This study failed to demonstrate an association of *TNFR2 T676G* polymorphism with pSS. These results, together with the lack of efficacy of infliximab in pSS[7] rather suggest a poor involvement of the TNF/TNF receptor system in the susceptibility to pSS.

To date, among rheumatic diseases, *TNFR2 T676G* polymorphism association with lupus led to conflicting results.[4 6 8 9] A recent case–control study performed in an Asian population of patients also failed to demonstrate any association of *TNFR2 T676G* polymorphism with SLE.[10] However, this study also provided a metaanalysis of seven case–control studies in eight different ethnic populations. Stratification by ethnicity revealed a significant association among Asians while the effect of *TNFR2 T676G* polymorphism on SLE was not significant in two case-control studies involving Caucasians (OR = 0.99, 95%CI = 0.68 to 1.45; p = 0.96). Thus, as observed among Caucasian SLE patients, our study provides evidence suggesting that *TNFR2 T676G* polymorphism is not involved in the genetic predisposition to pSS in French Caucasian patients.

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International survey on the diagnosis and management of gout

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The pathogenesis of urate crystal deposition is reasonably well understood, and with appropriate urate-lowering therapy (ULT) and lifestyle advice, the objective of management is cure. Nonetheless, many patients with gout continue to experience frequent and recurrent episodes of gout and progression of their disease. This is because the condition is often misdiagnosed, or diagnosed late, and treatment is frequently suboptimal.[1 2] These concerns led to the development of evidence-based recommendations relating to the diagnosis and management of gout by a task force from the European League Against Rheumatism (EULAR). The EULAR recommendations were developed using a combination of research-based evidence and expert consensus, and provide a valuable resource for physicians.[1 2]

The current diagnosis and management of gout was investigated in a survey conducted among delegates attending EULAR 2006. Delegates visiting the commercial stands were invited to complete a written questionnaire of 12 questions designed to evaluate awareness of the impact of gout, the goals of treatment, and patterns of clinical practice. A total of 741 respondents (6.7% of attendees), predominantly rheumatologists from Europe, completed the questionnaire.

The results indicate a good level of understanding concerning gout and its management, but also highlight some marked variations in clinical practice.

Respondents reported using both the measurement of serum uric acid (sUA) (97%) and the examination of synovial fluid (91%) to confirm suspected gout. Only 15% said they would prescribe ULT when the diagnosis of gout was made, most reported that they would usually wait for the next gout attack (24%) or for at least two further attacks (32%) to occur before initiating treatment. Whereas 29% said they would initiate ULT during an attack, the remainder confirmed they would wait until after an attack, and most (60%) would then begin therapy

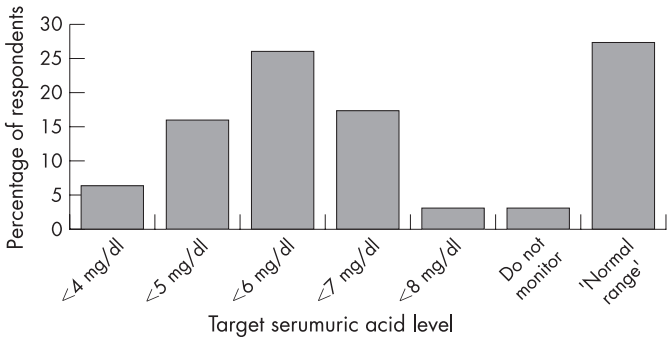

Figure 1 Discordance between doctors concerning the level of serumuric acid that is thought to inflect aduquete controlf of gout.

within 2 weeks. Sixteen per cent of respondents would treat asymptomatic hyperuricaemia with ULT, with a marked difference in practice being observed between rheumatologists and internal medicine specialists (15% versus 59%, respectively). Importantly, opinions varied considerably on what sUA level might be regarded as representing adequate control in a gout patient (fig 1).

It is interesting to consider some of these findings in light of the recent EULAR recommendations. These make clear, for example, that sUA levels do not confirm or exclude a diagnosis of gout. Despite this, nearly all respondents (97%) stated that they would rely on measuring sUA for precisely that purpose. For gout management the recommendations assign a central role to sUA and define the therapeutic goal of ULT as being to promote crystal dissolution, prevent crystal formation and improve clinical symptoms by maintaining sUA below 6 mg/dl. The survey findings show, however, that most physicians perceive a reduction in the frequency of acute attacks as the central goal of therapy and that the ability to cure the condition by maintaining sUA below 6 mg/dl is not yet universally appreciated.

A caveat of any such survey is that physicians may have responded on the basis of what they know should be done rather than what they actually do in practice. They may also have completed the survey in haste and possibly misinterpreted some of the questions. Nevertheless, a large number of practitioners representing several countries in Europe participated in this simple survey.

The authors hope that, as awareness of the EULAR recommendations grows, and the goal of maintaining sUA below 6 mg/dl becomes more widely recognised, all patients with gout will be spared the burden of this eminently treatable condition.

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Like father, like son

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We present a unique case of familial primary Sjogren's syndrome (pSS) involving a father and son that challenge several key features of this disease onset.

A 20-year-old man (fig 1) was referred with a 4-year history of recurrent parotitis and persistent bilateral parotid swelling. He also had increasing fatigue and night sweats for 3 months and cosmetically unacceptable parotid swellings. Although Schirmer's test was normal, he was found to have positive Ro, La and antinuclear antibodies. Computerised tomography scans of his abdomen and chest showed no abnormality. Subsequent parotid biopsy showed a low-grade mucosa-associated lymphatic tissue (MALT) lymphoma. A diagnosis of pSS that was complicated by MALT lymphoma was made. The parotid swellings were reduced by 50% with no further fatigue or night sweats after the introduction of prednisolone 30 mg once daily and hydroxychloroquine.

On subsequent inquiry, his 55-year-old father was found to have had a 15-year history of bilateral parotid swellings for which he never sought medical attention (fig 2). He was a mechanical engineer who gave a 15-year history of dry eyes and mouth and recurrent cervical lymphadenopathy. His Schirmer's test was strongly positive, with measurements of 2 mm in his left eye and 0 mm in his right. He also had positive Ro and La antibodies along with hypergammaglobulinaemia (immunoglobulin (Ig)G 25.2 (4.9–16.1) g/l, IgM 3.95 (0.60–2.1) g/l), lymphopenia (lymphocyte 0.4×10^9 (1.1–3.5)/l) and an erythrocyte sedimentation rate of 92 (1–10) mm/h. As he remained relatively asymptomatic apart from his ocular and oropharyngeal dryness, he was treated with artificial tears only.

This case report highlights several unusual features. Although familial clustering of different autoimmune diseases in individuals with pSS have been reported, familial cases of

Abbreviations: MALT, mucosa-associated lymphatic tissue; pSS, primary Sjogren's syndrome