PostScript

MATTERS ARISING

Treatment of shoulder pain

Hay and colleagues concluded, in their extended report of a trial of physiotherapy and injection for unilateral shoulder pain that physiotherapy and local steroid injection are of similar effectiveness.1 They suggest that “...the high overall success rates... argue against the need for further exploratory trials in this condition...”. I disagree. A large number of studies of shoulder pain have been bedevilled by diagnostic criteria that are not precise,2 and this study must unfortunately join the others.

Unilateral shoulder pain has a number of different causes. The study by Hay excludes a few specific conditions—in particular, a ruptured rotator cuff, but must by definition include a heterogeneous group of problems that are in fact quite discrete. These include frozen shoulder (adhesive capsulitis), rotator cuff injuries without full rupture, subacromial joint arthritis (sometimes known as subacromial bursitis), bicipital tendinitis, acromioclavicular joint disease, and subdeltoïd bursitis. It is barely credible to imagine that several of these could be successfully treated by a steroid injection into the subacromial joint. In particular, the subacromial joint does not communicate with the glenohumeral joint unless the rotator cuff is ruptured, so frozen shoulder cannot be treated with a subacromial injection. Thus any study of shoulder pain must separate the different causes into different groups. Others have done this and shown that the relative benefits of physiotherapy and injection may be different.3

Furthermore, it must be clear that any clinician contributing to a trial are working to the same diagnostic criteria. Even experienced consultant rheumatologists cannot agree on exact diagnoses, as I and colleagues have shown previously,4 and we concluded...”

In short, they report on 31 patients with rheumatoid arthritis (RA), 18 of whom used etanercept (ETA) first and then switched to infliximab (IFX) in most part because of inefficacy, and 13 patients who used IFX first and changed to ETA mostly owing to adverse events. They suggested using the other tumour necrosis factor (TNF) inhibitor when one of them fails. Although, in general, agreeing with their findings, we would like to present our experience which is somewhat different from theirs and to discuss the possible reasons for this.

We set up an IFX registry at the Hospital for Special Surgery in February 2000, with the start of IFX infusions. The registry collected prospective data on all the patients with RA who started treatment with IFX, and followed up them every 2 months until May 2001. All patients completed questionnaires about their RA history, treatment, and functional disability (modified Health Assessment Questionnaire (mHAQ)) at baseline and every 2 months thereafter. A 42 joint count for tender and swollen joints was performed at each visit. Patients were telephoned 3–5 days after infusions and asked about reactions while at home.

The availability of ETA before the approval of IFX and the fact that use of ETA did not require concomitant methotrexate has resulted in the treatment of more patients with ETA than patients with RA before trying IFX. However, after failure of ETA, several patients changed treatment to IFX. We compared response to treatment, adverse events, and discontinuation rates between patients for whom ETA had failed before IFX treatment (ETA-F) and patients who had not used ETA before—that is, etanercept-naive (ETA-N).

Eighty-eight patients were treated with IFX between February 2000 and May 2001. Seventy-three patients were women, mean (SD) age 61 (12.1) years, mean (SD) RA duration 13.4 (9.8) years, failed DMARDs 2). In 37 (42%) patients ETA had failed before IFX treatment (ETA-F) and patients who had not used ETA before—that is, etanercept-naive (ETA-N).

References

Do etanercept-naive patients with rheumatoid arthritis respond better to infliximab than patients for whom etanercept has failed?

We read with interest the article by van Vollenhoven et al. In short, they report on 31 patients with rheumatoid arthritis (RA), 18 of whom used etanercept (ETA) first and then switched to infliximab (IFX) in most part because of inefficacy, and 13 patients who used IFX first and changed to ETA mostly owing to adverse events. They suggested using the other tumour necrosis factor (TNF) inhibitor when one of them fails. Although, in general, agreeing with their findings, we would like to present our experience which is somewhat different from theirs and to discuss the possible reasons for this.

We set up an IFX registry at the Hospital for Special Surgery in February 2000, with the start of IFX infusions. The registry collected prospective data on all the patients with RA who started treatment with IFX, and followed up them every 2 months until May 2001. All patients completed questionnaires about their RA history, treatment, and functional disability (modified Health Assessment Questionnaire (mHAQ)) at baseline and every 2 months thereafter. A 42 joint count for tender and swollen joints was performed at each visit. Patients were telephoned 3–5 days after infusions and asked about reactions while at home.

