

specimens from 5 patients, 10 pg/ml PGE<sub>2</sub> was associated with reduced expression of matrix metalloproteinase (MMP)-13 and MMP-1, as well as downregulation of the pro-inflammatory cytokines interleukin-1 $\beta$  and tumor necrosis factor, and the marker of chondrocyte hypertrophy, COL10A1.

The authors conclude that PGE<sub>2</sub>, at concentrations many times lower than detected in inflamed OA cartilage, is chondroprotective and reduces collagen destruction. Furthermore, these results show that chondrocyte hypertrophy is linked to collagen cleavage, and normal chondrocyte activity is maintained by low PGE<sub>2</sub> concentrations.

**Original article** Tchetina EV *et al.* (2007) Prostaglandin PGE<sub>2</sub> at very low concentrations suppresses collagen cleavage in cultured human osteoarthritic articular cartilage: this involves a reduction in expression of proinflammatory genes, collagenases and COL10A1, a gene linked to chondrocyte hypertrophy. *Arthritis Res Ther* [doi:10.1186/ar2273]

### Risk factors for cartilage loss in knee osteoarthritis

Researchers from North America have investigated the risk factors associated with progression of knee osteoarthritis (OA) by using MRI to quantitatively assess the progressive loss of cartilage volume in different areas of the knee and studying the correlation of these changes with demographic, clinical, radiological, and structural variables. This longitudinal study included a subset of 107 patients with symptomatic knee OA selected from a larger trial assessing the effects of bisphosphonate therapy. Patients were evaluated at baseline and at 24 months with X-ray and MRI investigations of the knee.

The analysis revealed that the greatest percentage of cartilage volume loss over the 24-month period occurred in the medial condyle and plateau, followed by the lateral plateau and the trochlear area. In general, the central weight-bearing subregions were worst affected. The loss of cartilage at 24 months compared with baseline was significant for all subregions apart from the anterior and posterior portions of the lateral condyle and anterior portion of the tibial plateau ( $P < 0.0001$ ).

Patients at greatest risk of cartilage loss, particularly in the medial compartment, tended to be female, to have a higher BMI, and to have

a narrower joint space width. The most significant structural risk factors were severe medial meniscal tear or extrusion, and subchondral bone marrow hypersignal.

The authors conclude that their findings shed light on the natural history of knee OA, the evolution of symptoms, and the risk factors associated with cartilage loss.

**Original article** Pelletier JP *et al.* (2007) Risk factors associated with the loss of cartilage volume on weight-bearing areas in knee osteoarthritis patients assessed by quantitative magnetic resonance imaging: a longitudinal study. *Arthritis Res Ther* [doi:10.1186/ar2272]

### SmD1 and SmD3 peptide immunoassays are accurate for the diagnosis of SLE

Detection of autoantibodies found in patients with systemic lupus erythematosus (SLE), such as those directed against certain Smith antigens (Sm), is a useful means of diagnosis. Some assays currently used to detect anti-Sm antibodies, however, fail to differentiate patients with SLE from those with other autoimmune diseases. SmD is considered the most-SLE-specific Sm antigen; therefore, Mahler *et al.* evaluated the clinical accuracy of an SmD1 and an SmD3 peptide-based immunoassay as biomarkers for the diagnosis of SLE.

The two assays were tested on sera collected from patients with SLE ( $n = 48$ ) and controls (patients with other autoimmune diseases;  $n = 99$ ). After optimization of the cutoff value of the SmD1-peptide-based immunoassay, there was excellent agreement between the two assays; each assay had a sensitivity of 12.5% and a disease specificity of 100%. The concordance between the assays was confirmed in a second panel of sera selected on the basis of a positive anti-Sm test ( $n = 65$ ). The authors also assessed whether anti-dsDNA antibodies bridging via double stranded DNA (dsDNA) to the SmD1 peptide would affect the diagnostic accuracy of this assay. Although SmD1 had significantly higher binding properties for dsDNA than SmD3, this had no significant effect on the diagnostic accuracy of the SmD1-peptide-based immunoassay.

The authors suggest that previously reported differences in the sensitivities of the two assays probably resulted from the use of different cutoff values. Anti-SmD assays