Further studies are needed to clarify the biological significance of the soluble form of interleukin-2 receptor in systemic sclerosis and, by longitudinal follow up of several patients, its possible use for monitoring the progression of the disease.

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Nature of the ossicular joints and their involvement in rheumatoid arthritis

Sir: I read with interest the paper published recently in the Annals which, unlike most other rhinological articles I have seen, is based on evidence in experimental animals. I note the use of the word by Reiter et al as a reference for this statement, but examination of his paper shows that his assertion is not based on references, hypothetical or anatomic, to well-defined rhinological facts. In 1981 the ossicular joints were described by Anson as "diarthrodial joints surrounded by a thin capsular ligament. From the inner surface of the capsular ligament a wedge-shaped circular rim projects into the joint cavity and incompletely divides it."1 There was no mention of synovium or a cartilaginous disc. Modern understanding of the physiology of sound conduction through the middle ear emphasizes the importance of the ossicles acting only as a piston without significant rotation about the joints.2 In man the incudomalleolar joint seems to have no function apart from binding the two bones together as there is no movement at the joint. Indeed in many rodents there is no joint and the malleus and incus are one bone. The most important factor changing the low pressure, high displacement vibrations of air into high pressure, low displacement vibrations suitable for driving cochlear fluids is the ratio of the areas of the tympanic membrane and the stapes footplate.

Ankylosing spondylitis and selective IgA deficiency

Sir: We read with interest the account from Dr Herrera-Beaumont and colleagues of a patient with selective IgA deficiency and severe spondyloarthropathy.3 We report a further case of IgA deficiency in ankylosing spondylitis, a condition of an adult woman 4 years old who was noted to have spontaneous inflammation by her family doctor when she was aged 27 years.

Onset of symptoms

Onset of symptoms was at 17 years of age when she noticed swelling of bilateral knees and ankles, classical signs of Mediterranean fever. A biopsy from the iliac bone revealed a typical granulomatous lesion. Culture was sterile and she had no local or constitutional symptoms. This was followed by a few episodes of self-limiting upper respiratory tract infections. She was treated with ceftriaxone and erythromycin, and the symptoms resolved. At 18 years of age, she experienced her first episode of inflammatory back pain and stiffness at the age of 27. Over the following years she also experienced intermittent back pain and stiffness, often accompanied by constitutional symptoms such as fatigue and fever. She was treated with non-steroidal anti-inflammatory drugs, analgesics, and exercise. The course of her disease was unremarkable and did not feature the destructive peripheral arthropathy noted in the previous reports. At the time of presentation her disease activity was subjectively moderate, with no morning stiffness and with lower back pain rated as 26% on a visual analogue scale. The modified Schober index was 5 cm, chest expansion 2 cm, and she had no peripheral synovitis or enthesitis. Erythrocyte sedimentation rate was 43 mm/1st h, C reactive protein 27 mg/l (normal <10). The patient was otherwise healthy, and IgA deficiency was not suspected on clinical grounds. During serological studies of a sample of patients with ankylosing spondylitis (to be reported separately) she was noted to have barely detectable circulating antibodies to IgA antibodies. Serum total IgA concentration was <0.2 g/l (normal range 0.8–3.9) by laser nephelometry, and <50 mg/l by enzyme linked immunosorbent assay (ELISA). By contrast, her serum IgG to IgD (0.5–3.5) and IgG slightly raised at 17.1 g/l (7.2–16.2). Bone marrow examination was not indicated.

Although selective IgA deficiency has been associated with recurrent mucosal infection and allergy, and with an increased incidence of several autoimmune disorders, most patients are asymptomatic.4 IgA deficiency affects about 0.2% of the adult white population,3 and ankylosing spondylitis up to 1%.4 By chance alone the two disorders will therefore occur together in 0.002% (one in 50 000 people), suggesting that there are around 1000 such patients in the United Kingdom. Unlike the four previously reported cases our patient did not have 'severe' ankylosing spondylitis, and we argue against interpreting IgA deficiency as a poor prognostic factor in ankylosing spondylitis until more evidence has been accumulated.

At a mechanistic level the existence of such cases indicates that an intact IgA response is not essential to the pathogenesis of ankylosing spondylitis.

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Ankylosing spondylitis and selective IgA deficiency.

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