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Eighty-eight patients were treated with IFX between February 2000 and May 2001: 77 women, mean (SD) age 61 (12.1) years, mean (SD) RA duration 13.4 (9.8) years, failed DMARDs 2. In 37 (42%) patients ETA had failed before IFX was introduced. There was no difference in age, disease duration, and number of failed DMARDs between ETA-F and ETA-N patients. Sixteen ETA-F and 10 ETA-N patients...
ETA-N patients were excluded from the analysis owing to an insufficient number of data points. mHAQ, pain scores, and morning stiffness were significantly reduced in ETA-N patients, whereas no improvement was noted among the group of ETA-F patients, in the first year after they were receiving IFX. Six ETA-F and seven ETA-N patients discontinued treatment after 4 and 5.7 months, respectively. No significant difference in the number of adverse events was found between ETA-F and ETA-N patients.

We also analysed the functional and rate of adverse events among patients with RA treated with IFX for those receiving concomitant methotrexate (MTX-R) and those not (MTX-NR). Baseline age and disease duration of MTX-R and MTX-NR patients were similar. IFX treatment was discontinued in 15/42 (36%) MTX-R subjects and 12/46 (26%) MTX-NR subjects. After an average of 6.7 months' follow up 40/61 subjects experienced 96 adverse events (AEs) over a total of 648 infusions; 16/27 (59%) MTX-NR subjects had 46 AEs, compared with 24/34 (71%) MTX-R subjects who had 50 AEs (p = 0.51). Most of these AEs were minor and none resulted in IFX discontinuation. There was no difference in mHAQ, pain score, swollen and tender joint counts between the MTX-R and MTX-NR groups after 6 months of treatment.

Our clinical experience demonstrates a better clinical response to IFX among ETA-naive patients. Based on our data, we would suggest that if ETA fails there might not be a substantial benefit in trying IFX later on. Also, we did not note any difference in the rates of discontinuation or AEs, or response to treatment between MTX-R and MTX-NR patients beyond 6 months of IFX treatment.

We are limited by the number of our patients, just as van Vollenhoven et al were. We also do not have data for patients who switched from IFX to ETA because of the shortage of ETA at the time of our study. These results may reflect a population of refractory patients with RA who have more severe disease (patients for whom multiple DMARDs had failed) and are generally difficult to manage, or who are non-anti-TNF responders. Analysis of ETA-F patients who respond to IFX may show a subgroup who will benefit from different anti-TNF formulations. Given the cost of anti-TNF drugs, larger groups should be studied to determine the characteristics of patients who might benefit from a trial of another anti-TNF agent when one has already failed.

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Reference
1 van Vollenhoven R, Harju A, Brannemark, S, Klareksag L. Treatment with infliximab (Remicade) when etanercept (Enbrel) has failed or vice versa: data from the STURE registry showing that switching tumour necrosis factor α blockers can make sense. Ann Rheum Dis 2003;62:1195-8.

Authors’ reply
We take Dr Jawad’s points and agree fully. We also believe that classification criteria are too often wrongly used in diagnosis. Our aim in writing the leader was to highlight what we believe to be a not uncommon diagnostic situation—the patient with many of the features of the syndrome in whom tests remain stubbornly negative.

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Reference
Hoffmann-La Roche Ltd, because of ethical concerns; it was decided at the time that patients should not continue to receive treatment potentially associated with suboptimal efficacy.

However, upon analysis of the results from the fracture prevention study, it became evident that the suboptimal antifracture efficacy was due to under dosing and that higher doses would probably provide the required gains in bone mineral density (BMD) and decreases in biochemical markers of bone turnover. The proposition that higher doses of ibandronate would produce optimal efficacy is supported by the results from a study that evaluated the dose-response relationship associated with intermittent IV ibandronate injections in postmenopausal women with osteoporosis. In that study, a 2 mg dose of ibandronate provided significantly greater BMD increases and suppression of bone resorption markers than the 1 mg dose investigated in the fracture prevention study.

