PostScript

MATTERS ARISING

Long term treatment of psoriatic arthritis with infliximab

We read with great interest the article by Feletar *et al*.[1] We were surprised by their findings of only a modest response to infliximab treatment in patients with treatment refractory psoriatic arthritis (PsA), as this seems to be in contrast with our observations.

Since 2001 nine patients with refractory PsA according to American College of Rheumatology criteria have been treated with infliximab at our division. All patients had active joint disease with a tender joint count (TJC) and a swollen joint count (SJC) of at least 6, with the exception of one patient (patient 3), whose cervical spine was affected. Quantitative assessment of skin involvement was not available. Table 1 shows further demographic data.

Seven patients received infliximab for more than 52 weeks, and five of those for more than 78 weeks. Clinical assessment, including TJC and SJC, performed by experienced rheumatologists, and laboratory tests (haematology, biochemistry, antinuclear antibodies (ANA), including subsets) was made routinely at each visit (fig 1). During the treatment period toxicity occurred on three occasions, including one labial herpes infection (patient 4), one leucopenia of 2.6×10^9/l (patient 3), which led to the discontinuation of methotrexate, and one allergic reaction (patient 9). Patient 9 had to stop infliximab treatment permanently despite an excellent clinical response, whereas the other two patients received infliximab again after the adverse events resolved. Six of the nine patients became positive for ANA—titre >1/80 on at least two successive visits. No serum conversion of ANA subsets occurred and no lupus-like symptoms were seen.

Eight of the nine patients showed rapid improvement and a clinical response. Only 1 patient (patient 8) was unresponsive to the treatment regimen, which was stopped. At 52 weeks five patients met the PsA response criteria,[2] four patients were in complete remission (patients 1, 3, 5, 6) of joint tenderness and swelling, one patient (patient

Table 1 Demographic features of nine patients with PsA at baseline

Characteristic	Number or mean (SD)
Sex (M/F)	4/5
Age (years)	41 (13)
Disease duration (years)	10.5 (8.0)
DMARDs per patient (n)	3.6 (1.3)
Current DMARD	Methotrexate
Mean dosage (mg/week)	17.5
Tender joint count*	16.4 (8.6)
Swollen joint count†	10.5 (3.8)
HLA-B27 positivity	3

*Total number of joints counted 68; †total number of joints counted 66.

