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Dear Jun,

In answer to your letter of March 9, I would like to address some of the points you made. First, there is no evidence that the situation with HTLV is similar to EBV. On the contrary, the epidemiological evidence shows a close association between disease and HTLV infection. EBV is ubiquitous. Second, I don't understand why there is a problem with one virus causing "clonal inducer T-cell malignancies" and immunosuppressive disorders. In the cat system it's been accepted for years (at least 10) that FeLV more often induces an immunosuppressive state than leukemia. The age of initial infection, route of exposure and whether there is repeat exposure are all apparent factors in the disease outcome of FeLV infection. If the T4 cells are the target of HTLV and this infection abrogates their function (as shown by M. Popovic, B. Dupont, A. Fauci and myself), then I can easily see that infection could lead to immunosuppression. Third, I'm not surprised that you have not found p19 expression on fresh cells of "AIDS" patients. It's extremely rare to find fresh cells expressing the virus. As in the bovine system, cell culture seems to be necessary to induce virus. This is probably due to removal of inhibiting factors present in the patient. The antigens p24 and p19 are almost always detected simultaneously. Finally, we know now there are many variants of HTLV-I. We believe the cause of AIDS is a more highly cytopathic variant.

Sincerely yours,

Robert C. Gallo, M.D.

AHS:tas