remainder received estrogen or testosterone hormone-replacement therapy.

BMD was almost identical in female controls and women with GHD, despite markedly lower bone turnover in women with GHD than in female controls—estrogen replacement compensated for the bone losses caused by GHD. By contrast, men with GHD had markedly lower hip and spine BMD than either control men or women with GHD, and their bone turnover was similar to that of control men. Testosterone replacement did not normalize BMD in males.

Hitz and colleagues suggest that bone loss in males with GHD probably occurs before adulthood or happens slowly over many years. Compared with controls, men (but not women) with GHD consistently had slightly elevated parathyroid hormone levels, which would eventually lead to reduced BMD. They speculate that GHD might impair the bone-specific aromatase activity that results in reduced estrogen-dependent bone formation in men with GHD.

Original article Hitz MF *et al.* (2006) Bone mineral density in patients with growth hormone deficiency: does a gender difference exist? *Clin Endoclinol* **65:** [doi:10.1111/j.1365-2265.2006.02667.x]

Contraceptive pill decreases bone mass in young women

Previous studies raised concerns that oral contraceptives, which suppress endogenous estrogen production, might impair bone mineralization and increase fracture risk in young women. Hartard and colleagues accordingly assessed the effect of low-dose, monophasic, oral contraceptives on BMD in a cross-sectional study that involved 248 healthy women aged 18–24 years.

Examinations included measurement of volumetric BMD, bone mineral content (BMC) and bone geometry of the tibia by peripheral quantitative CT, and measurement of areal BMD of the femoral neck and lumbar spine by dual-energy X-ray absorptiometry. There was a correlation between contraceptive use and bone mass at all sites. In the 201 women who had ever used oral contraceptives, femoral-neck BMD, tibial cross-sectional area and tibial total BMC were significantly lower ($P<0.005$, $P<0.01$ and $P<0.05$, respectively) than in the 47 women who had never taken oral contraceptives. Further differences were observed that depended on the duration and time of initiation of contraceptive use. Women who started oral contraceptives within 3 years after menarche and had taken them for >2 years had 10% lower femoral-neck areal BMD, 7% lower distal-tibial BMC and 6% lower total BMC at the tibial shaft, compared with women who had never taken oral contraceptives.

The authors conclude that oral contraceptive use can potentially harm the skeletal development of young women; they call for long-term studies to investigate the pathogenic mechanism that underlies this finding.

Original article Hartard M *et al.* (2006) Detrimental effect of oral contraceptives on parameters of bone mass and geometry in a cohort of 248 young women. *Bone* [doi:10.1016/j.bone.2006.08.001]

Intensive insulin therapy reduces cortisol levels in critically ill patients

Previous studies have demonstrated that intensive insulin therapy improves survival in critically ill patients. It is also known that survival in critical illness is partly dependent on an appropriate cortisol response. Vanhorebeek and colleagues have now studied the effect of intensive insulin therapy on the cortisol response of patients who were critically ill for >5 days.

Patients were treated with either conventional insulin therapy (insulin was administered only when blood glucose levels exceeded 220 mg/dl and target glucose levels were between 180 and 200 mg/dl; $n=243$) or intensive insulin treatment (target glucose levels were between 80 and 110 mg/dl; $n=208$). In all patients, the levels of total serum cortisol, corticosteroid-binding globulin, cytokines and C-reactive protein were measured and free cortisol levels were calculated.

Intensive insulin therapy resulted in markedly lower levels of blood glucose and C-reactive protein, fewer severe clinical complications, and a higher survival rate, compared with conventional treatment. Total-cortisol and free-cortisol levels were only reduced by intensive insulin therapy in survivors. Serum cytokine and corticosteroid-binding globulin levels were not affected by either therapy. Hydrocortisone treatment (at a replacement dose) for presumed