(patient 2) and one niece were the only piano players, but the subjects’ four brothers have been active in karate for years. Apart from the subjects and their mother, who had OA in a knee and in several interfacet joints, no participants had musculoskeletal complaints.

In this family, the high frequency of varying degrees of hypermobility but low prevalence of health complaints point to a benign autosomal dominant condition. Both subjects qualify for a diagnosis of FAH (a minimum Beighton score of 4) [3]. They presented with symptoms suggestive of a carpal tunnel syndrome (confirmed in the first subject), probably the result of repetitive overuse [4, 5]. Each showed the typical radiographic features of OA of the ST/STT joints: joint space narrowing and subchondral sclerosis but no osteophytes [6].

Lone ST/STT OA is uncommon [1, 2] and occupationally induced OA of the upper extremities is said to be rare [7], but one of the authors (JRM) has observed isolated STT OA in two heavy equipment operators who also were loggers using chainsaws. Neither gave a history of hypermobility. In cases like these, there is no consensus on whether the degenerative process is the result of vibration-induced damage to the articular cartilage, heavy joint loading or both [7]. If heavy occupational stresses can cause STT OA in structurally normal joints, it is easy to imagine that lax or unstable joints would be more vulnerable to such stresses. Carpal instability (malalignment of one or more carpal bones in relation to each other or the radius) has been observed in generalized lax ligaments [8, 9]. Furthermore, STT OA has been associated with carpal instability [10, 11]. Some carpal instability was still present in the second subject and was perhaps present in both subjects earlier in life to a more marked degree, as joint ROM may diminish with age [12].

Considering how common FAH is [13], as this family shows, usually it is well tolerated. Few cases develop secondary OA and then, presumably, only under exceptional circumstances, such as unusual occupational and recreational mechanical stresses acting on susceptible joints. In the case of the propositus, there would have to be a strong suspicion that it was the exceptional demands of his occupation which were responsible for his OA. In the case of his affected sister, however, none of her activities, except possibly piano playing, stand out as a major determinant for her arthropathy.

We are indebted to Professor M. Byers, University of Washington, for the electrophoretic studies of the skin biopsy of patient 2, to Dr N. Turner for bringing the propositus to our attention, to Ms K. Hodgkinson for providing the pedigree, and to the family for their cordial participation in the study.

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saliva of Sjögren’s syndrome (SS) patients [2]. Perrier et al. [3] studied 36 patients with primary SS (pSS) and found that the IL1RN*2 allele was more frequent in definite pSS than in the possible form of the disease [3]. We have characterized two single-nucleotide polymorphisms of IL-1 complex genes (i.e. IL-1α –889 and IL-1β +3953 polymorphism) and the above-mentioned IL1RN polymorphism in DNA samples of 65 pSS patients (63 female and two male, mean age 60 ± 12 yr) using the PCR (polymerase chain reaction), RFLP (restriction fragment length polymorphism) and PAGE (polyacrylamide gel electrophoresis) [for methods see references 4 and 5]. Patients were selected using modified Californian criteria as described previously [6]. Moreover, all patients fulfilled the European criteria for pSS [7]. The data on the patients were compared with data obtained on 180 healthy female blood donors (Finnish Red Cross Blood Transfusion Centre, Tampere, Finland).

In these analyses the individual genotype distributions and allele frequencies of IL-1 complex genes were similar in pSS patients and in control subjects (χ² test for 2 × 2 and 2 × 3 contingency tables, 1 and 2 degrees of freedom; data not shown). Moreover, no difference was observed between IL1RN*2 distributions in definite (n = 30) and possible (n = 35) forms of pSS. As the IL-1 complex genes are in linkage disequilibrium, we carried out a haplotype analysis for these polymorphic loci using the expectation-maximization approach [8, 9]. In these analyses the IL-1 gene complex haplotype frequencies (Table 1) were nearly the same in the pSS group and in the control group (P = 0.47189, χ² test of 2 × 9 haplotype frequency table, 8 degrees of freedom). These complementary results strongly support the null hypothesis of no association between IL-1 gene complex polymorphism and pSS.

As the IL-1 gene complex polymorphism may affect the clinical course of pSS, we made an effort to analyse the potential associations of these markers with clinical and laboratory findings of pSS, such as joint pain, Raynaud’s phenomenon (RP), recurrent parotid gland swelling, hypergammaglobulinaemia and positivity for SS-A/ Ro or SS-B/ La antibody. Although no differences were observed in the IL-1 gene complex polymorphism distributions between the patients and controls, the IL-1 gene complex alleles were unevenly distributed in pSS patients with (n = 36) and without (n = 29) RP (diagnosed on the basis of patient history or the observation of cold-induced pallor and cyanosis of the fingers or toes). In the RP-positive patient group, the proportion of subjects homozygous for IL-1α –889 allele 1 was lower (33%) and the proportion of 1/2 heterozygous subjects higher (56%) than in the RP-negative group (homozygotes 69%, P = 0.0043; heterozygotes 38%, P = 0.0443; χ² test of 3 × 2 contingency table, 2 degrees of freedom). Moreover, the proportion of subjects homozygous for IL-1 +3953 allele 1 was significantly lower (55%) and the proportion of 1/2 heterozygotes higher (50%) in the RP group than in the RP-negative group (allele 1 homozygotes 76%, P = 0.0106; heterozygotes 14%, P = 0.0051). In line with these observations, the haplotype estimation analysis suggested a difference in haplotype frequencies between RP-positive and RP-negative pSS groups (P = 0.0386, χ² test of 2 × 8 haplotype frequency table, 7 degrees of freedom). The frequency of IL-1α allele 1, IL-1β allele 1 and IL1RN*1 haplotype (i.e. 1-1-1) was decreased and IL-1α allele 2, IL-1β allele 2 and IL1RN*3 haplotype (i.e. 2-2-1) increased in the RP group compared with the patients without RP (Table 1).

