9 Richmond J, Roy L M H, Gardiner D L, Alexander W R M, Duthie J J R. Effects of the intravenous administration of saccharated oxide of iron. *Ann Rheum Dis* 1958; **17:** 406–15.
10 Mowat A G, Hothersall T E. Iron content of synovial tissue in patients with rheumatoid arthritis and in normal individuals. *Ann Rheum Dis* 1968; **27:** 345–51.
11 Richmond J, Alexander W R M, Potter J L, Duthie J J R. Red cell survival measured by radio-active chromium. *Ann Rheum Dis* 1961; **20:** 133–7.

SIR, Professor Duthie misunderstands the purpose of our study, which was to examine the *mechanism* of the iron promoted synovial flare and *not* to evaluate the efficacy of this form of treatment of rheumatoid anaemia. As our article and Professor Duthie's letter point out many authors have described the synovial flare. We suggest that the reaction is mediated by iron (rather than dextran) promoted lipid peroxidation.

I have no criticism of Professor Duthie's haematological observations and failed to quote them only because our article did not address this issue. However, I would take the opportunity of describing the results of a study that was omitted from professor Duthie's extensive list of references. Bentley and Williams[1] gave iron dextran (800 mg intramuscularly) to 30 anaemic rheumatoid patients. Twenty six of the 30 patients showed a significant rise in haemoglobin at the two-month mark, but values fell back to the pretreatment level at nine months. The rise in haemoglobin was not related to pretreatment iron levels. They concluded and I quote: 'a therapeutic trial of parenteral iron will produce haematological improvement in the majority of anaemic patients and cannot therefore be taken as an indication that the anaemia was primarily due to iron deficiency. In the majority of anaemic rheumatoid patients, however, therapy should be directed at suppression of the activity of their rheumatoid disease, as this is the only current means by which lasting haematological improvement would be achieved'. Though we did not report the haematological indices in our patients, we found much the same . . . a temporary rise only. I would not choose to describe a treatment that causes the disease to flare, has no significant long-term effect on the haemoglobin, but occasionally causes anaphylaxis as 'very valuable'.

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Reference

1 Bentley D P, Williams P. Parenteral iron therapy in the anaemia of rheumatoid arthritis. *Rheumatol Rehabil* 1982; **21:** 88–92.

Systemic lupus erythematosus

SIR, The report of Soppi, Eskola, and Lehtonen[1] on identical twins discordant over 20 years for clinically evident SLE adds to our understanding of the importance of both genetic and environmental factor(s) in the expression of this disease. To date it remains unknown whether the genetic factor(s) is requisite and the environmental factor only stimulatory or whether a certain combined quantity (more or less of one and/or the other) of the two sufficient to reach an expressive threshold is critical.

In the twins described it is of interest that the clinically unaffected sibling had a persistently raised erythrocyte sedimentation rate (ESR), for although the presence of antinuclear antibodies and other protein aberrations. including low complement levels, need not be associated with active disease, a raised ESR, though non-specific, usually suggests the presence of active inflammation or tissue damage. Could something subtle have been missed? Twenty years' observation argues against this notion, but it is not necessarily reassuring for the future. I look forward to a follow-up report 10 years from now.

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275 Union Street,
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Reference

1 Soppi E, Eskola J, Lehtonen A. Evidence against HLA and immunological dependence of disease outbreak in SLE. Immunological characterisation of identical twins clinically discordant for SLE. *Ann Rheum Dis* 1985; **44:** 45–9.

Detection of anti-dsDNA as a diagnostic tool

SIR, We read with interest the paper by Drs Swaak and Smeenk[1] on the detection of antibodies to double-stranded deoxyribonucleic acid (anti-dsDNA) as a diagnostic tool. Of 441 patients without systemic lupus erythematosus (SLE) but with anti-dsDNA no fewer than 304 (69%) developed SLE within one year as judged by the preliminary classification criteria of the American Rheumatism Association (ARA).[2] Longer follow up showed a cumulative incidence of SLE of 85%. We have found different results in a group of patients who, in contradistinction to the Dutch study cases, did not have antinuclear antibodies (ANA) on conventional indirect immunofluorescent testing, despite high titres of anti-dsDNA.

By reference to the anti-dsDNA results file in the pathology service laboratory covering the period 1976–83, we have identified 20 patients (12 female, eight male; mean age 51 years) who were followed up for at least a year and who had anti-dsDNA titres in excess of 30 U/ml (anti-DNA kit, Amersham International) on two or more occasions. Tests for ANA with serum diluted 1:16 on a substrate of rat liver slices were negative on every occasion for every patient included in the study. Two patients subsequently developed ANA at 20 and 25 months respectively after the anti-dsDNA were first noted. The other 18 patients remained consistently ANA negative and were tested on three to 12 occasions (mean 5·1).