Correspondence

Antibodies to HTLV-I in sera from patients with connective tissue diseases

SIR, It has been suggested that HTLV-I infection may be associated with the development of some connective tissue diseases (CTD), including poly- and dermatomyositis (PM and DM), systemic lupus erythematosus (SLE), scleroderma, and Sjögrens syndrome (SS).^{1,2} This was based on eight case reports in which neither the ethnic origin of their patients nor the assays used to determine HTLV-I seropositivity were defined. Six cases of PM in HTLV-I antibody positive Jamaicans have also been reported (Rodgers-Johnson *et al*, quoted in ref 3). These observations prompted us to examine the HTLV-I antibody status of a large cohort of patients with CTD attending a rheumatology clinic in London. A total of 98 serum samples from 80 patients: 42 with SLE, of whom eight also had SS, and 38 with PM/DM, were screened using an HTLV-I enzyme linked immunosorbent assay (ELISA) kit (Dupont Ltd). Most (55) were Caucasian, 16 were of Indian, Middle, or Far East descent, and nine were West Indians. Positive samples were retested using the same assay system, and repeatedly positive sera were further tested by particle agglutination and immunofluorescence on HTLV-I infected cells. A total of 98 serum samples was tested and the results were as follows: screen positive: 5, repeat positive: 3, confirmed positive: 1. The two serum samples that were positive in the ELISA test on one occasion only were from the same patient, a child with DM; eight other serum samples from this child were negative in the ELISA. The two samples that were consistently positive in the ELISA, but not confirmed positive, were from one patient with PM and one with SLE, both of whom were Caucasian. The only confirmed HTLV-I antibody positive serum was from a West Indian woman with SLE.

Our results confirm the assertion in a recent *Lancet* editorial⁴ that results of positive HTLV-I antibody tests must be interpreted with caution and it is not acceptable to rely on one assay system alone. This is likely to be of special importance in patients with CTD, in whom the occurrence of antibodies of unusual specificity is common. We found no evidence to suggest that HTLV-I was associated with CTD in our group of patients.

Department of Virology,
John Radcliffe Hospital
Oxford

W IRVING

Virus Reference Laboratory,
Central Public Health Laboratories,
Colindale

P WHITE

Dept of Rheumatology Research,
University College and
Middlesex Hospital Medical School,
London

G CAMBRIDGE

References

- 1 Vernant J-C, Buisson G G, Sobesky G, Arfi S, Gervaise G, Roman G C. Can HTLV-I lead to immunological disease? *Lancet* 1987; ii: 404.
- 2 Vernant J-C, Buisson G, Magdeleine J, *et al*. T-lymphocyte alveolitis, tropical spastic paresis and Sjogren Syndrome. *Lancet* 1988; i: 177.
- 3 Marx J L. Leukemia virus linked to nerve disease. *Science* 1987; 236: 1059-61.
- 4 Anonymous. HTLV-I comes of age [Editorial]. *Lancet* 1988; i: 217-9.

Anticardiolipin antibodies in systemic lupus erythematosus

SIR, We read with interest the paper by Gharavi *et al* on the IgG subclass distribution of anticardiolipin antibodies in systemic lupus erythematosus (SLE).¹ We have performed a similar study and draw similar conclusions about the presence of anticardiolipin activity in all four subclasses. We have, however, examined serum samples from a wider variety of patients than Gharavi and coworkers and have found significant differences in anticardiolipin subclass distribution in patients with and without SLE.

Serum samples were obtained from 28 patients with high levels of IgG cardiolipin; 17 had a diagnosis of SLE or SLE-like illness and the remaining 11 had a variety of acute and chronic inflammatory illnesses (three rheumatoid arthritis, one chronic active hepatitis, one sarcoid, one lymphoma, one vasculitis, one emphysema, and three probable acute infections). Fourteen of the 17 patients had strong positive IgG antibodies to double stranded DNA (dsDNA) and the remaining three had had dsDNA antibodies at an earlier stage in their illness. (IgG antibodies to dsDNA were measured using an isotype specific enzyme linked immunosorbent assay (ELISA) similar to that described by Gharavi *et al* and used routinely in our laboratory.) Results were expressed in arbitrary units. Normals have a mean of 4 U/ml, whereas all our patients showed >20 U/ml. Sixteen of the 17 patients with SLE were female. In the non-SLE group no patient had double stranded DNA antibodies and only 1/11 was female.

Isotype and IgG subclass specific anticardiolipin assays were performed by an ELISA, using a method similar to that used by Gharavi *et al* with the following differences: (a) 10% newborn calf serum in phosphate buffered saline was used as the blocking agent and diluent; (b) patients' sera were diluted 1:10 for the isotype specific assay and 1:20 for the subclass assay; (c) anti-subclass monoclonal antibodies used were IgG1 (NL16), IgG2 (SH21), IgG3 (BAM08); (d) peroxidase conjugated rabbit antihuman IgG and antimouse IgG (Dako) were used for the final stage in the ELISA; (e) differences in the affinity of the monoclonal antibodies for the appropriate subclasses were