The available results from our study were analysed to enable further evaluation of the dose-response relationship of intermittent IV ibandronate. As reported, although our study was terminated prematurely, sufficient results were collected for analysis of data for the first year of the study. These results support those seen previously: intermittent IV ibandronate produced a dose dependent effect on BMD and biochemical markers of bone resorption, with the highest dose producing the greatest efficacy. Owing to the findings from our study, together with those from prior studies, a large non-inferiority trial is continuing to determine whether a higher dose (3 mg every 3 months) or a shorter dosing interval (2 mg every 2 months) of IV ibandronate provides equivalent therapeutic efficacy relative to an oral daily ibandronate regimen, which has proved antifracture efficacy (the Dosing Intravenous Administration study).

In their second observation on our study, the authors imply that the observed changes in BMD might have been due to the random variability associated with measuring BMD, and even when adjusted, the 3% change exceeded the 2.8% variability of the measurement procedure, it is considered clinically relevant.

In their third comment, the authors correctly state that analysis of variance was used to investigate the efficacy of the three ibandronate regimens. They go on to advise that because multiple comparisons were performed, adjustments should have been made to ensure the maintenance of the overall type I error. However, as only three tests were performed for the primary end point (the relative effect of treatment on lumbar spine BMD), all of which were highly significant, it was not deemed necessary to apply adjustments.

Additional analyses were conducted to determine the influence of baseline spinal BMD and/or time since menopause on the primary end point. In retrospect, as a number of comparisons were performed when conducting these secondary analyses, we agree with Maravic and Landais that relevant adjustments would be optimal. Thus, we have retrospectively applied a Bonferroni correction, a well recognised test to adjust for multiple comparisons. Despite the highly conservative nature of such statistical adjustments, we believe that the variability in the number of repeated tests is >5, only two results changed (table 1).

Therefore, reassuringly, the findings observed after applying the Bonferroni correction were similar to those reported in our publication.

In summary, although our randomised, double blind, placebo controlled study was prematurely discontinued, sufficient 1 year data from almost 600 patients were available to allow an adequate assessment of the efficacy of IV ibandronate injections in postmenopausal women without osteoporosis. We found that ibandronate dose dependency prevents postmenopausal bone loss, even when considering the variability associated with measuring BMD, and even when adjusting for multiple, repeated statistical assessments. Of note, the highest dose of ibandronate produced gains in BMD that were comparable to those observed with other therapeutic agents in this setting.

Table 1

<table>
<thead>
<tr>
<th>Stratum</th>
<th>Time since menopause (years)</th>
<th>Ibandronate</th>
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</thead>
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<tr>
<td></td>
<td>Placebo</td>
<td>0.5 mg</td>
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<tr>
<td>t score</td>
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</tr>
<tr>
<td>&gt;-1</td>
<td>1-3</td>
<td>-0.0</td>
</tr>
<tr>
<td>&lt;-1, &gt;-2</td>
<td>2.5-1</td>
<td>-0.7</td>
</tr>
<tr>
<td>&lt;-1, &gt;-3</td>
<td>3</td>
<td>-0.4</td>
</tr>
<tr>
<td>&lt;-1, &gt;-2.5</td>
<td>3</td>
<td>-0.2</td>
</tr>
</tbody>
</table>

*Difference between active group and placebo was significant (p<0.05), even if a Bonferroni correction was applied; †According to an ANOVA test, the difference between active group and placebo was significant (p<0.05). Significance was not achieved when a Bonferroni correction was applied.
Thus, as stated in the conclusion of our publication, intermittent IV ibandronate injections offer the promise of an effective and convenient alternative to current treatments for the prevention of postmenopausal bone loss.

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References

Atherosclerosis in primary antiphospholipid syndrome

We read with great interest the article by Medina et al about atherosclerosis in primary antiphospholipid syndrome (APS). They showed that the intima-media thickness (IMT) of carotid arteries from patients with primary APS was greater than that of age and sex matched controls.

We wish to comment on their methodology and findings. For the ultrasound technique Medina et al did not define the area where the measurement of IMT was taken, nor did they specify whether the anterior or posterior carotid wall was measured. The anterior or near wall is prone to reverberation artefact, making placement of the callipers difficult.

IMT should be measured at a site away from atheromatous plaques. The figures provided by Medina et al show that colour and power Doppler were used to define the lumen of the vessel, but there is no mention of how these were standardised. The gain, pulse repetition frequency, and selection of colour versus grey scale, use of contrast agents, angle of insonation, and the velocity of blood flow (systole or diastole) will all affect the position of the border of the colour echo in the image and hence affect the readings taken. Their fig 2B shows that the colour echo does not conform to the “white line” of the intimal-luminal interface. Furthermore, the placement of the measurement callipers in figs 2B and 3B is not correct. Here most workers advise that the image be magnified to facilitate accurate calliper placement. Also, the effect of laminar or plug flow patterns may influence readings. With their technique even IMT readings in their controls are far higher than those reported in the world literature (reviewed by Aminabhavi and Mancini).

