

noggin blocked endochondral bone formation in ankylosing enthesitis. The investigators also performed tests on human enthesial biopsies, which showed that progenitor cell proliferation and cartilage formation were "strikingly similar" to that observed in the mouse model.

In conclusion, BMP signaling appears to be a key molecular pathway in the pathologic cascade of spondyloarthropathies, and BMPs and their transduction machinery are attractive targets for disease modification.

*Rachel Murphy*

**Original article** Lories LJU *et al.* (2005) Modulation of bone morphogenetic protein signaling inhibits the onset and progression of ankylosing enthesitis. *J Clin Invest* **115**: 1571–1579

## Links between bone mineral density, marrow perfusion and marrow fat content

Previous studies have shown age-related changes in perfusion and fat content of vertebral marrow and have suggested that reduced perfusion might shift the balance between osteoblastic and osteoclastic activity, thereby altering bone density. Using dual X-ray absorptiometry (DXA), MRI and spectroscopy, researchers in Hong Kong investigated a possible relationship between bone-marrow fat content, perfusion and bone mineral density (BMD).

This prospective study initially enrolled 90 men, and 82 were included in the final analyses (mean age 73 years, range 67–101 years). DXA data enabled assignment of patients to one of three groups—normal BMD ( $n=42$ ), OSTEOPENIA ( $n=23$ ), or OSTEOPOROSIS ( $n=17$ ). Imaging of the lumbar spine was performed, and mean values for vertebral marrow fat content and perfusion determined for each patient group.

Average vertebral marrow fat content was significantly higher in patients with osteoporosis ( $P=0.002$ ) or osteopenia ( $P=0.034$ ) than in men with normal BMD. In contrast, vertebral marrow perfusion was significantly reduced in osteopenic ( $P=0.023$ ) and osteoporotic ( $P<0.001$ ) men.

Despite limitations of a small sample size and an all-male patient population, Griffith *et al.* conclude that a reduction in BMD is accompanied by decreasing vertebral marrow perfusion and increasing marrow fat content. The authors

suggest further studies to determine whether these are related or independent variables.

*Rebecca Ireland*

**Original article** Griffith JF *et al.* (2005) Vertebral bone mineral density, marrow perfusion, and fat content in healthy men and men with osteoporosis: dynamic contrast-enhanced MR imaging and MR spectroscopy. *Radiology* **236**: 945–951

## GLOSSARY

### OSTEOPENIA

Decrease in bone mineral density of between 1.0 and 2.5 standard deviations as compared to peak bone density, T-score, or to an age-matched population Z-score

### OSTEOPOROSIS

Decrease in bone mineral density of 2.5 standard deviations as compared to peak bone density, T-score, or to an age-matched population Z-score

## Systemic lupus erythematosus and neuropsychiatric syndromes

Neuropsychiatric syndromes in patients with systemic lupus erythematosus (SLE) might be attributable to non-SLE factors, according to a recent study by Hanly and colleagues. Nervous system involvement is a common feature of SLE; however, few trials have addressed the extent of non-SLE-specific causes of these neuropsychiatric events.

The Canadian study compared the prevalence, diversity and clinical significance of neuropsychiatric syndromes in 53 patients with SLE and 53 patients with rheumatoid arthritis (RA). The demographic features of the two groups were similar.

The trial showed that the number of patients with a neuropsychiatric event was higher in the RA group than the SLE group. With regard to health-related quality of life, fatigue, depression and anxiety, and cognitive dysfunction, there were no significant differences between the two groups. More SLE patients with cumulative neuropsychiatric syndromes, experienced cognitive distress than did RA patients with cumulative neuropsychiatric syndromes, although this did not translate into a lower health-related quality of life.

The finding that anxiety, headache and mood disorders were comparable in both groups is interesting: it suggests that SLE patients are no more likely to experience these symptoms than patients with other chronic rheumatic diseases. The etiology of neuropsychiatric syndromes in patients with SLE is multifactorial, and correctly identifying the cause is a significant challenge. In conclusion, there is sufficient evidence to suggest that in a substantial proportion of patients, neuropsychiatric syndromes are not attributable to SLE.

*Rachel Murphy*

**Original article** Hanly JG *et al.* (2005) Neuropsychiatric syndromes in patients with systemic lupus erythematosus and rheumatoid arthritis. *J Rheumatol* **32**: 1459–1466