GLOSSARY

QPCR
Quantitative polymerase chain reaction; a method for quantifying mRNA

LUMINA
LUpus in MInorities, NAture versus nurture

MEMBRANOUS GLOMERULONEPHRITIS
Inflammation of the glomerulus and changes in the glomerular capillary basement membrane leading to disruption of kidney function

increased, with more than a 400-fold increase compared with naive mice.

To study the effect of inhibition of IL-17 on the arthritic flares, polyclonal rabbit antibodies directed against murine IL-17 were injected 2 h before the arthritis flare-up. Anti-IL-17 antibodies significantly suppressed joint swelling and were more effective than blocking IL-1. The neutralizing IL-17 antibodies almost completely prevented exacerbation of synovitis, and clearly suppressed proteoglycan depletion of cartilage. The degree of bone erosion was also significantly suppressed, and osteoclast-like activity was decreased during anti-IL-17 treatment. From analysis of QPCR synovial biopsies, it was suggested that IL-17 acts upstream of tumor necrosis factor, IL-1β, receptor activator of NF-κB ligand, and chemokines.

The authors conclude that IL-17 blockade could be an interesting target in the treatment of antigen-specific relapses during rheumatoid arthritis.

Rachel Murphy

Original article Koenders MI *et al.* (2005) Blocking of interleukin-17 during reactivation of experimental arthritis prevents joint inflammation and bone erosion by decreasing RANKL and interleukin-1. *Am J Pathol* **167:** 141–149

Age is the strongest predictor of damage accrual and vascular events in women with systemic lupus erythematosus

Studies have suggested that lowered blood estrogen levels resulting from the female menopause decrease the activity of systemic lupus erythematosus (SLE); however, a recent investigation shows this not to be the case. In fact, age rather than menopausal status appears to predict damage accrual and vascular events in these patients.

Fernández *et al.* studied women from the multiethnic LUMINA cohort to investigate the association between menopausal status and a number of clinical and socioeconomic variables. At disease onset, 436 women were premenopausal and 82 postmenopausal. Premenopausal women were found to be at higher risk of renal involvement, severe proteinuria and MEMBRANOUS GLOMERULONEPHRITIS, and postmenopausal women of vascular arterial events and lipid abnormalities. Disease activity and other manifestations of disease were comparable between the groups. Damage accrual between baseline and last follow-up visit, however, was more frequent in postmenopausal women. Despite these results, on multivariate analysis age was found to independently contribute to damage accrual, renal involvement and vascular arterial events, suggesting that this, and not the menopause, is the main predictor of these events in women with SLE whose disease starts after menopause.

This study has limitations both in its classification of menopausal status and its gathering of data, but the results agree with those previously generated from the lumina cohort and the authors are confident in their validity. Their additional finding that Whites are more likely to contract SLE following the menopause than Hispanics and African Americans could warrant further investigation.

Pippa Murdie

Original article Fernández M *et al.* (2005) Systemic lupus erythematosus in a multiethnic US Cohort (LUMINA): XXI. Disease activity, damage accrual, and vascular events in pre- and postmenopausal women. *Arthritis Rheum* **52:** 1655–1664

Therapeutic target for spondyloarthropathies

Researchers from Belgium have suggested a potential target for treating spondyloarthropathies: bone morphogenetic proteins (BMPs). This strategy, which specifically targets bone and cartilage formation, could be an alternative or complementary approach to current immune-suppressing therapeutics for gaining control of spondyloarthropathies.

To test the hypothesis that BMP signaling has a direct role in joint ankylosis, Lories *et al*. performed a murine study transferring cDNA of the BMP antagonist, noggin, intramuscularly to male DBA/1 mice with ankylosing enthesitis. The results showed that compared with controls, mice with the *noggin* gene had reduced incidence and clinical severity of spontaneous arthritis, and reduced clinical disease progression in developed arthritis.

The investigators noted that *noggin* gene transfer affected local expression of other molecules that might be involved in ankylosing enthesitis, including proliferating cell nuclear antigen, the cartilage transcription factor SOX9, BMP2, BMP6, and tumor necrosis factor. Results from histopathology suggested