Temporomandibular Joint Disease in Ankylosing Spondylitis. C. Davidson, J. Wojtulewski, P. A. Bacon, and D. Winstock (Departments of Rheumatology, St. Bartholomew's Hospital and Westminster Hospital, London). Published in full in the Annals, 1975, 34, 87.

Adult Human Articular Cartilage in Organ Culture. Synthesis of Glycosaminoglycan, Effect of Hyperoxia, and Zonal Variation of Matrix Synthesis. R. K. Jacoby and M. I. V. Jayson (Department of Medicine, University of Bristol, Bristol Royal Infirmary, and Royal National Hospital for Rheumatic Diseases, Bath)

There has been considerable interest in the metabolism of cartilage proteoglycan in recent years. The study of living human articular cartilage should increase our understanding of some of the pathophysiological changes in osteoarthrosis.

Normal articular cartilage was obtained at arthroscopy; the culture technique of Dingle, Fell, and Lucy (1966) was used. 90 cartilage explants were maintained for up to 8 days in either 20% O₂ or 95% O₂. The percentage chondrocyte survival was determined. 50 explants were maintained in either of these two gases for 2 days in the presence of 35S (SO₄)²⁻ and the incorporation into sulphated glycosaminoglycan measured. The uptake of sulphate was related to the DNA and uronic acid content and dry weight. In 25 explants, radioautographs were prepared from horizontal cryostat sections which were grouped so that the cartilage was divided into three layers. This allowed us to relate the synthetic activity of the chondrocytes both qualitatively and quantitatively to the DNA and uronic acid of each layer.

Results

(1) Mean chondrocyte survival at 8 days: 91·0% in 20% O₂; 21·5% in 95% O₂. (2) Radioautographs showed little synthesis by the most superficial cells, in contrast to the deeper ones. (3) 16% of the sulphate uptake, 8% of the uronic acid, and 28% of the DNA were found in the top third of the explants. Glycosaminoglycan synthesis is less at the surface than in the deeper layers. (4) There was a significant correlation between sulphate uptake and DNA (P < 0·001) and sulphate uptake and uronic acid (P < 0·001) (sulphate uptake and dry weight not significant). (5) A significant drop in the sulphate uptake/kg uronic acid was seen (P < 0·01) at 95% O₂ compared to 20% O₂. Similarly sulphate uptake/kg DNA fell (P < 0·01). However, sulphate uptake/mg dry weight was borderline significant (0·05 < P > 0·01).

Reference


Complement Activation in Reiter’s Syndrome. D. R. Yates, R. N. Maini, J. T. Scott and J. C. Sloper (Departments of Rheumatology and Experimental Pathology, Charing Cross Hospital, London)

Infecitve agents have been invoked in the aetiology of Reiter’s syndrome, but its chronicity and multisystem involvement has led to the further hypothesis (JAMA, 1973) of immunological mediation. Two previous studies (Brandt and others, 1968; Kinsella and others, 1969) of synovial membrane from 3 patients with Reiter’s syndrome have shown deposits of IgG and IgM, but without complement deposition. Intracellular leucocyte inclusions in synovial fluid have been found in Reiter’s syndrome (Peltier and others, 1967) as well as in rheumatoid arthritis, but there is conflicting evidence (Pekin and others, 1967; Vaughan and others, 1968) concerning the depression in the synovial fluid: serum complement ratio usually associated with complement activation.

Eleven patients with Reiter’s syndrome have been studied: all had at least 2 of the triad of arthritis, urethritis, and ocular involvement, and 9 of the 11 were HLA-27 positive. Synovial membrane from actively involved knee joints was examined by immunofluorescence for IgG, IgM, IgA, and B₂C deposition. At the time of synovial biopsy, all synovial membranes were observed to be acutely inflamed, and in each case histology revealed an acute exudative phase of synovitis (Britton and others, 1970). Focal, lumpy interstitial deposits of B₂C were seen in the synovium and subjacent tissue of 7 patients, and intracellular granular deposits shown in 3 of these. With anti IgG and IgA, prominent intracellular staining was seen in 6 patients, and focal, lumpy deposits of immunoglobulin were shown interstitially in 5 cases. IgM staining, both intracellular and interstitial, was observed but was less marked than IgG or IgA. Intracellular IgG and IgA staining was most conspicuous in the three membranes which also showed intracellular granular deposits of B₂C. In 2 of these, true complexes of IgG–B₂C were shown by a differential fluorochrome technique. Complement levels in paired serum and synovial fluid samples were estimated in 8 patients. A significantly depressed synovial fluid: serum complement ratio was found in 4 of these. These results suggest complement activation in Reiter’s syndrome, and lend support to the concept of immunological mediation.

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*Ann Rheum Dis* 1975 34: 468
doi: 10.1136/ard.34.5.468-a

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