distribution in these peoples is still unknown, although casual observation suggests that there may be significant differences between Africans and Caucasians, and possibly even between different populations within the African group.

In the present study two epidemiological surveys were carried out, one in a rural, semitribal African population and the other in a fully urbanized African community in Johannesburg. The diagnosis of rheumatoid arthritis was based on the Rome criteria using the three parameters of clinical polyarthritis, radiological evidence of erosive arthritis, and positive serological tests for rheumatoid factor.

In the rural African population the prevalence of 'definite' rheumatoid arthritis was 0.12%; and of 'definite' and 'probable' rheumatoid arthritis combined, 0.87% of those over 15 years. Such clinical and radiological changes as occurred were invariably mild and no one in the entire survey had features characteristically associated with established rheumatoid arthritis.

In marked contrast, the prevalence of inflammatory polyarthritis in the urban Africans was similar to that in Caucasian populations: 'definite' rheumatoid arthritis occurred in 0.9% and 'definite' and 'probable' rheumatoid arthritis combined in 3.3% of those over 15 years. The difference between the two African populations was statistically highly significant ($P=0.01$). Moreover, the form and severity of the clinical and radiological features in the urban community closely resembled those seen in Caucasians; 24 respondents (4.4%) had clinical polyarthritis, 2 with the classical changes of long-standing rheumatoid arthritis, and 25 (4.5%) had radiological signs of erosive arthritis. In both African groups a positive latex fixation test was recorded in over 10% of the population tested. No obvious cause for this was found.

The marked intraracial differences in disease pattern shown point to the importance of sociological and environmental factors in the pathogenesis of rheumatoid arthritis.

Rheumatoid Synovitis and Joint Disease. The Relationship Between Arthroscopic and Histological Changes. D. B. Yates and J. T. Scott (*Charing Cross Hospital, London W6*). Published in full in the *Annals*, 1975, **34**, 1.

Raised Plasma Kininogen Levels in Rheumatoid Arthritis. Response to Indomethacin and Relationship to Clinical Indices. J. N. Sharma, I. J. Zeitlin, P. M. Brooks, and W. C. Dick (*Department of Physiology and Pharmacology, University of Strathclyde, Glasgow; and Centre for Rheumatic Diseases and University Department of Medicine, Royal Infirmary, Glasgow*)

All previous work suggests that plasma kininogen concentrations in patients with inflammatory seropositive rheumatoid arthritis fall within the normal range (Jasani, Katori, and Lewis, 1969). These results were obtained from patients who were receiving anti-inflammatory drugs.

Plasma kininogen levels were measured by the method of Brocklehurst and Zeitlen (1967) in 7 patients with classical rheumatoid arthritis after withdrawal of all therapy for 48 hours, and again after one week of indomethacin therapy. The levels for plasma kininogen ob-

tained after withdrawal of therapy were markedly raised and fell after a week of indomethacin therapy but were still above the levels observed in normal subjects ($n=7$) and in patients convalescing in an orthopaedic ward with fractures (Table I). The reduction in plasma kininogen values was mirrored by a rise in indomethacin levels and a fall in the articular index of joint tenderness, pain index, and grip strength in these same patients (Table II).

Table I

	Plasma kininogen level (Bradykinin equivalents/ml) (mean $\pm$ SD)
Rheumatoid arthritis ($n=7$)	
Pre-indomethacin	12.9 $\pm$ 1.2
Post-indomethacin	7.2 $\pm$ 1.0
Fracture patients ($n=5$)	4.9 $\pm$ 1.2
Normals ($n=3$)	
Pre-indomethacin	5.0 $\pm$ 1.2
Post-indomethacin	5.0 $\pm$ 1.2

Table II

	Pre-indomethacin	Post-indomethacin
Articular index (mean $\pm$ SEM)	16.6 $\pm$ 9.3	11.9 $\pm$ 4.2
Pain index (mean $\pm$ SEM)	3.3 $\pm$ 0.7	1.9 $\pm$ 0.4

Time course studies in a further 3 patients with rheumatoid arthritis in whom all drugs were withheld for 48 hours and indomethacin (150 mg/d) was administered for 7 days confirm these findings and show that the fall in plasma kininogen occurs within 4 hours of drug administration. Indomethacin will not produce a fall in kininogen values in normal subjects nor in fracture patients and does not directly affect the bioassay. We have shown that the plasma kininogen concentration in patients with untreated rheumatoid arthritis is raised and falls with the use of indomethacin. The interaction and inter-relationships between anti-inflammatory drugs and prostaglandin and kinin, chemical mediators of inflammation, requires reconsideration. Whereas it is recognized that indomethacin will inhibit the prostaglandin synthetase enzymes, this is the first report which documents an effect upon the kinin system.

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