17:1881-8
7. Phair J, Jacobson L, Detals R, et al. Acquired Immune Deficiency Syndrome Occuring Within 5 Years of Infection with Human Immunodeficiency Virus Type-1: The Multicenter AIDS Cohort Study. J Acquir Immun Defic Syndr 1992;5:490-496
8. Des Jarlais DC, Friedman SR, Marmor M, et al. CD4 lymphocytopenia among injecting drug users in New York City. J Acquir Immun Defic Syndr 1993;6:820-822

9. Nicolosi A, Musico M, Saracco A, et al. Incidence and risk factors of HIV infection: A prospective study of seronegative drug users from Milan and Northern Italy, 1987-1989. Epidemiology 1990;1:453-459
10. Moffat EH, Bloom AL, Mortimer PP. HTLV-III antibody status and immunological abnormalities in haemophilic patients. Lancet 1985;I:935
11. Ludlam CA, Steel CM, Cheingsong-Popov R, et al. Human T-Lymphotropic Virus Type-III (HTLV-III) Infection in Seronegative Haemophiliacs after Transfusion of Factor VIII. Lancet 1985;II:233-236
12. Jason JM, Evatt B, Chorba TL, et al. Acquired immunodeficiency syndrome (AIDS) in hemophiliacs. Scandavian Journal of Haematology 1984;33:349-356
13. Tsoukas C, Gervais F, Shuster J, et al. Association of HTLV-III Antibodies and Cellular Immune Status of Hemophiliacs. N Engl J Med 1984;311:1514-1515
14. Kessler CM, Schulof RS, Goldstein AL, et al. Abnormal T-Lymphocyte Subpopulations Associated with Transfusions of Blood-Derived Products. Lancet 1983;I:991-992
15. Moore PS, Allen S, Sowell AL, et al. Role of nutritional status and weight loss in HIV seroconversion among Rwandan women. Journal of acquired immune deficiency syndromes and human retrovirology 1993;6:611-616
16. The Antiretroviral Therapy (ART) Cohort Collaboration. Lancet 2006;368:451-458