Matters arising


**AUTHORS’ REPLY:** Our study of circulating T cell subtypes was designed to eliminate the known biological, technical and pharmacological factors which might distort the results. We did not find any difference between patients with polymyalgia rheumatica/giant cell arteritis (PMR/GCA) before the initiation of steroid therapy, and age and sex matched controls.1

Elling and colleagues take issue with our conclusion that the reported depletion of CD8 cells in patients with PMR/GCA remains to be proven. They also dismiss our concerns about the enumeration of lymphocyte subtypes after Ficoll-Hypaque separation of mononuclear cells.

Although the use of Ficoll-Hypaque separation in normal individuals has shown that 13–3% of lymphocytes are lost from the interface to the bottom of the tube, and that this fraction contains an increased proportion of CD8 cells.2 Direct comparison has shown that the percentage of CD8 cells measured after Ficoll-Hypaque separation may be significantly reduced compared with that after use of a whole blood technique, and this effect may be of practical importance. The reported loss of CD8 cells is expected to reduce the absolute percentage of CD8 cells in control subjects and to produce a slight increase in total lymphocyte count, from a median of 1·67 × 10^9/l in controls to 1·42 × 10^9/l in patients (a change of 15%), which is comparable to the 13% reduction in total lymphocytes described previously by Elling and colleagues when unprepared PMR patients were compared with controls.3

We do not feel that this small change is of any biological significance, but the finding explains the slight reduction in the absolute CD8 count, in the absence of any change in T cell proportions.

In summary, we remain of the opinion that the reported depletion of CD8 cells remains to be proven. Studies after Ficoll-Hypaque separation of mononuclear cells are complicated by the possibility of differential migration of T cell subtypes in control and study groups. However, if such a difference proves to be the explanation for the discrepancy in the results of studies of T cell subtypes in PMR/GCA, characterisation of the basis for this observation may be of value.

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**Silicon nephropathy and myeloperoxidase antibodies**

We read with interest the article by Sanchez-Roman and colleagues describing a high prevalence of clinical and biological autoimmune manifestations in 50 workers after occupational exposure to silica.1 In 1990 at the 3rd International Anti-Microbial and Anti-Cyttoplasmic Autoantibody (ANCA) Workshop, we first described three silicotic patients with renal involvement, in a group of 28 ANCA positive patients. By contrast, no ANCA were found in seven silicotic patients with renal involvement in a patient with lupus-like syndrome without renal abnormalities, and in another with lupus-like syndrome and focal and segmental glomerular sclerosis (FSGS). The three patients differed from those previously reported with silicon nephropathy, usually of the rapidly progressive glomerulonephritis (RPGN) type.2 All three were slate workers and had a proven pulmonary silicosis. They did not fulfill the criteria for RPGN, either clinically (two had stable chronic renal failure) or histologically (no diffuse extra-capillary proliferation). All had ANCs with anti-myeloperoxidase (MPO) specificity, which are more frequent in RPGN.3 Patients 1 (table) had focal and segmental hyalinosis with stable renal function over eight years. MPO-ANCA at the same time were already present in a stored frozen serum obtained at the beginning of the renal disease. Patient 2 had FSGS with mild renal failure. MPO-ANCA were detected when he developed end stage renal failure with fatal pulmonary haemorrhage one year later. Patient 3 had a mild proteinuria with a stable advanced chronic renal failure of unknown aetiology (no biopsy).
Authors' reply

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