seen, but for the majority of patients the slight reduction in longevity is in no way equivalent to that in the lymphomas. While we may anguish over the ravaging and debilitating aspects of this disease, I do not think this justifies, at the present time, trials with these drugs which have known severe short and long term side effects, especially when we have not even illustrated that we can carry out adequate trials of our current second line agents nor agree on acceptable measures of disease outcome.

One further point is pertinent. If sums of money similar to those poured into cancer research had been made available for research into rheumatic diseases, always unfashionable with both public and media, then I am sure we would be in a better position to answer many of the above questions.

Department of Rheumatology,
Selly Oak Hospital,
Birmingham B29 6JD.

G R STRUTHERS

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Sulphasalazine therapy in RA

SIR, Pullar *et al* reported recently the effect of acetylator phenotype on the efficacy of sulphasalazine therapy in rheumatoid arthritis (RA).¹ We would largely agree with their conclusion that there is little practical value in determining acetylator phenotype before therapy² but would like to comment on the incidence of slow and fast acetylator phenotypes in RA and the suggestion that fast acetylators may be subject to more severe disease.

During the course of investigations on the use of sulphasalazine in RA we have phenotyped 108 patients using a modification of Schroder's method.³ Thirty three patients (30.6%) were male and 80 (74.1%) were seropositive. Sixty two (57.4%) of our patients were slow acetylators, a figure which is in agreement with the reported incidence in the general UK population⁴ and other studies of RA patients.^{5, 6}

It is always dangerous to decide what is serious or mild disease at a single point in the evolution of a chronic relapsing disorder, but all of our patients were felt to have disease of sufficient severity to warrant second line therapy, and the mean erythrocyte sedimentation rates (mm/1st h) at the start of treatment with sulphasalazine were 50.2 (SD 29.4) in the slow acetylators and 51.7 (SD 24.5) in the fast. There is no statistical difference between these figures.

We would conclude therefore that the distribution of acetylator phenotype in patients with RA is the same as that in the general population and that acetylator phenotype has no bearing on the severity of RA.

Rheumatism Research Unit,
Nether Edge Hospital,
Sheffield S11 9EL

R S AMOS
D E BAX
M S GREAVES

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Retroviruses in rheumatoid arthritis

SIR, Rheumatoid arthritis (RA) is characterised by both in vivo and in vitro depression of cell mediated immunity. The mechanism for this depressed cellular immunity is not known but may be due to deficient interleukin-2 production.¹ Since retroviruses have been associated with deficiencies of immune reactivity we used standard techniques to look for the presence of antibodies to the human T cell retroviruses (HTLV-I, II, and III) in the sera from 60 patients with classical or definite rheumatoid arthritis.^{2, 3} The results were uniformly negative. Since RA is linked to HLA-DR4 these findings indicate that false positivity on this account might be a rare event.⁴ It may be concluded from this work that if retroviruses are involved in the pathogenesis of rheumatoid arthritis then they are not serologically related to the known retroviruses as detected by presently available techniques.

Rheumatology Unit,

G S PANAYI

Dept of Medicine,
Guy's Hospital Medical School,
London SE1 9RT.

The Institute of Cancer Research,
Chester Beatty Laboratories,
Fulham Road,
London, SW3 6JB.

A G DALGLEISH

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