AUTOANTIBODIES IN RHEUMATIC DISEASES

SIR,—Professor Monteiro and colleagues (Oct. 13, p. 797) claim that immunofluorescence is a much more sensitive method for dectecting thyroid microsomal antibodies than is the tanned-red-cell test (MCHA). This is not correct. In 1977 Abreau et al.[1] clearly showed that the MCHA test is much more sensitive than immunofluorescence. This has been confirmed by others. Doniach and Bottazzo[2] strongly recommend dropping immunofluorescence as a test for thyrolglobulin and microsomal thyroid antibodies.

Monteiro et al. report studies on five patients with rheumatoid arthirtis (RA) selected for the presence of thyroid microsomal antibodies, and they wonder why four had overt thyroid diseases and one had juvenile (insulin dependent?) diabetes mellitus. The presence of microsomal antibodies in the serum correlates well with the degree of lymphoid infiltration of the thyroid,[3] and these patients with RA will thus have been a highly selected for the presence of thyroid disorders and polyendocrine disease such as IDD type Ib.[4]

In 27 unselected cases of RA in our general outpatient clinic we found no significantly increased relative risk for RA in the presence of MCHA, compared with 528 controls without evidence for thyroid disease, autoimmune disorder, or inflammatory process (table). Our study is reported elsewhere.[5]

FREQUENCY OF MCHA

	Controls	RA	Graves' disease	Hashimoto's thyroiditis
MCHA pos.	6·3%	11·1%	62·5%	73·3%
Relative risk	1	1·9	30·3	41·3
p	"	0·2	<0·0005	<0·0005

Since Buchanan et al.[6] reported that Hashimoto's thyroiditis is associated with RA there have been many reports of thyroid autoimmune diseases in patients with RA, most of them case-reports that were highly selected. In a clinicopathological study of Hashimoto patients and matched controls, Masi et al.[7] found no significant associations with RA. So we think that although rheumatoid complaints and low titres of rheumatoid factors[8] are common in Graves' thyrotoxicosis and Hashimoto's thyroiditis, RA, as defined by W.H.O. criteria, is not increased in unselected patients with thyroid diseases.

Dr Blake and colleagues (Aug. 4, p. 224) detected thyroid autoantibodies, mainly thyroglobulin antibodies, in synovial fluid from 50 patients with various arthritides. With sensitive methods low concentrations of thyroglobulin are found in the general circulation of about 60% of normal individuals.[9,10] Thus thyroglobulin escaping from the thyroid acini can provide a source of antigen to induce autoantibody formation. Normal individuals have about 0·1% of thyroglobulin responsive lymphocytes in the blood.[11] In experimental thyroiditis and other diseases Freund's adjuvant functions as a polyclonal activator of T lymphocytes.[12] By electronmicroscopy an increased number of lysosomes was observed in the early stages of cell damage in the thyroid gland.[13] Besides Freund's adjuvant, bacteria and bacterial products also have adjuvant effects and can lead to autoimmunity.[14]

We suggest that lysosomal activity, increased in the synovial fluid in all types of arthritis, can activate T helper cells and B lymphocytes reactive against self-components, possibly by destroying suppressive factors produced by T suppressor lymphocytes. The lack of thyroid antibodies in the sera of most of these people indicates that this activation is a local problem, and has nothing to do with the general breakdown of natural tolerance in autoimmune thyroid diseases.

Medizinische Universitäts-Poliklinik,
74 Tübingen, West Germany W. SCHERBAUM

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