Letters to the editors

Human T-cell lymphotropic viruses in patients with multiple sclerosis

R. J. Swingle¹, P. J. Hughes¹, J. A. Munro², and D. A. S. Compston¹

¹ Section of Neurology, University of Wales College of Medicine, Heath Park, Cardiff CF4 4XN, Wales, UK

² Public Health Laboratory Service, University Hospital of Wales, Heath Park, Cardiff CF4 4XN, Wales, UK

Sirs: It has recently been claimed that a novel retrovirus may be involved in the aetiology of multiple sclerosis [5]. Serological tests have demonstrated antibodies to human T-cell lymphotropic viruses (HTLV) I, II and III (HIV) in up to 70% of patients and molecularly cloned probes have revealed the existence of HTLV-I related nucleotide sequences in cells from the cerebrospinal fluid of some affected individuals. It has been suggested that these observations result from cross-reactivity with an as yet unidentified retrovirus. There is a periodic reduction in circulating CD8 positive cells in patients with multiple sclerosis, and we have shown similar abnormalities in their unaffected relatives living in close contact but not in other related or unrelated controls [2]. Our observation of low CD8 positive cells in the cohabitants of affected individuals prompted an investigation of exposure to common infectious agents within the family unit. Serial antibody titres, measured in parallel with CD8 positive cells, did not show any differences between groups and no changes were seen in individuals related to changes in T-cell number [3]. Since the T-cell phenotypic findings would be consistent with the presence of an unusual transmissible agent selectively affecting CD8 positive cells, we have since examined sera collected from 13 patients with multiple sclerosis, 11 close contacts (9 spouses and 2 cohabitant siblings), 11 non-cohabitant siblings and 13 unrelated controls for the presence of retrovirus antibodies using commercial enzyme-linked-immunosorbent assay (ELISA) kits for HTLV I (Dupont) and HTLV III (Organon). The last available sample from each individual was tested, and these had been stored at -70°C for up to 16 months. In accordance with local ethical committee guidelines informed consent was obtained and the implication of positive tests were explained in advance. Although reactivity varied between subjects in the four groups, no individual was seropositive for either HTLV I or III (HIV) compared with results in antisera known to contain antibodies against these retroviruses.

Our failure to demonstrate serological evidence for exposure to HTLV I and III (HIV) in patients with multiple sclerosis contrasts with the findings of Koprowski et al. [5] but is in agreement with more recent results [1, 4, 7, 8]. Although they are regarded as reliable indicators of HTLV exposure in population screening, Koprowski et al. [6] suggest that commercial ELISA tests against disrupted virions are insensitive com-

pared with assays detecting antibody to the major core protein p24 of HTLV I alone because the antibodies are present in low titre, cross-reactive and only present sporadically in the course of the disease. However, the demonstration of serological abnormalities in patients with multiple sclerosis is not new and does not necessarily implicate a causal role for the viruses involved; nucleic acid hybridisation has demonstrated several agents in tissue from patients with the disease and epidemiological evidence indicates that if a transmissible agent is present in patients with multiple sclerosis it is either not particularly infectious or will not cause demyelination in isolation. It is probable that an interaction of genetic, immunological and environmental factors is involved in the pathogenesis of the disease.

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