

increase haemoglobin synthesis and erythropoiesis. Further clinical and fundamental research is warranted to establish the possible beneficial effects of (oral) iron chelation treatment on RA activity and the anaemia of chronic disease.

G VREUGDENHIL
Department of Internal Medicine
ZuidertZiekenhuis
Rotterdam, The Netherlands

A J G SWAAK
Department of Rheumatology
Dr Daniel den Hoed Cancer Center
Rotterdam, The Netherlands

C DE JEU-JASPARS
H G VAN EIJK
Department of Chemical Pathology
Erasmus University
Rotterdam, The Netherlands

Correspondence to: Dr A J G Swaak, Dr Daniel den Hoed Clinic, Department of Rheumatology, Groene Hilledijk 301, 3075 EA Rotterdam, The Netherlands.

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Trauma and seronegative spondyloarthropathy

Sir: We have always been intrigued by the topic of trauma and seronegative spondyloarthropathy because of its many implications. Recent reports in the *Annals*¹⁻⁶ have been particularly stimulating and shown how much work is still needed in this area. We decided to

Occurrence of trauma before arthritis onset and B27 positivity distribution among 209 patients with seronegative spondyloarthropathy

	Patients		
	B27+	B27-	Total
With trauma	3	10	13
Without trauma	68	128	196
Total	71	138	209

Fisher's exact test: two tailed $p=0.55$; uncorrected $\chi^2=0.73$; $p=0.39$.

contribute to the subject by considering the following questions: (1) Does a trauma immediately precede the onset of seronegative spondyloarthropathy? (2) Does a trauma immediately precede the onset of a peripheral arthritis in a patient with seronegative spondyloarthropathy? (3) Is there an association between trauma, onset of arthritis, and HLA-B27?

We studied 209 patients affected by different forms of seronegative spondyloarthropathy: 138 with psoriatic arthritis, 49 with arthritis during ulcerative colitis, and 22 with ankylosing spondylitis. The prevalence of HLA-B27 was 34.0% (71 patients: 45 with psoriatic arthritis, six with ulcerative colitis, and 20 with ankylosing spondylitis). In two cases (1.0%) an articular trauma immediately preceded the onset of the seronegative spondyloarthropathy. In both cases the HLA-B27 phenotype was absent.

When we extended the definition of trauma to include every acute disorder that immediately preceded arthritis onset, even extra-articular disorders, 11 more patients were identified (5% of the total). Surgery had been carried out in five cases, spontaneous abortion had occurred in two, and thrombophlebitis, bilious colica, myocardial infarction, and phosphoric ester intoxication in one case each respectively. Three of these 11 patients had the HLA-B27 phenotype.

We then evaluated the possibility that HLA-B27 positivity was associated with the onset of arthritis following trauma. As shown in the table, no significant relation was found.

We then considered from among the total number of patients those with a peripheral arthritis (130 patients: 102 with psoriatic arthritis, 23 with ulcerative colitis, five with ankylosing spondylitis). The prevalence of HLA-B27 was 25.4% (33 patients: 27 with psoriatic arthritis, three with ulcerative colitis, three with ankylosing spondylitis). An articular trauma immediately preceded the onset of peripheral arthritis in three cases, all HLA-B27 negative (2.3%). When the extended definition of trauma (previously mentioned) was used 10 more subjects (7.7%) were included (the above mentioned cases with the exception of one who had had surgery). In three cases HLA-B27 was positive. Again, no significant association was found between the presence of HLA-B27 and peripheral arthritis onset after trauma (Fisher's exact test: two tailed $p=1.00$; uncorrected $\chi^2=0.04$; $p=0.84$).

Therefore on the basis of our results we can conclude that: (1) articular trauma can immediately precede onset of seronegative spondyloarthropathy, though the percentage of cases in which this occurs is small; (2) trauma defined as every acute disorder immediately preceding seronegative spondyloarthropathy onset is detectable in a higher

percentage of cases; (3) HLA-B27 does not seem to predispose to arthritis onset following trauma in patients with seronegative spondyloarthropathy.

RAFFAELE SCARPA
ANTONIO DEL PUENTE
CARLO DI GIROLAMO
GIROLAMO DELLA VALLE
GIOVANNI LARIZZA
ENNIO LUBRANO
PASUALE ORIENTE
Cattedra di Reumatologia
I^o Facoltà di Medicina
Via S Pansini 5
80131 Naples, Italy

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Habitual knuckle cracking and hand function

Sir: In a recent survey Castellanos and Axelrod evaluated 300 consecutive outpatients at Mount Carmel Mercy Hospital to determine whether habitual knuckle cracking is a risk factor for hand dysfunction.¹ They found no relation with osteoarthritis, but noted that 'knuckle crackers were more likely to have hand swelling and lower grip strength' and concluded that 'habitual knuckle cracking results in functional hand impairment'. I believe they have not established cause and effect in these interesting correlations.

Not everyone can crack their knuckles. Some do so with ease, whereas others are quite incapable of performing the feat. No one has determined how the joints of these groups differ. It is quite possible, for instance, that metacarpophalangeal joint laxity may both facilitate knuckle cracking and impair hand function. As this hypothesis implies that hand swelling and diminished grip occur secondary to articular structure rather than abuse, it may be that nervous citizens of Detroit can continue to crack their knuckles without fear of injury.

'Will cracking my knuckles hurt my hands?' remains a common gambit when a rheumatologist is identified as such among new acquaintances striving to make conversation. I still believe that the answer to this question is no, but perhaps it is time that we really found out.

PETER A SIMKIN
School of Medicine
Department of Medicine, RG-28
Division of Rheumatology
University of Washington
Seattle, Washington 98195
USA

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Habitual knuckle cracking and hand function.

P A Simkin

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