The experimental results are presented in Fig. 1. Serial p24 levels are shown for each cell type infected with the appropriate HIV-1 strain and cultured in the presence of varying AZT concentrations. According to these data, AZT inhibits the replication of HIV-1 as effectively in monocytes as in the T-cell line. Similar results were also obtained with 2',3'-dideoxycytidine and 2',3'-dideoxyadenosine (data not shown).

These findings place into question the results of a previously published report which concluded that AZT is ineffective at inhibiting HIV-1 replication in macrophages.

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REFERENCES


No Evidence of HTLV-I Infection in Intravenous Drug Abusers in West Germany

To the Editor: Human T-cell leukemia virus (HTLV-I), the prototype human retrovirus, is closely associated with adult T-cell leukemia and tropical spastic paraparesis (1,2). HTLV-I antibodies can be detected in ~0–0.1% of American blood donors (Alan E. Williams, personal communication). However, intravenous drug abusers (IVDA) in the United States have a high prevalence of HTLV-I (and the closely related virus HTLV-II) (3,4). HTLV-I/II antibody prevalence rates were 6.6% (New Orleans) to 8.5% (New Jersey) among non-black IVDA and were significantly higher in black IVDA (49.3 and 30.2% in New Orleans and New Jersey, respectively). The higher HTLV-I antibody prevalence rates among black IVDA may reflect the background prevalence of HTLV-I in Caribbean blacks (1) and blacks in the southeastern United States (5). However, HTLV-I/II prevalence rates are far higher among IVDA than among non-IVDA from the same population, suggesting that HTLV-I may be spread as a consequence of drug paraphernalia sharing.

In Europe, detection of an infectious cluster in a white population led to a report that HTLV-I is endemic in southern Italy (6). In addition, a high prevalence of putative HTLV-I infection (4.3–27%), has been reported among IVDA in many parts of Italy (7,8), also suggesting that HTLV-I may be spreading among IVDA outside of known endemically infected areas. To analyze further the seroepidemiology of HTLV-I in Europe, we have looked for the presence of this virus among German IVDA.

The sera of 200 IVDA (ages 20–45 years, including 132 men, 65 women, three of unknown gender, all non-black) from Berlin (167) and elsewhere in West Germany (33) were investigated in 1987. Only two of the 200 sera reacted in an HTLV-I whole virus enzyme-linked immunosorbent assay (ELISA, Dupont), both of which were negative by Western blot; none of the 200 sera was reactive in a radioimmunoaassay using purified HTLV-I p24 antigen. Thus, the HTLV-I antibody prevalence was 0/200 [95% confidence interval (CI) 0–1.5%]. In contrast, 72 of the sera (36%, 95% CI 29.3–42.7%) had antibodies against HIV-1 by ELISA (Organon) with confirmatory Western blot and immunofluorescence test on K 37 cells.
In our cohort of IVDA from Germany we found no evidence of HTLV-I infection. Therefore, our results agree with Tedder and co-workers, who detected HTLV-I antibodies in only one of 113 IVDA from London (9). In a recent article, a new human retrovirus, HTLV-V, was described in Italian patients with T-cell leukemia (10). HTLV-V appears to be closely related to HTLV-I, and perhaps some of the HTLV-I reactivity in Italian IVDA was in reality due to antibodies to HTLV-V.

The epidemiology of HIV clearly differs from HTLV-I. HIV prevalence in Berlin IVDA rose from 0.2% in 1982 (11) to 36% in 1987. With the evidence that retroviral infections are transmitted very effectively in this population, we conclude that HTLV-I has probably not yet been introduced into this group. Hence, among German IVDA there is still a window of opportunity for public health intervention efforts to limit the spread of HTLV-I.

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REFERENCES