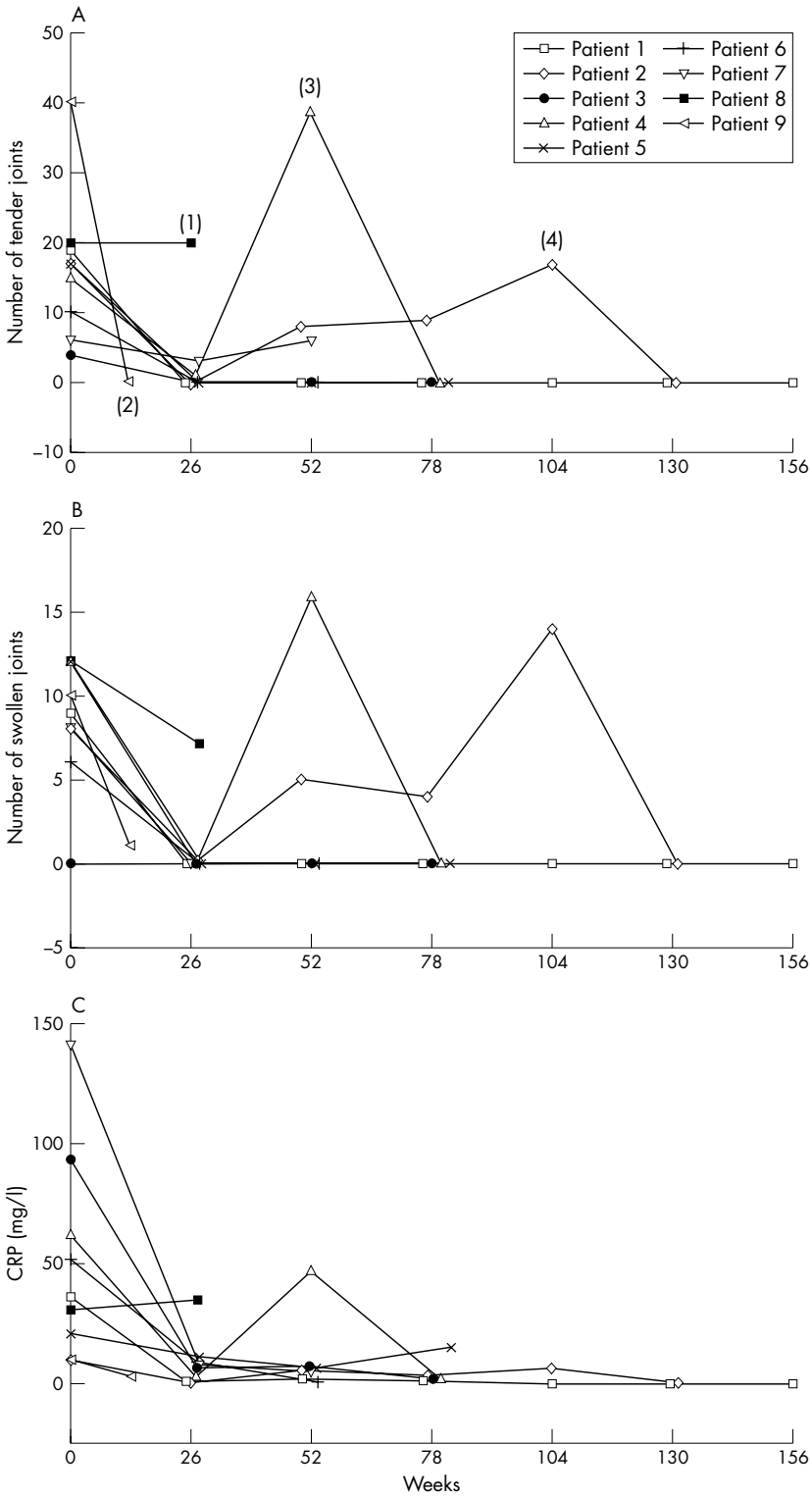

Figure 1 Changes in (A) TJC, (B) SJC, and (C) C reactive protein levels (normal value <9 mg/l) of each patient during the respective treatment period. Infliximab treatment was stopped because of insufficient response (1) or allergic reaction (2). Disease exacerbation occurred when infliximab was stopped owing to herpes labialis infection (3). Shortening of the infliximab application interval to 4 weeks (4).

2) had an incomplete response. Patient 4 stopped infliximab treatment in week 49 for 10 weeks because of a labial herpes infection that was treated with acyclovir. Thereafter tumour necrosis factor blocking therapy was successfully restarted.

At 78 weeks 4/5 patients (patients 1, 3, 4, 5) were in complete remission. One patient (patient 2) who had an incomplete response with a 6 weeks' treatment interval showed complete remission after the interval was shortened to 4 weeks. Patient 1 who received his first infliximab dose at our division 160 weeks ago had been responding so well to the treatment that we stopped giving infliximab after his 16th infusion at the end of week 120. Because of disease exacerbation, treatment had to be restarted 26 weeks later and within a few days (data not shown) the patient regained complete remission.

These data, although only retrospectively obtained, suggest that infliximab is a valuable therapeutic tool for treating refractory PsA. So far, permanent discontinuation of treatment owing to toxicity or inefficiency has only been necessary for one of the nine patients.

One possible reason for the different outcome in the effect of infliximab on PsA in our patients and those of Feletar *et al* might be the severity of the joint disease in Feletar's patients. Fifteen out of 16 of their patients had mutilating arthritis, whereas this form of joint destruction was not present in our group. To clarify this possibility, further studies with larger groups and well defined joint disease are necessary.

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References

1 **Feletar M**, Brockbank JE, Schentag CT, Lapp V, Gladman DD. Treatment of refractory psoriatic arthritis with infliximab: a 12 month observational study of 16 patients. *Ann Rheum Dis* 2004;**63**:156–61.
2 **Clegg DO**, Reda DJ, Abdellatif M. Comparison of sulfasalazine and placebo for the treatment of axial and peripheral articular manifestations of the seronegative spondylarthropathies: a Department of Veterans Affairs cooperative study. *Arthritis Rheum* 1999;**42**:2325–9.

Author's reply

Yazdani-Biuki *et al* were surprised that our data on 16 patients with arthritis treated with infliximab demonstrated only a modest response.[1] They contrast our data with data from nine patients with psoriatic arthritis (PsA) treated with infliximab in their centre. Although they mention nine patients, only seven were treated for more than 52 weeks. They claim that eight of the nine patients demonstrated rapid clinical response, but provide information on only five patients who demonstrated a PsA American College of Rheumatology response, four of whom are said to be in complete remission. It is not clear whether the other patients discontinued the drug, in which case their withdrawal rate would be 22%.

