

Food intolerance in rheumatoid arthritis

Sir: We read with interest the two papers by Drs van de Laar and van der Korst on food intolerance in rheumatoid arthritis (RA).^{1,2} It appears from this extensive but short study of patients with seropositive RA that rheumatoid disease in a small subgroup is sensitive to dietary manipulation. Identification of these patients is, however, a problem.

We have previously shown that significant gut abnormalities are associated with raised levels of IgA rheumatoid factor (RF) and dietary protein specific IgG in patients with RA and suggested that these changes might identify patients who would respond to dietary manipulation.³

Gendre and colleagues have described villous atrophy and other histological abnormalities of the small intestine in patients with RA.⁴ These changes were more significant among IgM RF negative patients (who are often IgA RF positive).

In the recent study the number of patients examined histologically was small, therefore a firm conclusion cannot be reached. Moreover, it is likely that by excluding IgM RF negative patients with RA some food intolerant patients might have been missed. Furthermore, investigation of intestinal structure and function in RA is severely hampered by the damaging effects of non-steroidal anti-inflammatory drugs, which are so often used by patients with RA. Thus a fundamental question remains extremely difficult to answer—that is, whether gut abnormalities and any of the manifestations associated with these abnormalities in patients with RA are secondary to the disease process and its treatment or are of primary pathogenic significance.

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- 1 van de Laar M A F J, van der Korst J K. Food intolerance in rheumatoid arthritis. I. A double blind, controlled trial of the clinical effects of elimination of milk allergens and azo dyes. *Ann Rheum Dis* 1992; 51: 298–302.
- 2 van de Laar M A F J, Aalbers M, Bruins F G, van Dinther-Janssen A C H M, van der Korst J K, Meijer C J L M. Food intolerance in rheumatoid arthritis. II. Clinical and histological aspects. *Ann Rheum Dis* 1992; 51: 303–6.
- 3 O'Farrelly C, Marten D, Melcher D, et al. Association between villous atrophy in rheu-

matoid arthritis, rheumatoid factor and gliadin-specific IgG. *Lancet* 1988; ii: 819–23.

- 4 Gendre J P, Luboinski J, Prier A, Camus J P, le Quintrec Y. Anomalies de la muqueuse jejunaie et polyarthrite rhumatoide: 30 cas. *Gastroenterol Clin Biol* 1982; 6: 772–5.

AUTHORS' REPLY We appreciate the interest of Drs Abuzakouk and O'Farrelly in our papers on food intolerance in rheumatoid arthritis (RA).^{1,2} In the first paper the existence of intolerance for food in a minority of patients was suggested and in the second paper the patients with food intolerance were described in more detail. We hoped that the clinical, immunological, and histological investigations in these patients might provide some clues to the problem of how to identify food intolerant patients. However, possibly because of the limited number of patients, such predicting parameters were not found. As suggested by Drs Abuzakouk and O'Farrelly IgA rheumatoid factor (RF) might by such a parameter—this possibility was not considered in our paper. We studied several dietary protein specific IgGs, however, but a discriminatory high level of these antibodies was not found.³

Histological abnormalities of the small intestine are of particular interest in connection with food intolerance in RA.^{4,6} However, as this needs an invasive technique the availability of material for study is limited. Moreover, several other factors, such as the effect of non-steroidal anti-inflammatory drugs, do influence intestinal histology.

The existence of food intolerance in RF negative RA might indeed exceed that in positive disease. In these studies, however, we preferred to study the more homogeneous type of RA—namely, RF positive cases.

We should mention that we are at present studying the identification of food intolerant patients with RA. In this study many of the suggestions made by Drs Abuzakouk and O'Farrelly are included.

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- 1 van de Laar M A F J, van der Korst J K. Food intolerance in rheumatoid arthritis. I. A double blind, controlled trial of the clinical effects of elimination of milk allergens and azo dyes. *Ann Rheum Dis* 1992; 51: 298–302.
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- 3 van de Laar M A F J, Aalbers M, van der Korst J K. Radioallergosorbent tests to foods in rheumatoid arthritis. In: van de Laar M A F J, ed. *Rheumatoid arthritis and food allergy?* Amsterdam: Thesis Publishers, 1991: 81–91.
- 4 Marcolongo R, Bageli P F, Montagnani M. Gastrointestinal involvement in rheumatoid arthritis: a biopsy study. *J Rheumatol* 1979; 6: 426–40.
- 5 Doube A, Collins A J. Is the gut intrinsically abnormal in rheumatoid arthritis. *Ann Rheum Dis* 1988; 47: 617–9.
- 6 van de Laar M A F J, van der Korst J K. Rheumatoid arthritis, food and allergy. *Semin Arthritis Rheum* 1991; 21: 12–23.

Correction

Ankylosing spondylitis and HLA-B27: restriction fragment length polymorphism and sequencing of an HLA-B27 allele from a patient with ankylosing spondylitis

In the paper by C M Higgins, T Lund, M E Shipley, et al (*Ann Rheum Dis* 1992; 51: 855–62) we regret that fig 1 was incorrectly reproduced. The correct version of this figure is shown below.