We would have liked to see the antiphospholipid antibody (aPL) titre of the patients and how they ranked if entered in their regression model in table 3. A previous study identified IgG anticalcineurin antibody (aCL) as an independent predictor of carotid IMT, suggesting a possible dose effect of IgG aCL on IMT. Therefore aPL titres at the lower end of the medium range (20–80 GPL) may be less atherogenic than those at the higher end of the same range.

To confuse matters further, most of their patients with primary APS had other significant conventional risk factors for atherosclerosis, whether in isolation or combination. The prevalence of hypertension, hyperlipidemia, and obesity was 36%, 54%, and 25%, respectively, in their cohort with several patients having more than one risk factor. We question the author’s conclusion of atherosclerosis in primary APS. As it stands, their scanning technique overestimates IMT readings, and other conventional cardiovascular risk factors are overrepresented in their cohort, potentially masking the atherogenic potential of aPL.

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References

Authors’ reply

We thank Dr Ames et al for their interest in our study and for their comments about our methodology and findings of atherosclerosis in primary antiphospholipid syndrome.

We agree about the difficulties of anterior carotid wall measurements. Therefore our intima-media thickness (IMT) measurements were performed on the posterior wall, as is shown in fig 2B. Our patients did not have atheromatous plaques, consequently this artefact did not alter the IMT measurement. The colour and power Doppler ultrasound were standardised in our hospital according to the method of Cantú-Brito et al. Briefly, the sensitivity and specificity of the method were 91.3 and 92.7%, respectively. A composite measure that includes the maximal common carotid artery IMT and maximal internal carotid artery IMT was obtained by averaging these two measurements after standardisation with subtraction of the mean and division by the standard deviation for the measurement.

Colour Doppler cannot define the “white line”, but this technique does define the intimal-luminal interface. The position of the callipers in fig 2B is correct for IMT measurement, but in fig 3B the callipers are showing the indentations in the lumen vessel.

The carotid artery IMT in our controls was 1.2 (0.44) mm. This value is higher than that reported in other studies but, however, only 7/28 controls had an IMT >1 mm. The mean IMT in these seven controls was 1.65 mm (range 1.1–2). The explanation of these results is that the controls also had other cardiovascular risk factors and they were hospital workers. In other studies performed in Mexico, IMT was detected in more than 60% of people from the general population.

In our regression model we did not include the titres of anticalcineurin antibodies, because we did not determine them simultaneously with the carotid artery IMT study. We have only historical data of antiphospholipid antibodies. We are currently analysing the association of antiphospholipid antibodies and other new risk factors with carotid artery IMT.

In relation to conventional risk factors for atherosclerosis, we agree that these are confusing variables, but the logistic regression analysis controlled for these factors. Certainly, it is very difficult to have patients and controls who do not have traditional risk factors for atherosclerosis.

In view of all the above mentioned information, we think that our patients with primary APS had a significantly greater IMT of the carotid arteries than the controls.

The results are not overestimated because all ultrasound scanning was performed under similar conditions in both groups, by a single experienced radiologist who was unaware of the clinical information, and using the same sonograph equipment.

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References
3 Rodríguez-Saldarriaga J, Cantú-Brito C, Sosa Espinoza P, Reynoso-Mareno MT, Zuckermann Foullon D, Barragarantermeier-Aldaz F.
Inflammatory rheumatic disorders

I was interested to read the paper by Guillemin et al, where the authors used a detailed questionnaire for a telephone survey of patients to assess rheumatic problems.1 I have used a similar but somewhat more extensive questionnaire, which was initially designed for doctors such as general practitioners to complete and return through my cyberclinic project.2

In this study we evaluated the feasibility of a rheumatology consultation and advisory service using internet and email. We devised and posted a detailed rheumatology pro forma on our website (www.cyberrheum.org; accessed 26 February 2004) for general practitioners to complete and return on the internet. Two trainee doctors interviewed and completed the pro forma for 271 new patients. Based on this information from the questionnaire alone we were then able to provide provisional diagnoses and management plans, which we compared with those drawn up after a face to face assessment of the same patients in the outpatient clinic. Diagnostic concurrence was noted in the majority of patients (86%), no changes in further investigations, such as radiographs and blood tests, in a sizeable proportion of patients (62%) were necessary, and the majority of patients (74%) required no changes in the treatment plans suggested. Our results show that it is quite feasible to offer an internet based outpatient consultation and advisory service in rheumatology and possibly other disciplines. Perhaps it is more practical in rheumatology because most of the diagnostic clues lie in a detailed history taken from the patients (as in the patient questionnaire we devised), only then aided by clinical examination and laboratory tests.