This is in contrast with the results of our study, which demonstrated a high toxicity rate, and high level of discontinuation (6/16 or 38%). We had previously suggested that the difference between our study and others might reflect the severity of our patient group. The group treated by Yazdani-Biuki *et al* was demographically somewhat different from our group. There were more men in our study (12/16 *v* 4/5), they were older (48 *v* 41), and had longer disease duration (14 *v* 10.5). However, the same average number of disease modifying antirheumatic drugs had been used in the two groups. Yazdani-Biuki *et al* did not provide information on damage in their patients. Our group included five patients with arthritis mutilans; however, all these patients had evidence of inflammatory arthritis. The other five patients may have had evidence of damage but did not demonstrate arthritis mutilans. Because we have previously shown that the presence of damage predisposes not only to progression of damage but also to early mortality in this patient group, we felt it was important to give these patients the best available treatment.[2 3]

We agree with Yazdani-Biuki *et al* that the difference between the results of our two studies may be related to the difference in disease severity between the two groups. However, the number of patients is too small to draw valid conclusions. It is important to gain more experience with anti-tumour necrosis factor agents both in randomised clinical trials and clinical observational studies, including a wide range of patients with PsA, so that the true level of response and toxicity can be identified.

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References

1 **Feletar M**, Brockbank JB, Schentag C, Lapp V, Gladman DD. Treatment of refractory psoriatic arthritis with infliximab: a 12 month observational study of 16 patients. *Ann Rheum Dis* 2004;**63**:156–61.
2 **Gladman DD**, Farewell VT, Husted J, Wong K. Mortality studies in psoriatic arthritis. Results from a single centre. II. Prognostic indicators for mortality. *Arthritis Rheum* 1998;**41**:1103–10.
3 **Gladman DD**, Farewell VT. Progression in psoriatic arthritis: role of time varying clinical indicators. *J Rheumatol* 1999;**26**:2409–13.

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Immunohistochemistry of normal synovium

A recent paper published in the *Annals of the Rheumatic Diseases* by Singh *et al* claimed to be the first report of immunohistochemical features of knee synovium in such a large number of healthy normal subjects.[1] This statement is incorrect and I am surprised that the authors of this paper appear to be unaware of our paper published more than 12 months previously in the same journal.[2]

We studied 20 normal patients attending a sports medicine clinic with unexplained knee pain who had no evidence of any form of arthritis on history, examination, and laboratory tests and had normal *x* ray findings and normal knee arthroscopy. Although Singh *et al* may argue about the source of our ''normal'' subjects, it is noted that several of their subjects were not entirely normal either. We are, at least, in a better position to state that our patients were as close as possible to normal than the study of Singh *et al*, who relied on normal *x* ray examinations to exclude joint pathology (including early osteoarthritis) and included patients with either a positive rheumatoid factor or a raised erythrocyte sedimentation rate (ESR), neither of which was included in our patient group.

In the two studies the patient group and the variables measured in the synovial membrane were similar, and the conclusions of both studies were also similar. Like the study of Singh *et al*, we found quite a variability in the architecture of the normal synovial membrane, particularly in relation to the thickness of the lining layer and the subintimal cell infiltrate, but we also measured cytokine production, cell adhesion molecule expression, and mediators of osteoclast formation, which were not included in the study of Singh *et al*. We did find evidence of B cells and occasional plasma cells in the normal synovial membranes, unlike Singh *et al*, but these cell populations were quite sparse.

It is, however, very surprising that neither the journal reviewers nor the authors of this paper appeared to be aware of a similar paper published in the same journal before this paper was accepted for publication, and we do dispute the statement of Singh *et al* that they have published the ''first report of immunohistochemical features of knee synovium in such a large number of healthy normal subjects''.

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References

1 **Singh JA**, Arayssi T, Duray P, Schumacher HR. Immunohistochemistry of normal human knee synovium: a quantitative study. *Ann Rheum Dis* 2004;**63**:785–90.
2 **Smith MD**, Barg E, Weedon H, Papangelis V, Smeets T, Tak PP, Kraan M, *et al*. Microarchitecture and protective mechanisms in synovial tissue from clinically and arthroscopically normal knee joints. *Ann Rheum Dis* 2003;**62**:303–7.

Authors' reply

We thank Dr Smith for alerting readers and us to the existence of his excellent paper that came out after our study was done and during the preparation of our manuscript.