These results suggest that individual IL-1α –889, IL-1β +3953 or IL1RN polymorphisms or their haplotypes do not predispose patients to pSS. It can further be concluded from this data that factors other than an allelic imbalance of IL-1 gene complex genes cause the aberrant cytokine profiles observed in pSS. The observations made within the pSS group suggest that IL-1 complex genes or other, still unidentified, adjacent genes linked to them may contribute to the manifestation of RP in pSS. Due to the subtle nature of allelic effects, substantially larger numbers of patients or pedigree data sets are needed to confirm these preliminary results. It also remains to be explored whether genes of the IL-1 complex are associated with primary RP or with secondary RP that is associated with other diseases.

### Table 1. IL-1 haplotype frequencies

<table>
<thead>
<tr>
<th>Haplotype^a</th>
<th>Group 1 (n = 65)</th>
<th>Group 2 (n = 180)</th>
<th>Group 3 (n = 36)</th>
<th>Group 4 (n = 29)</th>
<th>Pairwise comparisons of groups^b</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-1-1</td>
<td>0.412</td>
<td>0.402</td>
<td>0.313</td>
<td>0.526</td>
<td>3 &lt;4, P = 0.0134</td>
</tr>
<tr>
<td>1-1-2</td>
<td>0.263</td>
<td>0.260</td>
<td>0.298</td>
<td>0.231</td>
<td>NS</td>
</tr>
<tr>
<td>2-2-1</td>
<td>0.191</td>
<td>0.237</td>
<td>0.281</td>
<td>0.096</td>
<td>3 &gt;4, P = 0.0135</td>
</tr>
<tr>
<td>2-1-2</td>
<td>0.030</td>
<td>0.035</td>
<td>0.038</td>
<td>0.022</td>
<td>NS</td>
</tr>
<tr>
<td>2-1-1</td>
<td>0.041</td>
<td>0.022</td>
<td>0.045</td>
<td>0.031</td>
<td>NS</td>
</tr>
<tr>
<td>1-2-1</td>
<td>0.0025</td>
<td>0.022</td>
<td>0.0</td>
<td>0.053</td>
<td>NS</td>
</tr>
<tr>
<td>1-1-3</td>
<td>0.008</td>
<td>0.011</td>
<td>0</td>
<td>0.017</td>
<td>NS</td>
</tr>
<tr>
<td>2-2-2</td>
<td>0.030</td>
<td>0.006</td>
<td>0.025</td>
<td>0.023</td>
<td>NS</td>
</tr>
<tr>
<td>1-2-2</td>
<td>0.000</td>
<td>0.005</td>
<td>0</td>
<td>0</td>
<td>NS</td>
</tr>
</tbody>
</table>

^aPresented in genetical order (sites from left to right: IL-1α –889, IL-1β +3953, IL1RN).

^bPost hoc χ² test for 2 × 2 haplotype frequency table with 1 degree of freedom.
such as limited or diffuse cutaneous systemic sclerosis or systemic lupus erythematosus.

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Time to review the distribution of rheumatology National Training Numbers

Sir, In the survey by Price et al. [1] on behalf of the British Society for Rheumatology Research and Training Committee, East Anglia emerged with the dubious distinction of having the smallest number of rheumatology NTNs (National Training Numbers) of any deanery in the UK and also the highest proportion of training posts occupied by non-NTN holders. The true position was actually bleaker as one of the five NTNs was only temporary. This high occupancy of NTNs by LAT (Locum Appointment for Training) holders results in an exceptionally high turnover of trainees.

Our efforts to increase our number of rheumatology specialist registrars over the past 5 yr have been singularly unsuccessful, due partly to a freeze on the national quota of NTNs and partly to the understandable reluctance by regions to relinquish any of their trainees, with the consequent inevitable disruption of popular and successful rotations. The situation has scarcely improved following the redrawing of deanery boundaries in April 2001 to create a new Eastern Deanery encompassing East Anglia, Essex and Hertfordshire. This exercise added just two extra rheumatology NTNs, giving a meagre total of six for this large geographical area. Moreover, as the two additional posts both rotate with hospitals in Thames Deanery, there has been no opportunity to satisfy the training aspirations of the rheumatology departments in Ipswich, Peterborough and Stevenage or to upgrade the LAT posts in Bury St Edmunds and Luton.

The present distribution of rheumatology training posts is largely historical. As the provision of rheumatology services has expanded and become more uniform across the UK, it is time that the spread of training provision was changed to reflect this.

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A comparison of the views of rheumatologists, general practitioners and patients on the treatment of osteoarthritis

Sir, Osteoarthritis (OA) is the most common form of joint disease and is an almost universal problem in people aged over 65 yr [1, 2]. Current management remains largely symptomatic [3] and involves a wide variety of options provided by a multitude of health professionals [4]. Given the number of professions involved in treatment and the importance of service provision for the successful management of chronic conditions [5], it is perhaps surprising that there is very