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PREVALENCE OF ANTIBODY TO HUMAN T-LYMPHOTROPIC VIRUS TYPE-1 (HLTV-1) IN AUSTRALIAN ABORIGINES, AND DETECTION IN INDONESIAN SERA

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Summary. — The first finding of antibody to human T-lymphotropic virus type I in Australia, specifically in Australian Aborigines, is reported. The overall results suggest that this is a new area to be added to the known endemic areas for this virus. Antibody prevalence in each of two widely separated areas was found to be approximately 16% in 1977, and in one of these areas this had increased to approximately 34% in 1984/86. In this area no antibody to this virus was detected in children under 4 years of age.

Key words: *human T-lymphotropic virus type 1*; *Australian Aborigines*; *virus antibody*

Introduction

Human T-lymphotropic virus type 1 (HTLV-1) is the causative agent of adult T-cell leukaemia/lymphoma (ATL) (Tajima and Tominaga, 1985) and has been linked to the myeloneuropathy tropical spastic paraperesis. Serum antibodies to HTLV-1 are common in individuals in South-west Japan, parts of Africa and the Caribbean basin. The finding of such antibody in Europe and the U.S.A. appears to be rare (Goudsmit *et al.*, 1987; Williams *et al.*, 1988). While an absence of antibody to HTLV-1 in New Zealand has been reported (Reddy *et al.*, 1987), a survey in the East Sepik region of Papua New Guinea indictated antibody to HTLV-1 in 24% of the adult sera tested (Kazura *et al.*, 1987). Confirmation of antibody to HTLV-1 in adults in Papua New Guinea was provided by Brindle *et al.* (1988), together with the finding such antibody in sera from a number of islands in the Pacific Ocean. A maximum prevalence of 19% was reported, from Vanuatu (Brindle *et al.*, 1988). World-wide studies on this virus are continuing and have been intensified to try to identify all endemic areas (Levine *et al.*, 1988).

Extensive studies of HTLV-1 in Japan have indicted not only a wide difference in prevalence of antibody in different parts of Japan but a difference between villages on the same island (Tajima *et al.*, 1987). In this

study villages were classified as low (with <15% of adult sera positive for HTLV-1 antibody), medium (15—35% positive) or high (>35% positive). We have detected antibody to HTLV-1 in sera from Aborigines in Australia but not in sera from Europeans. We report here on the age distribution of HTLV-1 antibody in two widely separated populations of Aborigines, and also the detection of antibody to HTLV-1 in sera from Indonesia but not in sera from Niue.

Materials and Methods

For testing for HTLV-1 antibody the HTLV-1 ELISA and HTLV-1 Western blot kits made by Du Pont (U.S.A.) were used. 475 Aboriginal and 61 European sera were obtained as a result of an extensive medical audit in the Fitzroy Crossing area of the north of Western Australia in 1977. 178 Aboriginal and 60 European sera, collected from individuals in hospital for a variety of reasons in 1977 in Central Australia, approximately 1000 km to the south east of Fitzroy Crossing, were also tested. Approximately equal numbers of sera from males and females from both the Fitzroy Crossing and Central Australian areas were used. As both groups of sera were collected prior to the first reported case of AIDS in Australia (1982) they are presumed to be HIV-1 antibody negative, obviating the need for checking them for potentially cross-reacting HIV antibodies.

Parasite infestation of the individual has been shown to effect the levels of serum HTLV-1 antibody detected (Tajima and Tominaga, 1985; Kazura *et al.*, 1987), necessitating the further use of Western blot analysis for confirmation of antibody positivity. Parasite infestion is relatively widespread amongst the rural Aboriginal population in Australia. Sera were initially considered as positive if they reacted in the ELISA to a level equivalent to or greater than the positive sera supplied with the kit. They were then checked by Western blot, with the Western blot acting as a calibration standard for the ELISA.

A total of 80 Central Australian Aboriginal sera again from individuals in hospital for a variety of reasons, but collected more recently in 1984 and 1986, were also tested for HTLV-1 antibody. As well 150 sera from the Jogjakarta area of Indonesia (collected 1978/1979), and 20 sera collected in 1983 from the Pacific island of Niue were tested.

Results and Discussion

The age distribution and percentages of Aboriginal sera giving positive reactions for HTLV-1 antibody are shown in Tables 1 and 2, for sera collected in 1977 from the Fitzroy Crossing area and Central Australia respectively. HTLV-1 antibody positive sera tested in Western blots reacted typically for such sera (Fang *et al.*, 1988), and examples of a range of such reactions are shown in Figure 1. A reaction with at least both of the HTLV-1 proteins p19 and p24 was considered necessary to call a serum positive for HTLV-1 antibody.