To our knowledge this was the first and only internet based service available to general practitioners. This, when established, can enable centres to streamline referral processes and provide early diagnosis and management plans for patients, even before they reach the clinic, which therefore works as a review appointment (as all the results and details of the patients are available when first attending the clinic). As a result we can cut the consultation time in the clinic to half the usual time required, a consequence of which is that we can see more new patients and perhaps also see more review patients.

We noted that general practitioners were finding it difficult to complete the pro forma on the website with the patients. Therefore, subsequently we devised a patient completed questionnaire, available on the same website, so that patients could complete this pro forma at the GP surgery with the help of an assistant such as a nurse. This facilitates referral and reduces demand on general practitioners’ time. We have been able to devise a separate questionnaire,3 again posted on our website, to facilitate referrals relating to osteoporosis services, and reduce our response time to management plans.

We are therefore not surprised that the authors found such a patient questionnaire helpful for telephone surveys. Compared with telephone consultation in this manner, an internet based questionnaire format would appear to be more convenient and less time consuming. Patients can take their own time in a relaxed manner to do the same. Furthermore, two people are not tied down at the same time—that is, patients can complete and return the questionnaire when convenient, and the researchers or the clinicians can assess such questionnaires at leisure or when convenient. Furthermore, more patients have access to telephone than internet and email, and many do not yet know how to use the latter. Hence, in recent years we have provided “hands-on” training and instructions with a number of “road shows” for our patients to keep abreast with new information technology and to increase “equity” and access for our patients in these days of “modernisation” in the health services.

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References
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Authors’ reply

We appreciate Dr Pal’s presentation of a rheumatology consultation and advisory service posted on a website for use by general practitioners, further completed with patient’s targeted online questionnaire to facilitate referral. Admittedly, this is not only because people have more access to telephone than internet and email but also, above all, because people in the general population, the target of such a survey, including people with and without disease, will not spontaneously answer an unexpected questionnaire without a minimum of information and invitation. There would be a severe risk of underestimating the denominator and thus biasing the prevalence.


Mycobacterial knee infection in patients with idiopathic inflammatory myositis

We read with interest the report by Callaghan and Allen about a patient with inflammatory myositis who developed Mycobacterium malmoense infection in her right knee.1 The authors state in their discussion that they reviewed the literature and found no cases of isolated joint infection with this organism. However, in this journal, we have previously reported three patients with inflammatory myositis who developed mycobacterial knee infections, one of which was with M malmoense.2 It is interesting to see that another case of infection with this rare organism has been described in a patient with inflammatory myositis. We suggest that it is sensible to ensure that synovial fluid aspirated from joints of patients with autoimmune rheumatic disease receiving long term immunosuppressive drugs is sent specifically for mycobacterial culture. Possibly, some atypical mycobacterial infections are being undiagnosed. Such infections could become more common with the increasing use of biological therapies for autoimmune rheumatic disease.

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References

Author’s reply

Apologies for not picking up this interesting article in our literature search. Drs Isenberg and Haq are, of course, right. Thank you for letting us know.

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Increased risk of cancer in patients with scleroderma: no risk in patients with morphoea?

In a recent issue of the Annals Hill et al reported a risk of cancer in patients with (systemic) scleroderma. The authors found an increased risk for all cancers, but the greatest significantly increased relative risk was for lung cancer.

Published data do not indicate an increased risk of cancer for patients with localised scleroderma or morphea in general.2 But that statement needs a closer look. Although there are no population based studies in this field, there is some evidence that patients with pansclerotic or generalised
morphea with longstanding ulcerations of skin or scars are at risk for the development of squamous cell carcinoma. In a recent overview we found seven case reports on squamous cell carcinoma in patients with pansclerotic or generalised morphea.1 The number seems remarkably high because this disease is extremely rare. The observation has not only some impact on follow up but on treatment as well. The use of ultraviolet based treatments like UVAI or PUVA (psor- alen plus UVA) widely used successfully for more severe or widespread forms of morphea1 might further increase the risk of squamous cell carcinoma in these patients.

