diabetes mellitus and hypertension, inactive lifestyle at a nursing home with chronic osteoarthritis and minimal persistent left pleural disease of over one year's duration and of uncertain cause. The patient had also been treated for herpes zoster infection about one year before the development of nocardial arthritis. Possibly, therefore, he might have had a subnormal immune defence mechanism against opportunistic infections. The patient's chest radiograph showed marked improvement in pleural disease after treatment of the arthritis with trimethoprim-sulphamethoxazole/amoxycillin-clavulanate. This suggests that the portal of entry was the respiratory tract, and the patient might have had chronic pleural disease with culture negative nocardial infection.

Successful treatment of nocardial septic arthritis seem to require prolonged antibiotic treatment for three to six months or longer.[5] From among all the antibiotics tested in vivo and in vitro, the use of trimethoprim-sulphamethoxazole in the treatment of human nocardiosis has been well established.[5 6] There is some evidence to suggest that amoxycillin-clavulanate may be useful in the treatment of nocardial infections.[7]

In summary, septic arthritis due to *Nocardia caviae* occurred in an elderly patient with multiple underlying diseases and was successfully treated with trimethoprim-sulphamethoxazole/amoxycillin-clavulanate combination antibiotic regimen. To our knowledge, *N caviae* arthritis has not been previously reported. Nocardial infection must be considered when undiagnosed, longstanding pleuropulmonary disease persists in an immunocompromised patient who later develops septic arthritis. Finally, although it requires the long term use of antibiotics, septic arthritis due to *N caviae* can be treated successfully.

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1 DiVittorio G, Carpenter J T Jr, Bennett J C. Arthritis in systemic nocardiosis. *South Med J* 1982; **75**: 507–8.
2 Wilkerson R D, Taylor D C, Opal S M, Curl W W. Nocardia asteroides sepsis of the knee. *Clin Orthop* 1985; **197**: 206–8.
3 Rao K V, O'Brien T J, Anderson R C. Septic arthritis due to Nocardia asteroides after successful kidney transplantation. *Arthritis Rheum* 1981; **24**: 99–101.
4 Cons F, Trevino A, Lavalle C. Septic arthritis due to Nocardia brasiliensis (letter). *J Rheumatol* 1985; **12**: 1019–21.
5 Curry W A. Human nocardiosis, a clinical review with selected case reports. *Arch Intern Med* 1980; **140**: 818–26.
6 Adams H G, Beeler B A, Wann L S, Chin C K, Brooks G F. Synergistic action of trimethoprim and sulphamethoxazole for Nocardia asteroides: efficacious therapy in five patients. *Am J Med Sci* 1984; **287**: 8–12.
7 Kitzis M D, Gutmann L, Acar J F. In-vitro susceptibility of Nocardia asteroides to 21 B-lactam antibiotics, in combination with three B-lactamase inhibitors, and its relationship to the B-lactamase content. *J Antimicrob Chemother* 1985; **15**: 23–30.

Sex ratios in HLA related autoimmune disease

Sir: With respect to the interesting article by James[1] we would like to describe data which support the hypothesis that sex ratios are disturbed in the families of probands with HLA related autoimmune disease.

Primary Sjögren's syndrome is a heterogeneous disease with a strong female preponderance.[2] A large family study of the disease has been completed in north east England and confirmed the association between HLA-DR3 and primary Sjögren's syndrome.[3] The families were ascertained from 40 white probands who all satisfied Fox's criteria for definite primary Sjögren's syndrome.[4] Two hundred and seven relatives from a potential pool of 224 were included. The table shows the female:male sex ratio among the family members.

Female:male sex ratio among family members of patients with primary Sjögren's syndrome

	Women	*Men*	χ^2 *or Fisher's test*	*p Value*
Index cases (n)	38	2	20·3	<0·001
Total relatives* (n)	150	74	13·3	<0·001
Relatives in study† (n)	142	65	95·4	<0·001
Siblings in study (n)	49	27	3·25	<0·1
Offspring of probands (n)	19	22	0·11	<0·1

*Refers to the potential pool of relatives.
†Refers to the relatives actually seen.

There was a trend for a female excess in siblings, though this did not reach statistical significance. A significant female preponderance, however, was seen in index cases and for the relatives as a whole. The availability of all the family pedigrees shows this to be a true phenomenon and not merely attributable to those consenting to the study.

