those without diabetes (adjusted 30-day MMR 1.91, 95% CI 1.40–2.61, $P<0.01$).

The authors conclude that patients with type 2 diabetes are at significantly increased risk of death from pneumonia, and that blood glucose is an important predictor of pneumonia outcome in all patients.

Original article Kornum JB *et al.* (2007) Type 2 diabetes and pneumonia outcomes: a population-based cohort study. *Diabetes Care* **30:** 2251–2257

Fluoride increases BMD but also increases risk of osteomalacia

Fluoride increases BMD, but does not seem to reduce fracture rates in patients with osteoporosis, possibly because fluoride alone has no effect on bone resorption. Antiresorptives are effective for fracture prevention; Reid *et al.* hypothesized that combining low-dose fluoride with an antiresorptive agent might improve the efficacy of fracture prevention treatment. In a randomized trial, the New Zealand research team added glutamine monofluorophosphate (MFP; 20 mg/day) or placebo to hormone replacement therapy with calcium supplementation in 80 postmenopausal women with osteoporosis.

After 4 years, BMD in the lumbar spine had increased by 22% in the MFP group, compared with 6% in the placebo group. In the femoral neck, BMD had increased by 4.6% in the MFP group, but decreased slightly in the placebo group. There were significantly fewer new vertebral fractures in the MFP group (hazard ratio 0.2 for MFP vs placebo). There were, however, more nonvertebral fractures in the MFP group than in the placebo group. Analysis of bone turnover markers showed significant stimulation of bone formation by MFP; however, MFP was associated with hyperosteoidosis in five of seven patients who provided bone biopsies.

The authors conclude that fluoride treatment has potent effects on bone formation, but causes unacceptable bone mineralization abnormalities at the 20 mg/day dose used in the study. They recommend further trials of lower doses of MFP.

Original article Reid IR *et al.* (2007) Addition of monofluorophosphate to estrogen therapy in postmenopausal osteoporosis: a randomized controlled trial. *J Clin Endocrinol Metab* **92:** 2446–2452

Antidepressants and traditional anticonvulsants are best for diabetic neuropathic pain

Patients with diabetic neuropathy often report neuropathic pain. Treatment for these patients involves tight glycemic control to slow the progression of diabetic neuropathy and the administration of analgesics, commonly antidepressants and anticonvulsants, to reduce the intensity of neuropathic pain. Wong *et al.* carried out a systematic review to assess the effectiveness of different analgesics in treating diabetic neuropathy in adults.

The authors searched scientific databases and identified randomized, controlled trials that compared topically applied or orally administered analgesics with placebo. From this search, 25 English language full-text articles were identified that met the inclusion criteria; after further exclusion on the basis of suitability of data for analysis, 17 studies were included in the meta-analysis of treatment efficacy. The primary outcome was an approximate 50% reduction in pain as a result of treatment. Tricylic antidepressants, traditional anticonvulsants and opioids were found to be better for short-term pain relief than were newer generation anticonvulsants, a selective serotonin reuptake inhibitor and a serotonin noradrenaline reuptake inhibitor.

The authors provide a treatment algorithm for painful diabetic neuropathy on the basis of their findings, but stress that the studies they reviewed involved only short-term treatment (<6 months). The review also highlighted the lack of, and need for, studies on the long-term effects of anticonvulsants and antidepressants, which are widely used to treat these patients.

Original article Wong MC *et al.* (2007) Effects of treatments for symptoms of painful diabetic neuropathy: systematic review. *BMJ* **335:** 87

Serum total inhibin assay shows good results for detecting epithelial ovarian cancer

Inhibins are produced by ovarian follicles in premenopausal women, but decrease to near-undetectable levels in postmenopausal women; some ovarian tumors, however, continue to produce these markers beyond the menopause. Tsigkou and colleagues, therefore,