Our study described asymptomatic subjects,[1] a somewhat different group from that in Dr Smith's study[2]—namely, patients with unexplained knee pain with clinically and arthroscopically normal knee joints. It is fascinating that in his study arthroscopy could not detect moderate inflammation seen in some biopsies. Thus, some synovitis can be present even when examinations and gross arthroscopy are normal. Future studies of patients like ours and those of Smith *et al* would benefit from longer follow up. What was the final cause of the knee pain that led to the need for arthroscopy? Will the cellular pattern be consistent, resolving, or progressive in some patients? Do the infiltrates seen in some "normal subjects" have any prognostic significance?

It appears that our two studies[1 2] with many similar findings reinforce the point, also noted before by Lindblad and Hedfors,[3] that there may be more variability in synovial histology and immunohistochemistry than many may have appreciated.

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References

1 **Singh JA**, Arayssi T, Duray P, Schumacher HR. Immunohistochemistry of normal human knee synovium: a quantitative study. *Ann Rheum Dis* 2004;**63**:785–90.
2 **Smith MD**, Barg E, Weedon H, Papengelis V, Smeets T, Tak PP, *et al*. Microarchitecture and protective mechanisms in synovial tissue from clinically and arthroscopically normal knee joints. *Ann Rheum Dis* 2003;**62**:303–7.
3 **Lindblad S**, Hedfors E. The synovial membrane of healthy individuals—immunohistochemical overlap with synovitis. *Clin Exp Immunol* 1987;**69**:41–7.

Low dose prednisolone for treatment of RA

We read with interest the report of the WOSERACT trial[1] that compared the addition of 7 mg daily prednisolone or placebo to sulfasalazine in early rheumatoid arthritis (RA). A number of important aspects of the trial have been dealt with well: the sample size is adequate; appropriate attention has been paid to confounders; two separate and independent readers scored the radiographs; the 2 year trial was of adequate length; and the completeness of the data is satisfactory. Given these strengths, it is all the more disappointing that the results of the main outcome, radiographic damage, cannot be adequately interpreted as they are reported. Indeed, the validity of these results is open to serious doubt. We feel there is a real possibility of a type II statistical error (missing a true difference between treatment arms). There are two (possibly three) reasons for this.

Firstly, there is an absolute difference between the *x* ray scores of the two readers of about 40 Sharp points. This raises strong doubts over the proficiency of either or both readers. In early RA Sharp scores are typically very low, with most patients scoring 0 and only a few with higher scores. Scores of 80, let alone 159, after one year of RA are without precedent in the literature, and even the baseline medians of 6 and 8 recorded for the conservative reader are quite high. In contrast with the authors, we cannot be "reassured" by their assertion that "the change in *x* ray score was consistent between the two readers": these data are simply not provided in the report. All we have is an unsatisfactory correlation of absolute scores between readers of 0.8 (whereas in most trials the intraclass correlation coefficient (the recommended and more severe test of reliability between readers) exceeds 0.9), and the comparison between readers of differences between the median start and end scores in the two study groups. Unfortunately, the difference between medians at baseline and end point is not the same as the median change.

Secondly, most trials choose two readers who read either with sequence known or unknown (the jury is still out on which is the preferred method), and report the mean of these two readings. This report has two readers, each of whom uses a different one of these options, and this makes it impossible to pool the results. Also, even with sequence unknown, films should be read as sets (all films belonging to one patient assessed simultaneously), not totally at random. Reading totally at random strongly decreases the signal to noise ratio.[2] Which method did the "random" reader apply exactly?

A third concern is with the analysis, although this may only be a question of the way in which the data are presented. Although the authors state that the main outcome measure is the change in radiographic damage, they only report medians and ranges of the absolute scores in the groups. From our reading of the report, we fear the analysis has (statistically) compared the distributions of these absolute scores rather than their changes.

This is an important study, and has the potential to add valuable information to our understanding of the best way to treat RA, but in its present form the radiographic results are more likely to cloud the issues than clarify them. We suggest that the radiographs are made available to be re-read by two new, experienced readers, with either the sequence known or unknown to both. We also suggest that the analysis should present the median, range, etc, of the changes in each group, and the test of the difference between these. (If they have a skewed distribution, then either transformation before parametric analysis or the use of non-parametric methods would be the best way to compare the groups.)

