LEADERS
Fish oil, rheumatologists, and arthritis p 71
The recognition that Eskimos have a reduced incidence of rheumatoid arthritis and are great consumers of fish oil has inspired several studies of a possible connection between these two observations. Fish oil from fatty fish is rich in eicosanoids and these have the property of stabilising inflammatory cell membranes and hence producing an anti-inflammatory effect. Would that it were that simple, however, because in animal studies they may precipitate an adjuvant arthritis, and scientific study is now difficult as so many of our patients read the newspapers and as a result are already consuming large amounts of fish.

Epidemic reactive arthritis p 73
The study of reactive arthritis holds many fascinations, but one of the fundamental problems that remains to be solved is why most episodes clear up after varying periods but a few of the patients go on to develop chronic disease. The common association with both HLA-B27 and B7 indicates some sort of genetic predisposition, and several bacterial infections of the gut have been clearly identified as common triggers to reactive arthritis. The development of DNA probes will help us, perhaps, to identify bacterial antigens and provide further clues.

SCIENTIFIC PAPERS
Fish oil and rheumatoid arthritis p 76
This crossover study comparing coconut oil with fish oil supplements in patients with rheumatoid arthritis (RA) suggested that fish oil was superior in suppressing the clinical symptoms and signs in RA. The study was only over a 12 week period for each type of oil and it would be good to see an attempt at a longer trial. The authors are to be congratulated, however, for introducing science into an area reeking with anecdote.

Surface markers in RA p 81
This Australian paper reports a study of lymphocyte surface markers on peripheral blood and synovial T lymphocytes in RA, psoriatic arthritis, and Reiter's syndrome. Although expression of major histocompatibility class II antigens is seen in many forms of arthritis, it was noted to be greater on RA synovial T cells. Other surface markers were less evident. Does this mean prior activation of RA T cells by an unknown antigen, as the authors suggest?

Measurement of joint inflammation in RA and osteoarthritis (OA) p 88
Indium-111 scintigraphy purports to measure synovial inflammation. This study compared such scans in RA and severe OA. In the patients with RA there was a significant correlation between pain, swelling, and abnormality of the joint and a positive scan, while in OA the correlation was only with pain in the joint. Furthermore, the positivity of the scan was much more evident in RA. The authors point out, however, that as no synovial biopsies were carried out they cannot be sure that a positive scan necessarily implied inflammation in the joint.

Anaemia in RA p 93
Anaemia in RA is often far from simple and this paper showed that only one third of patients had just one type of anaemia. Folic acid and vitamin B12 deficiency may mask iron deficiency by increasing the mean cell haemoglobin and mean cell volume. Both disease activity and erythropoietin responsiveness are likely to be major factors in the causation of the anaemia of chronic disease.

Sulphur baths, RA, and the Dead Sea p 99
We now turn from the laboratory to consider treatment. Balneotherapy has been greatly used in the treatment of RA for many years, though some rheumatologists have been very sceptical about it. A controlled trial, however, showed that the effect of mud packs and sulphur hot baths in a Dead Sea spa conveyed a significant improvement in symptoms (but not laboratory measurements) for up to three months. Our management of RA is not so perfect that we can afford to ignore any form of treatment that can be shown to work.

Genetic studies of mononuclear cells in Felty's syndrome p 103
An examination of the phenotypic and genotypic characteristics of peripheral blood mononuclear cells in Felty's syndrome showed in one patient an increase of those with the phenotype CD3+ Leu-7+ CD16+ with a rearrangement of the T cell receptor B chain gene. The other patients showed a germline configuration of the T cell receptor B chain gene. The study suggested that patients with this form of RA are heterogeneous, with at least three different peripheral blood mononuclear cell phenotypic subsets.

Infective endocarditis, rheumatoid factor, and anticardiolipin antibodies p 107
In patients with infective endocarditis B cell production of rheumatoid factor, anticardiolipin antibody tests, and the Venereal Diseases Research Laboratory test seemed to be both separate and distinct. No thrombotic complications were noted, as has been observed before, where anticardiolipin antibody production is seen to occur with an infection. When these are increased in systemic lupus erythematosus complicated by valve lesions it is likely to be the underlying disease process that is responsible.

Anticardiolipin antibodies in systemic lupus erythematosus (SLE) p 109
In 100 patients with SLE about one third had either IgG or IgM anticardiolipin antibodies or both present. The study showed that IgG antibody was associated with thrombosis and thrombocytopenia and IgM antibody with haemolytic anaemia and neutropenia. The specificity and predictive value of the tests increased with the titre found and clearly their identification may help with the management of lupus patients’ problems.

Neuropsychiatric complications of SLE and anticardiolipin antibodies p 114
Examination of four patients with SLE and acute central nervous system complications showed infarct-like cerebral
lesions on nuclear magnetic resonance imaging. All four had anticardiolipin antibody present in the serum but not the cerebrospinal fluid, and the authors of this paper suggest that this preliminary study may indicate that the anticardiolipin antibody has a direct pathogenic role in the acute encephalopathy of lupus.

Cystic bone lesions in SLE p 118
Skeletal abnormalities are uncommon in SLE but this Finnish study suggests that in patients with an increased acute phase response bone cysts are more likely to be seen, and that this constitutes a subset of the disease. These bone lesions are different from those seen in RA, and such patients are more likely to have a deforming arthropathy of the hand and other complications.

Osteoblasts and synovial fluid inhibitor p 121
In the synovial fluid of patients with RA a polypeptide inhibitor of osteoblast proliferation is described. It has a molecular weight of 81,000 and seems to be unique. This may be a cause for the juxta-articular osteoporosis so commonly seen in this disease. This is the first description of this inhibitor.

Indomethacin and extracellular calcium p 125
Non-steroidal anti-inflammatory drugs may affect calcium absorption by the gut, and they are implicated also in a possible effect on the glomerular filtration rate and the renal excretion of calcium. This paper looks at calcium homeostasis in healthy volunteers treated with indomethacin. The only significant change noted was a decrease in calcium excretion, possibly owing to renal retention of calcium. The significance of this is as yet unexplained.

REVIEW ARTICLE

Heat shock proteins and inflammation p 128
From studies of the larvae of Drosophila busckii in 1962 to evaluation of physiological stress in all living organisms seems a long march in scientific progress, but from such unlikely beginnings has the understanding of heat shock proteins evolved. Second line drugs used in the management of RA probably affect the inducers of heat shock protein in humans so it behoves us to try and puzzle out the process. This comprehensive review advances our understanding, though sadly and inevitably with the comment that many questions remain unanswered.

EDITOR