## **Evaluating strontium ranelate treatment for knee OA**

Strontium ranelate—which is approved for the treatment of postmenopausal osteoporosis—has substantial structuremodifying activity and slows the progression of knee osteoarthritis (OA), according to results from a 3-year randomized controlled trial in 1,371 patients with the disease.

Strontium ranelate therapy is known to slow radiological progression of spinal arthritis and improve back pain, and is thought to inhibit subchondral bone resorption and promote cartilage matrix formation. These effects prompted Jean-Yves Reginster and colleagues to evaluate its effect on the radiological and clinical progression of knee OA.

Patients were randomly allocated to one of three groups: strontium ranelate 1 g per day (n = 558); strontium ranelate 2 g per day (n = 558); or placebo (n = 559). Knees were assessed radiographically at baseline and annually during the study; 3-year change in joint-space width (JSW) was the primary endpoint. Secondary endpoints encompassed jointspace narrowing, changes in WOMAC (Western Ontario and McMaster Universities Osteoarthritis Index) and global knee pain scores, and urinary CTX-II levels.

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Notably, treatment with both doses of strontium ranelate was associated with smaller degradations in JSW than placebo (1 g per day:  $-0.23 \pm 0.56$  mm; 2 g per day:  $-0.27 \pm 0.63$  mm; placebo:  $-0.37 \pm 0.59$  mm; P < 0.001 and P = 0.018for 1 g per day and 2 g per day versus placebo, respectively). The 2 g per day regimen also resulted in significantly lower WOMAC score (P = 0.045) and pain subscore (P = 0.028) at 3 years, compared with placebo; these reductions were not significant for the 1g per day dose. Changes in urinary CTX-II measurements were also significant for both doses compared with placebo (1g per day, P=0.003; 2g per day, P=0.021).

The authors conclude that strontium ranelate therapy retards structural deterioration in patients with knee OA and, furthermore, that treatment with 2 g per day of the drug has a noticeable effect on symptoms. Although the exact mechanism of action is currently unknown, the authors speculate that the drug might exert its effects by influencing chondrocytes and bone cells through a number of aetiological pathways.

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