There are other difficulties with the study, although these are less important than the essential concerns noted above. For example, we are baffled by the statement in the introduction that the COBRA combination[3] "showed radiological advantage over sulfasalazine alone but the study was not powered to detect differences in *x* ray change". In fact, the differences in *x* ray change were among the key findings of the COBRA study, and have since been shown to increase over time.[4] So the study was not only adequately powered but also showed an unexpectedly large effect.

The authors diminish the value of the report by inappropriate interpretation of their secondary data, especially on the adverse effects. In the discussion they comment, "While observed toxicity from corticosteroids in terms of hypertension, weight gain, and osteoporosis could be reduced by active assessment and prompt intervention, there is no room for complacency". However, in their results section they report that, "Low dose aspirin and treatment for ischaemic heart disease remained similar, whereas the use of antihypertensive agents increased in both groups, as did prescription of lipid lowering agents. The use of any treatment for osteoporosis also increased in both groups" In fact there was no difference between the groups and thus there was no observed toxicity from glucocorticoids in their study. Further, the authors make no comment on their observation that (many) more patients in the placebo group than in the glucocorticoid group stopped sulfasalazine treatment owing to side effects.

In relation to weight gain, inappropriate attention to within-group changes leads the authors to conclude that body weight "increased significantly" in the glucocorticoid group (median gain 4 kg), with only a "borderline increase" in the placebo group (median gain 3 kg). Body mass index is handled in the same way. However, the only really relevant comparisons, those between groups, do not even show a trend to significance (all p values ≥ 0.10). As with the radiographic findings, the presentation of the table suggests end point results were compared rather than change scores.

Our interpretation of the clinical results contradicts that of the investigators, and we conclude that the effects on symptoms are in line with previous reports of limited and temporary advantages for disease activity, blunting of sulfasalazine toxicity, and extremely limited side effects when appropriate caution is applied. It is not possible to assess adequately the main results on *x* ray progression, which are at variance with several previously published studies,[3–8] and we urge the authors to allow a second read of the radiographs so that their important dataset can be added to the existing evidence.

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References

1 **Capell HA**, Madhok R, Hunter JA, Porter D, Morrison E, Larkin J, *et al*. Lack of radiological and clinical benefit over two years of low dose prednisolone for rheumatoid arthritis: results of a randomised controlled trial. *Ann Rheum Dis* 2004;**63**:797–803.
2 **Van der Heijde D**, Boonen A, Boers M, Kostense P, van Der Linden S. Reading radiographs in chronological order, in pairs or as single films has important implications for the discriminative power of rheumatoid arthritis clinical trials. *Rheumatology (Oxford)* 1999;**38**:1213–20.
3 **Boers M**, Verhoeven AC, Markusse HM, van de Laar MA, Westhovens R, van Denderen JC, *et al*. Randomised comparison of combined step-down prednisolone, methotrexate and sulphasalazine with sulphasalazine alone in early rheumatoid arthritis [see comments] [published erratum appears in *Lancet* 1998;**351**:220]. *Lancet* 1997;**350**:309–18.
4 **Landewe RB**, Boers M, Verhoeven AC, Westhovens R, van De Laar MA, Markusse HM, *et al*. COBRA combination therapy in patients with early rheumatoid arthritis: long-term

structural benefits of a brief intervention. *Arthritis Rheum* 2002;**46**:347–56.
5 **Kirwan JR**, Arthritis and Rheumatism Council Low Dose Glucocorticoid Study Group. The effect of glucocorticoids on joint destruction in rheumatoid arthritis. *N Engl J Med* 1995;**333**:142–6.
6 **Hickling P**, Jacoby RK, Kirwan JR. Joint destruction after glucocorticoids are withdrawn in early rheumatoid arthritis. *Br J Rheumatol* 1998;**37**:930–6.
7 **Rau R**, Wassenberg S, Zeidler H. Low dose prednisolone therapy (LDPT) retards radiographically detectable destruction in early rheumatoid arthritis—preliminary results of a multicenter, randomized, parallel, double blind study. *Z Rheumatol* 2000;**59**(suppl 2):II/90–6.
8 **van Everdingen AA**, Jacobs JW, Siewertsz Van Reesema DR, Bijlsma JW. Low-dose prednisone therapy for patients with early active rheumatoid arthritis: clinical efficacy, disease-modifying properties, and side effects: a randomized, double-blind, placebo-controlled clinical trial. *Ann Intern Med* 2002;**136**:1–12.