We have reported definite/probable primary Sjögren's syndrome occurring exclusively in the female relatives with a strong association with HLA-DR3 (5/8 (63%)).[3] In addition, a cohort of young relatives (under 45 years) who expressed some features consistent with primary Sjögren's syndrome were also identified. Twenty eight of these 45 subjects were female (female:male ratio 1·65:1, $\chi^2=1·365$, NS). For these women there was a strong association with HLA-DR3 (18/28 (64%)) compared with the association for the men (5/17 (29%), $\chi^2=3·85$, $p<0·05$). We suggested that these women may be at risk of developing definite primary Sjögren's syndrome in the future, and a prospective study will help to support or refute this hypothesis.

This suggests that the inheritance of HLA-DR3 may influence the sex ratio of the offspring as well as the susceptibility to primary Sjögren's syndrome.

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1 James W H. Sex ratios and hormones in HLA related rheumatic diseases. *Ann Rheum Dis* 1991; **50**: 401–4.
2 Strand V, Talal N. Advances in the diagnosis and concept of Sjögren's syndrome (autoimmune exocrinopathy). *Bull Rheum Dis* 1979; **30**: 1046–76.
3 Foster H, Walker D, Charles P, Kelly C, Cavanagh G, Griffiths I. Association of DR3 with susceptibility to and severity of primary Sjögren's syndrome in a family study. *Br J Rheumatol*. In press.
4 Fox R I, Robinson C, Curd J, Cozin F, Howell F V. Sjögren's syndrome: proposed criteria for rheumatoid arthritis. *Arthritis Rheum* 1986; **29**: 577–85.

Nanocolloid scintigraphy for rheumatic diseases of the hands

Sir: The importance of early diagnosis of inflammatory rheumatic diseases has been emphasised recently. Nevertheless, the medical history and the physical examination of patients are difficult to assess, and laboratory and radiological modifications are often delayed. For several years scintigraphy has been proposed as a helpful device for diagnosis of initial or atypical bone and joint diseases. Most studies were performed with technetium-99m (^{99m}Tc) bisphosphonates, but other isotopes are now available.[1] Among these, ^{99m}Tc labelled nanocolloid, which spreads to the extravascular space at sites of inflammation with increased vascular permeability[2] is under study. As its clinical usefulness is still a matter for debate we compared joint pain, joint swelling, radiographs, and a palmar view scintigraphy (45 minutes after intravenous injection of 925 MBq of ^{99m}Tc nanocolloid; 200 000 counts or 15 minutes) of the hands of 27 subjects (seven controls, five patients with nodular osteoarthrosis, and 15 with classical or definite rheumatoid arthritis (RA)) who were examined in our department. The wrist, the metacarpal joints, the interphalangeal joint of the thumb, and the second to fifth proximal interphalangeal joints of each hand were included in the study, giving a total of 594 joints.

Joint pain and swelling, radiographic abnormalities, and nanocolloid scintigraphy were estimated on a four point scale: 0 representing a normal and 3 a very painful or swollen joint, a severe radiological change, or a marked increase of the isotopic uptake. The clinical assessment was made just after radiography and scintigraphy by an observer who was unaware of the results of the previous investigations. Six patients with RA also underwent ^{99m}Tc diphosphonopropane-dicarboxylic acid scintigraphy on adjoining days.

Joint isotopic uptake was usually easily discernible, but interpretation was difficult in several subjects owing to diffuse longitudinal uptake (fig 1) attributed to tenosynovial accumulation in three of seven controls, three of five patients with nodular osteoarthrosis (without tenosynovitis), and four of 15 patients

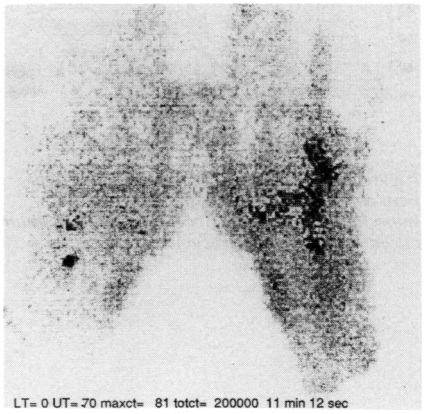

Figure 1 Technetium-99m labelled nanocolloid scintigram. Accumulation in the joints and along the tenosynovial sheath of the fourth left finger.