

association pointed out that the company was in a healthy financial position, with worldwide net profits last year (US\$ 400) million and a turnover of (US\$ 3 billion). Abbott representatives said that the firm's screening test could be used immediately on equipment available at blood transfusion centres around New Zealand. A visiting Australian professor from the Hunter immunology unit in

New South Wales joined Mr Burdon in denouncing delays in introducing the blood-screening tests as irresponsible.

Yours faithfully

Parliamentary Correspondent,

Wellington.

Correspondence

Letters to the editor should be signed by all authors, typewritten in double spacing, and not exceed 600 words of text excluding references. References should be in the Vancouver style. Over-long letters may be shortened without reference to the authors unless it is specifically stated that they may not.

False positive anti-HTLV III serology

Sir, — A young man, currently aged 25 years, presented in 1980 with the nephrotic syndrome, diagnosed by renal biopsy to be due to focal glomerulosclerosis (FGS). Extreme proteinuria (> 50 g/day) led to consideration of bilateral nephrectomy, and many plasma infusions were given as short term protein supplements. Renal function eventually deteriorated and he was commenced on continuous ambulatory peritoneal dialysis (CAPD) in August 1982. In March 1983 he received a cadaveric renal transplant. Heavy proteinuria (12 g/24 h) redeveloped and a graft biopsy indicated recurrent FGS. Renal function deteriorated dramatically two months later, associated with graft pain and hypertension. Anti-rejection therapy was unsuccessful and a markedly swollen haemorrhagic kidney was removed in June 1983. Histologically there was marked oedema and haemorrhage, and occasional glomerular tufts had capsular adhesions and central and hilar sclerosis. Vessels showed endothelial proliferation and intimal thickening consistent with rejection, and an occasional thrombus was identified. The changes were considered a combination of progressive recurrent FGS and rejection.

The patient returned to CAPD but failed to thrive. In December 1984 he presented with a three month history of intermittent fever associated with night sweats, lymphadenopathy, diarrhoea and weight loss. Extensive investigation, including lymph node biopsy, failed to disclose a cause for the illness. Dialysis continued in a technically satisfactory fashion but biochemically was inadequate. There was a marked aversion by the patient to converting to home haemodialysis. Blood transfusion with 1-2 units of packed cells had been required at 1-2 month intervals for 1 year, and this fact along with the past history of many plasma infusions suggested the possibility of exposure to HTLV III virus. ELISA assay was positive for anti-HTLV III antibody but by Western blot was negative on several post-transplantation sera. All sera stored prior to transplantation were negative for anti-HTLV III and HLA-DR antibodies. A mild lymphopenia was evident in peripheral blood but a normal T4/T8 ratio was preserved on repeated testing.

A letter to the *Lancet* (Kuhnl, P; Deidl, S; Holzberger, G. 1985; 1: 1222-3) indicated that antibody to the histocompatibility antigen DR4 may cross react in the ELISA assay for anti-HTLV III antibody as the virus used in the assay had been grown in DR4 positive H9 leukaemic cells. The HTLV III virus coated beads could therefore potentially be contaminated with DR4 antigen. This alerted us to the possibility that our patient had formed anti-DR4

antibody generating a false positive result for anti-HTLV III antibody.

Sera prior to transplant did not contain anti-DR4 but post-transplant a complex immune response resulted in formation of this antibody. The donor had had the tissue type HLA-A3, 29; B5, 44; DR2, 7. and the recipient HLA-A11, 32; B5, 7; DR2, 5. DR7 antigen strongly cross reacts with DR4. Absorption of post-transplant sera with class 1 HLA antigens on platelets did not diminish anti-DR4 or HTLV III reactivity but chronic lymphatic leukaemia B lymphocytes with the tissue type HLA-A3, -; B7, -; DR4, totally absorbed anti-HTLV III and anti-DR4 reactivity. Although our patient has recovered from his febrile illness, he continues to do poorly on CAPD. As we believe this case demonstrates an example of post-transplant false positive anti-HTLV III antibody formation, we are considering retransplantation from a live donor source, using cyclosporine as immunosuppressant, in order to restore our patient to good health. Clearly, the anti-DR4 status of potential donors and recipients for organ transplantation requires evaluation before accepting the validity of positive anti-HTLV III serology.

Yours faithfully,

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Fluoridation and dental decay

Sir, — One must view with scepticism writers who continually reference themselves when writing letters to journals. Your correspondent John Colquhoun does it no less than five times in his letter on Fluoridation (*NZ Med J* 1985; 98: 659).

Because he suggested in his articles that "dental fluorosis ... has increased dramatically" and that "benefit from water fluoridation is now recognised to be very small", does not necessarily make these statements fact. It is equally possible to provide references, from highly reputable sources, and suggest that exactly the opposite is the case.

There is a substantial body of evidence to demonstrate the dental benefit of fluoridation in New Zealand and that this benefit was not evident, and neither were decay rates