From Tables 1 and 2 it can be seen that antibody to HTLV-1 is present in both of the widely separated Aboriginal populations. Overall prevalences were the same in both, with approximately 16% of sera from the Fitzroy Crossing area positive and also approximately 16% from Central Australia positive. However, if the relatively larger number of infants below the age of 4 in the Central Australian population sample is not taken into account, bringing this sample more into line with that from the Fitzroy Crossing area, the prevalence for the Central Australian area is increased to approximately 20%. No clear difference in the prevalence of HTLV-1 antibody

Table 1. Prevalence of antibody to HTLV-1 in Aboriginal sera collected in the North (Fitzroy Crossing area) of Western Australia in 1977

Age (years)	<4	4—10	11—20	21—30	31—40	41—50	51—60	>60
Per cent of sera positive	33	23	10	7	14	22	32	20
Number of sera positive from number tested	1/3	15/64	13/127	4/61	10/73	13/59	12/37	10/51

between males and females was apparent in either of these populations. No positive reactions for HTLV-1 antibody were found in any of the European sera from either the Fitzroy Crossing area or from Central Australia. The European sera were from individuals age- and sex-matched to the Aborigines from whom sera were obtained.

Although there were too few sera from infants and very young children, below the age of 4, available from the Fitzroy Crossing area for significant consideration, it is clear from Table 1 that a subtantial percentage of children from 4—10 year age-group had antibodies to HTLV-1. Similar prevalences were only again obvious in the 41—50, 51—60 and over 60 year age-groups. Too few sera from Central Australia compared to the Fitzroy Crossing area were available to make a meaningful comparative analysis of the age distribution of prevalences. However, it can be seen from Table 2 that in Central Australia no Aboriginal infants below the age of 4 were positive for HTLV-1 antibody, and the relatively higher number of sera tested in this age-group (35) lends more weight to this observation. It should be noted that one individual from this age-group, from the very small such sample from the Fitzroy Crossing area, was found to be positive. Studies in Japan have shown that in some areas there is a substantial increase in the prevalence of antibody to HTLV-1 in children up to the age of 3, with another subtantial increase not evident until around the age of 40 (Tajima and Tominaga, 1985; Tajima *et al.*, 1987; May, 1988).

Table 2. Prevalence of antibody to HTLV-1 in Aboriginal sera collected in Central Australia in 1977

Age (years)	<4	4—10	11—20	21—30	31—40	41—50	51—60	>60
Per cent of sera positive	0	25	0	18	19	13	24	38
Number of sera positive from number tested	0/35	3/12	0/10	5/28	6/32	3/24	5/21	6/16

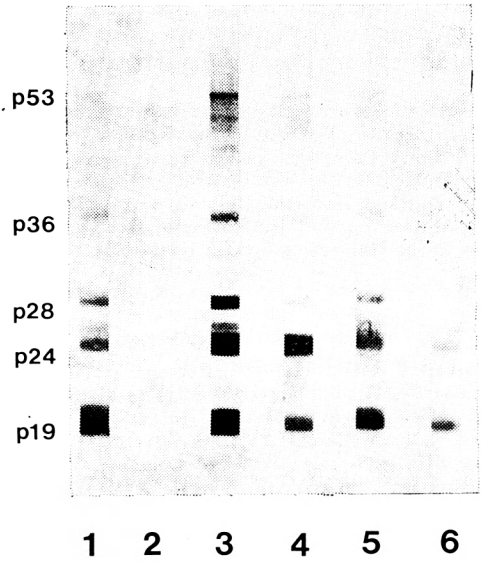

Fig. 1.
Reaction of antisera in HTLV-1 Western blots
Lane 1 — supplied HTLV-1 positive serum, lane 2 — supplied negative serum, lanes 3 to 6 — different HTLV-1 antibody-positive sera from Aborigines, reacting to different degrees.

Of the 80 Aboriginal sera collected in Central Australia in 1984 and 1986, 27 (34%) were positive for antibody to HTLV-1 after testing by ELISA and Western blot. The age range of the individuals from whom these sera were obtained was similar to that of the Aborigines from whom the sera were obtained in 1977, and none of the 1984/86 sera were from the same individuals tested with the sera from 1977. Although these sera were not tested for HIV antibody it would seem reasonable to conclude that antibody to HTLV-1 was still present more recently in Central Australian Aborigines. This is particularly so as the p19 of HTLV-1, detected on Western blots, appears to be unique to HTLV-1, and there have so far been no published reports of AIDS in Central Australian Aborigines. It is interesting to note that the prevalence of HTLV-1 antibody in the Central Australian Aboriginal population would appear to be higher in 1984/86 than in 1977, approximately twice as high if the raw figures are considered.

Of the 150 sera tested from Indonesia, 2 were positive for HTLV-1 antibody after testing by ELISA and Western blot. The ages of the individuals from whom the sera were obtained ranged from 2 months to 83 years. Antibody positivity to HTLV-1 has not previously been reported from Indonesia, although Hinuma *et al.* (1983) reported a HTLV-1 positive individual from Surinam to be Indonesian. Brindle *et al.* (1988) reported a sporadic incidence of antibody positivity to HTLV-1 from the Pacific islands they surveyed. They did not survey Niue, but we found no evidence of antibody to HTLV-1 in a small sample of 20 sera from individuals from this Pacific island.

From this and other surveys it is clear that the incidence of at least antibody to HTLV-1 is widespread throughout the world, and occurrence is not limited to isolated pockets. Efforts are now required to follow up these

surveys to try to isolate and grow the virus, and to determine more precisely the extent and consequences of HTLV-I infections in the Australiasian and Pacific regions.

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