4) How are your articular symptoms? (joint swelling, spine stiffness, tendinitis).
5) Did you modify your NSAID regime? (more, unchanged, reduced/discontinued).
6) Do you consider that the effect of the diet justifies continuing it?

When at least 4/6 variables were improved or positive, and none deteriorated the therapeutic effect was considered as good, and when 2/6 were improved and none deteriorated, as moderate. The compliance to the diet (poor, questionable or good) was assessed from the questions to the patient.

In the SA group results at six weeks (table) showed a relatively good compliance to the diet (18/25). When patients were questioned about its benefit, 13/25 reported good efficacy (no precise symptom was more sensitive) and 4/24 a moderate improvement; among the good responders, 8/13 could discontinue their NSAID therapy. Conversely, despite good compliance, no patients with RA improved at six weeks or could reduce NSAIDs and they all decided to discontinue the trial.

When follow up of the 17 SA responders was carried out, 12/15 were still satisfied and kept up the diet at three months, 10/10 at six months and 8/9 at nine months. Our longest follow up is now over two years; six patients are still observing the diet and remain free from any other therapy; interestingly, none reported discomfort or frustration with the diet even after the longest duration.

No association between response and variables such as sex, age, axial versus peripheral involvement, enthesopathies, sacroiliitis, HLA-B27, intestinal, genito-urinary or cutaneous symptoms, duration of disease could be demonstrated. According to some patients, psoriatic lesions remained unchanged but the number of such patients was limited (n = 5).

In summary, this study indicated that more than half of the patients with SA felt a subjective improvement of their symptoms with a diet without milk products; they felt better, pain severity decreased, morning stiffness improved, joint and spine symptoms got better, NSAID consumption was reduced and a large number of patients agreed to continue the diet for a longer period. So far, possibly because of its heterogeneity, the subgroup of responders could not be further characterised.

Benefit to the patients appeared within six weeks and most of the responders decided to keep up with the diet for months or years. Interestingly, three patients reported a transitory relapse of their complaints within a few days when there was a relapse in the diet and one patient on the diet was free of recurrent episodes of uveitis for over two years. Yet before interpreting these data, one should be aware of the numerous biases: small, heterogenous and not randomised groups, subjective evaluation of efficacy, diet without excluding all milk products, no control diet, subjective measures of outcome and compliance, and in addition the course of SA which can be spontaneously favourable.

A placebo effect is present in this study; yet a high proportion of SA responders – in contrast to RA patients – continued the trial for a relatively long period of time and in the six month follow up analysis, NSAID and salazopyrine/methotrexate could be discontinued in respectively 8/10 and 3/3 patients.

The reasons for possible benefit from the diet remain unclear. Digestion is capable of producing a hypersensitivity reaction and certain associations between connective tissue diseases and food (ingredients) have been proposed.[5] Approximately one third of SA patients "believe" that certain foods can increase morning stiffness, pain and swelling.[6] A diet free of dairy products could modify the content of the intestinal flora and consequently reduce the proliferation of pathogenic bacteria. Some gram negative bacterial fragments could persist despite the ultra high temperature processing of milk.

Alternately, a chronic intestinal allergy to milk products could contribute to the gut permeability alterations in SA.

Effect of the six week diet

	SA	*RA*	*total*	*p value*
Compliance (1)				
Bad + questionable	3 + 4	1 + 0	8	
Good	18	9	27	
Total	25	10	35	0·391
Efficacy (2)				
None	7	10	17	
Moderate + good	4 + 13	0 + 0	17	
Total	24	10	17	<0·001
Not evaluable	1	0		

1) Two tailed Fisher's exact test comparing good responders to pooled bad and questionable ones: no difference in compliance could be shown to the diet between both groups of patients.
2) Chi-square test comparing 'no efficacy' to pooled 'moderate and good efficacy'. A significant difference in terms of efficacy of the six week diet could be made between the SA and the RA group ($p < 0.001$).

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MATTERS ARISING

The relationship between rheumatoid arthritis and bronchiectasis

We enjoyed the recent article by McMahon *et al* which attempted to rationalise the relationship between rheumatoid arthritis (RA) and bronchiectasis.[1] There is now good evidence that bronchiectasis is associated with RA and this paper hints that airways obstruction in patients with bronchiectasis and RA may be more pronounced than in those with bronchiectasis alone. The reason for this remains unclear but the suggestion that it may be mediated by the presence of secondary Sjögrens syndrome was novel. However, we would like to provide two items of evidence which appear to make this theory less tenable.

In neither physiological[2] nor pathological[3] studies of the airways of patients with primary Sjögrens syndrome (PSS) have we found evidence of bronchiectasis, although high resolution computed tomography (HRCT) demonstrated peribronchiolar infiltrates in 30% of PSS patients complaining of dyspnoea.[3] Of more direct relevance we have recently completed a HRCT study of 40 patients with RA, 10 of whom had evidence of bronchiectasis.[4] Schirmer's tear tests were measured in all patients and abnormal results were found in 60%. Using the same criteria as McMahon *et al*, we found 18 of 30 patients with RA without bronchiectasis and 6 of 10 RA patients with bronchiectasis to have 5 mm or less wetting from either eye over five minutes.

Thus we have been unable either to demonstrate the presence of bronchiectasis in patients with PSS, or to show a relationship between Schirmer's tear test results and the presence of bronchiectasis in RA patients. The difference between our results and those of McMahon *et al* may be explained by the relatively low percentage of RA patients without bronchiectasis (controls) with an abnormal Schirmer's tear test in their study (22%). This is lower than that found in their patients with pure bronchiectasis and may represent an underestimate of the true value. We conclude that the case for Sjögrens syndrome contributing to the development of bronchiectasis in patients with RA remains at best unproven.

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AUTHOR'S REPLY: We thank Drs Kelly and Gardiner for their interest in our paper[1] and for their informed comments on our hypothesis that elements of Sjögrens syndrome may account for the link between RA and bronchiectasis.

We acknowledge the limitations of inferring a diagnosis of Sjögrens syndrome from a Schirmer's test and that the small numbers of patients involved make confident

claims unwise. However, the significance of our findings is heightened by the close matching of our rheumatoid alone (RA) and rheumatoid and bronchiectasis (RABR) groups and we suspect that Kelly and Gardiner's groups were not so matched. Clearly the point concerning the relative frequency of Sjögrens syndrome in RA and RABR will not be settled until it has been examined with more rigour and sufficient numbers of subjects. Secondly, there are clinical, genetic and serological differences[2 3] which make primary and secondary Sjögrens distinct entities, and if bronchiectasis is associated with secondary Sjögrens syndrome, it does not necessarily follow that a similar relationship will be found with primary Sjögrens syndrome.

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