withheld from patients who have risk factors for gout. Hypertension and cardiovascular morbidity should be evaluated when a patient presents with gout.

Original article Janssens HJEM *et al.* (2006) Gout, not induced by diuretics? A case–control study from primary care. *Ann Rheum Dis* **65:** 1080–1083

VDR polymorphisms might not predict osteoporotic fracture

After a multitude of studies, it has become clear that assessments of bone mineral density and known risk factors for osteoporosis are insufficient to identify individuals who are likely to sustain osteoporotic fractures. It has been postulated, therefore, that some of the unknown risk factors are genetic. Several polymorphisms in the vitamin D receptor gene, *VDR*, have been investigated for associations with fracture, but study results are inconsistent. Uitterlinden *et al.* have conducted a large-scale meta-analysis of studies from across the world, to investigate the effect on fracture incidence of four restriction-fragment-length polymorphisms in *VDR*: *Fok*I; *Bsm*I; *Apa*I; *Taq*I; and one *CDX2*-promoter polymorphism.

Data were gathered for 26,242 participants (18,405 women), of whom 6,067 had a history of fractures and 2,088 had vertebral fractures. Genotyping and analysis of data revealed that the *CDX2* polymorphism was associated with a reduced risk of fracture; however, the decrease in risk was modest (risk reduction 9%, 95% CI 0–18%, $P = 0.039$) and restricted to vertebral fractures. No other associations were found.

These results contradict previous findings of an association between these polymorphisms and bone mineral density, and do not support the existence of a relationship between *VDR* polymorphisms and the incidence of osteoporotic fracture. The authors caution that their finding of an association between the *CDX2* polymorphism and vertebral fracture might simply be a chance finding, because of the large number of analyses they performed.

Original article Uitterlinden AG *et al.* (2006) The association between common vitamin D receptor gene variations and osteoporosis: a participant-level meta-analysis. *Ann Intern Med* **145:** 255–264

Death from infection is under-reported in patients with RA

Infection is a frequent cause of death in patients with rheumatoid arthritis (RA), and a case–control autopsy study has now shown that a high proportion of patients with RA die from previously undetected infections.

The data for this study were obtained from the autopsy register of Kivelä Municipal Hospital, Helsinki, between 1952 and 1991. The data included requests for autopsy, autopsy reports, and information from death certificates. Patients were deemed to have RA if it was mentioned in the patient's medical records, autopsy request, or autopsy report (if supported by autopsy findings suggestive of RA). Records from 369 patients with RA and 371 age-matched and sex-matched control patients without RA were reviewed.

The results showed that more patients with RA died of infectious causes (36%) than patients without RA (26%). Respiratory infections, followed by pyelonephritis, were the most common infectious causes of death in both groups. On the autopsy request, infection was not recognized in 55% and 50% of RA and non-RA patients, respectively. The proportion of patients who died with an unrecognized infection did not change substantially over the 40-year study period.

This study shows that a high proportion of infections that precede death go unreported, especially in patients with RA, which indicates the difficulty of diagnosing such infections. Autopsies continue to be valuable in determining cause of death.

Original article Koivuniemi R *et al.* (2006) Infectious causes of death in patients with rheumatoid arthritis: an autopsy study. *Scand J Rheumatol* **35:** 273–276

New biomarker for atherosclerosis in SLE and RA

Atherosclerosis is a common comorbidity in systemic lupus erythematosus (SLE), although the reasons for this association remain unknown. It is difficult to predict which patients will develop atherosclerosis. McMahon *et al.* suggest that proinflammatory HDL (which does not protect LDL from oxidation as normal HDL does) might be a new biomarker of increased risk for atherosclerosis in patients with SLE and in patients with rheumatoid arthritis (RA).