Authors' reply

We agree with John Kirwan and Maarten Boers that the assessment of radiographic damage in the WOSERACT study is of importance.[1] The method of reading radiographs has evolved since this study was planned in 1995.[2] Because we consider that ''the jury is out'', on the optimal way to read radiographs in studies the films were read (*a*) at random by one reader and (*b*) in sequence by the other reader, and the same conclusion was reached. This strengthens rather than weakens the case for a true result.

The study of Paulus *et al*,[3] in which there was no beneficial effect on radiographic outcome in 197 patients with rheumatoid arthritis (RA), supports the WOSERACT study findings. It was unfortunate that in the Arthritis and Rheumatism Council (ARC) low dose corticosteroid study the two groups were not well matched at the outset, making interpretation of the true effect of prednisolone at 2 years and of the subsequent report difficult.[4 5]

The COBRA study[6] used a high initial corticosteroid dose and the effects contributing to prompt disease control were multifactorial. It is similarly not possible to extrapolate from the study of van Everdingen *et al*,[7] because they used a protocol of steroid without initial disease modifying antirheumatic drug, which is not a practice supported by current guidelines on RA management.

There was considerable discussion among the WOSERACT investigators about the approach to glucocorticoid side effects. It was decided that management of these would be the responsibility of the individual consultant, who remained unaware of the treatment assignment. For this reason there is likely to be a great deal of background noise. This issue was not a primary end point of our study but information is available from other studies.[8] We do agree that the blunting of sulfasalazine toxicity in the active group is of interest, although it would be inappropriate to advocate the use of prednisolone for this reason alone.

At a time when multiple treatments are increasingly used in early RA it is vital to be certain what contribution, if any, oral corticosteroids might make. The fact that both the ARC study and ours showed no sustained clinical benefit, makes *x* ray interpretation all the more important.

Thus we suggest that with John Kirwan and Marten Boers an approach is made to the ARC (the original sponsors of the 1995 study), or to EULAR, for sufficient funding to allow independent readers and statisticians to evaluate all appropriate datasets. This would allow films from relevant studies to be copied and made available as a central repository for future study. The films from the study of Rau *et al*[9] would also be useful for this initiative.

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References

1 **Capell HA**, Madhok R, Hunter JA, Porter D, Morrison E, Larkin J, *et al*. Lack of radiological and clinical benefit over two years of low dose prednisolone for rheumatoid arthritis: results of a randomised controlled trial. *Ann Rheum Dis* 2004;**63**:797–803.
2 **van der Heijde D**, Boonen A, Boers M, Kostense P, van der Linden S. Reading radiographs in chronological order, in pairs or as single films has important implications for the discriminative power of rheumatoid arthritis clinical trials. *Rheumatology (Oxford)* 1999;**38**:1213–20.
3 **Paulus HE**, Di Primeo D, Sanda M, Lynch JM, Schwartz BA, Sharp JT, *et al*. Progression of radiographic joint erosion during low dose corticosteroid treatment of RA. *J Rheumatol* 2000;**27**:1632–7.
4 **Kirwan JR**, Arthritis and Rheumatism Council Low Dose Glucocorticosteroid Study Group. The effect of glucocorticosteroids on joint destruction in rheumatoid arthritis. *N Engl J Med* 1995;**333**:142–6.
5 **Hickling P**, Jacoby RK, Kirwan JR. Joint destruction after glucocorticostoids are withdrawn in early rheumatoid arthritis. *Br J Rheumatol* 1998;**37**:930–6.
6 **Landewe RB**, Boers M, Verhoeven AC, Westhovens R, van De Laar MA, Markusse HM, *et al*. COBRA combination therapy in patients with early rheumatoid arthritis: long-term structural benefits of a brief intervention. *Arthritis Rheum* 2002;**46**:347–56.
7 **van Everdingen AA**, Jacobs JW, Siewertsz Van Reesema DR, Bijlsma JW. Low dose prednisolone therapy for patients with early active rheumatoid arthritis: clinical efficacy, disease-modifying properties, and side-effects: a randomized, double-blind, placebo-controlled clinical trial. *Ann Intern Med* 2002;**136**:1–12.
8 **Fries JF**, Williams CA, Ramey D, Bloch DA. The relative toxicity of disease-modifying antirheumatic drugs. *Arthritis Rheum* 1993;**36**:297–306.
9 **Rau R**, Wassenberg S, Zeidler H. Low dose prednisolone therapy (LDPT) retards radiographically detectable destruction in early rheumatoid arthritis – preliminary results of a multicenter, randomized, parallel, double blind study. *Z Rheumatol* 2000;**59**(suppl 2):II/90–6.

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