

assumptions with respect to scale interpretation and statistical analyses.

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## Epidemiology of adult Still's disease

We read with interest the study by Magadur-Joly and colleagues about the epidemiology of adult Still's disease (ASD) published recently in *Annals*.<sup>1</sup> We would like to describe a patient who was significantly older than the age incidence mentioned in their study and posed considerable diagnostic difficulty.

A 66 year woman was referred urgently by her general practitioner with a two week history of sore throat, fever, weight loss, arthralgia/myalgia, and a rash. Apart from a history of hypertension she had previously been well.

When examined, she had a non-suppurative pharyngitis, a generalised polyarthritis with bilateral knee joint effusions and wrist and ankle joint synovitis bilaterally. She had an erythematous macular rash affecting her arms, legs, and trunk. She was pyrexial, with a temperature of 40°C. There was no obvious site of infection. Examination of the abdomen was normal; in particular, there was no hepatosplenomegaly. Investigation revealed erythrocyte sedimentation rate 123 mm/1st h, haemoglobin 12.5g%, leucocyte count  $28.6 \times 10^9/l$  (differential count, 94% polymorphs), mildly increased concentrations of urea and creatinine (16.6 mmol/l and 169  $\mu\text{mol/l}$ , respectively), and normal liver function. Throat swab, blood cultures, and midstream urine were negative. Antistreptolysin O was normal. Echocardiography, electrocardiogram, and chest radiograph were normal. She was negative for rheumatoid factor, antinuclear factor and antineutrophil cytoplasmic antibodies on two occasions. Serum ferritin concentration was 17 952  $\mu\text{g/l}$  (normal range 15-200).

Over the next two weeks, the patient remained pyrexial, with her temperature peaking consistently in the evenings with recrudescence of her rash. Her joint symptoms initially responded to non-steroidal anti-inflammatory agents, but she continued to be pyrexial. Her temperature persisted despite administration of aspirin, and she was prescribed prednisolone enteric 60 mg/day, with resolution of the rash and pyrexia.

A diagnosis of ASD was made on the basis of the clinical features and exclusion of other pathology. She was discharged home, taking steroids and aspirin, and on subsequent review in clinic was greatly improved, with almost complete resolution of her rash and fever.

This patient illustrates several important clinical points, not stressed in the article

published by Magadur-Joly and colleagues, but important for any future prospective studies on incidence. First, the diagnosis of ASD is often very difficult to make. Second, it is a diagnosis of exclusion. Third, given the rarity of the condition, diagnosis may be further delayed in patients of this age, though there are reports of ASD in this age group.<sup>2,3</sup>

Furthermore, the study by Magadur-Joly's group gave an approximation of the incidence of this disease in a French population obtained by written survey. We suspect that many cases of ASD are mild and go undiagnosed. Identification of such cases would therefore be difficult in any prospective study on ASD. Clearly, what is required is greater awareness of the disease and further research into its pathogenesis, in addition to prospective studies on incidence.

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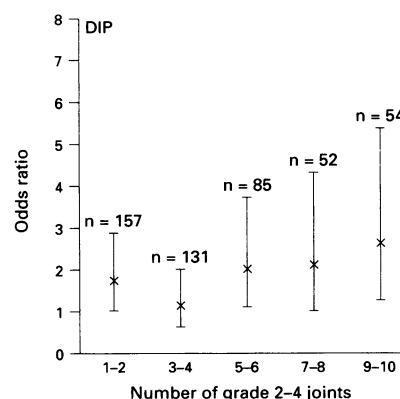
### AUTHORS' REPLY:

Drs Dunne and Davies reported a case of adult Still's disease in an elderly patient and emphasised the difficulties of diagnosis, particularly in this age group. We share their opinion as to the difficulty of making a diagnosis of ASD, which is the reason why we used the criteria of Ohta *et al* (sensitivity 96%; specificity 92%)<sup>1</sup> in all but one case. Nevertheless, in our experience diagnosis does not seem any more difficult in elderly patients: in our study we identified two patients who were 61 and 62 years old.

Drs Dunne and Davies suspect many cases of ASD to be mild. In our study we did not distinguish between mild ASD or full ASD, but required the symptoms to fit with the criteria of Ohta *et al*.

In conclusion, we agree that what is required is greater awareness of this disease, and we believe that making studies of its incidence is one approach to achieving progress towards that aim.

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# LETTER TO THE EDITOR

## Racial variation in rheumatoid arthritis

MacGregor and colleagues<sup>1</sup> reported a lower prevalence of rheumatoid arthritis in black Caribbeans than in white subjects in Manchester. There are grounds for supposing that one cause of RA is low testosterone levels.<sup>2</sup> It has been reported that in the USA, black males have significantly higher testosterone levels than white males.<sup>3-5</sup> A similar difference has been reported in the USA between (pregnant) black and white females.<sup>6</sup> It seems likely that part of this racial variation of testosterone levels is not genetic.<sup>7</sup> Meanwhile, it would be interesting to know whether there is racial variation in testosterone level in this country.

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## Correction

### Association of hand and knee osteoarthritis: evidence for a polyarticular disease subset

Hirsch *et al* (*Ann Rheum Dis* 1996; 55: 25-29)

The publishers and typesetters apologise to the authors for errors that led to publication of a version of figure 3 that omitted point estimates shown in the original (below).