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References

Author’s reply
Dr Wollina’s observations are of great clinical and research interest. Unfortunately, we excluded patients with localised scleroderma in our study1 owing to incomplete ascertain- ment in South Australia. We agree that further investigation of this interesting area is needed, particularly in relation to the influence of treatment. However, epidemiological studies of rare diseases are a challenging area of research.

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doi: 10.1136/ard.2003.014829

MSc Programme in Clinical Rheumatology
Applications are invited for places on this MSc programme, starting September 2004, which provides an excellent academic basis for those aiming at a career in rheumatology or a related subject. Applicants should be medically qualified and should have had at least 2 years of general medical experience after qualification. Previous experience in rheumatology is desirable, but not essential. The programme is undertaken part time over 2 years and is now well established, entering its 10th year.

Topics covered will include: basic science, clinical skills, peripheral joint problems, spinal problems, connective tissue disease and vasculitis, and the epidemiology of musculoskeletal diseases. A supervised pro- ject, which may be either clinical or labora- tory based, is an integral part of the programme. The closing date for applications is 31 May 2004.

Further details can be obtained from Miss Lisa McClair, ARC Epidemiology Unit, Stopford Building, University of Manchester, Oxford Road, Manchester, M13 9PT, UK. Tel: (0) 161 275 5993. Fax: (0) 161 275 5043. Email: Lisa.mcclair@man.ac.uk

NOTICE
MSc Programme in Clinical Rheumatology

First European Course:
Capillaroscopy and Rheumatic Diseases
10–12 September 2004; Genova, Italy
Contact: Scientific Secretariat: Professor Maurizio Cutolo, Division of Rheumatology, DIMI, University of Genova, Italy Email: mcutoledo@unige.it Organising Secretariat: Michela Civelli, EDRA spa, Viale Monza , 133 – 20125, Milan, Italy Tel: +39 02 281 72300 Fax: +39 02 281 72399 Email: edracorrigens@dsmedigroup.com

Xth International Conference on Behcet’s Disease
27–31 October 2004; Antalya, Turkey
Contact: Congress Secretariat, Figur Congress and Organization Services Ltd. STI, Ayazmadesi Cad. Karadut Sok. No: 7 80888 Dikilitas, Istanbul, Turkey Tel: (+90 (0212) 258 6020 Fax: (+90 (0212) 258 6078 Email: behcet2004@figur.net Website: www.behcet2004.org

4th International Congress on Autoimmunity
3–7 November, 2004; Budapest, Hungary
Deadline for receipt of abstracts: 20 June 2004
Contact: 4th International Congress on Autoimmunity, Kennes International—Global Congress Organisers and Association Management Services, 17 rue du Cendrier, PO Box 1726, CH-1211 Geneva 1, Switzerland Tel: +41 22 908 0488 Fax: +41 22 732 2850 Email: autoim04@kennes.com Website: www.kennes.com/autoim2004

8th EULAR Postgraduate Course in Rheumatology
28 November–3 December 2004; Prague, Czech Republic
Contact: EULAR Secretariat, Wiltikonstrasse 15, CH 8032 Zurich, Switzerland Tel: +41 1 383 96 90 Fax: +41 1 383 98 10 Email:secretariat@eular.org Website: www.eular.org

Osteoarthritis Research Society International
2–5 December 2004; Chicago, USA
Contact: 17 000 Commerce Parkway, Suite C, Mt Laurel, NJ 08054, USA Email: oarsi@oarsi.org Tel: +1 856 439 1385 or visit http://www.oarsi.org

Vth European Lupus Meeting
3–5 March 2005; Royal College of Physicians, London, UK
Contact: Julia Kerme, Conference organiser of the British Society of Rheumatology Email: Julia@rheumatology.org.uk

Future EULAR congresses
8–11 June 2005; EULAR 2005; Vienna, Austria
21–24 June 2006; EULAR 2006; Amsterdam, The Netherlands

Future ACR meeting
16–21 October 2004; 68th Annual Scientific Meeting: San Antonio, Texas
Increased risk of cancer in patients with scleroderma: no risk in patients with morphea?

U Wollina

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