The relationship between obesity and bone health is complex. The main outcome of low bone mineral deposition is a diagnosis of osteoporosis, a condition that generally affects older adults. Osteoporosis is diagnosed on the basis of low bone mineral density (BMD). In adults, low BMI is seen as a risk factor for low BMD and for fracture, while a high BMI is associated with higher BMD (De Laet et al., 2005). It is thought that this is a response to the greater mechanical load on the skeleton that is associated with excess weight, which will stimulate bone deposition (Ribot et al., 1994). However, obesity is not protective against fractures, which are the key clinical outcomes of osteoporosis. Ong et al. (2014) surveyed female patients over the age of 50, who were attending a fracture clinic, and found that although obese women were less likely to have a diagnosis of osteoporosis, they were more likely to suffer fractures.

The effects of excess adiposity upon the skeletal development of adolescents appear to be different to what are observed in adults, with higher BMI generally associated with lower bone mineralization and impaired growth. The main predictor of bone mass before puberty is lean mass, but beyond puberty, fat mass becomes more strongly associated with bone growth (Mosca et al., 2013). El Hage et al. (2013) reported a lower whole-body bone mineral content in obese boys compared to those of healthy weight. Distribution of body fat may also play a key role, with central adiposity being associated with impaired bone growth at weight-bearing sites (Laddu et al., 2013). Goulding and colleagues (2000) noted that obese children aged 3–19 had greater bone mineral content for their age, compared to those with lower body fat, but when bone mineral and area were expressed relative to body weight, they were lower. This suggests that there may be a mismatch between bone growth and rapidly increasing weight in obese children.

The mechanisms that link obesity to bone growth are still not fully understood and may be complex. Adiposity may disrupt the endocrine signals that drive bone growth and maturation and is known to impact upon production of growth hormone (Perotti et al., 2013) and sex hormones (Vandewalle et al., 2014). Bone is also responsive to adipokines (including leptin and adiponectin) and pro-inflammatory cytokines (tumour necrosis factor α) that are produced by adipose tissue, and these agents may impact upon activity of osteoblasts and osteoclasts, resulting in altered rates of bone deposition (Campos et al., 2013; Mosca et al., 2013).
Nutritional status
Vegetarian adolescents appear to be at greater risk of having inadequate intakes of a number of nutrients. The low digestibility of plant foods and the poor bioavailability of minerals from plant sources (particularly with high phytate intake) mean that intakes of protein, zinc and iron may be of concern in vegetarians (Amit, 2010). This is of major importance at a time of rapid growth. Plant foods are rich in phytic acid, which inhibits absorption of iron and calcium, and oxalates, which inhibit calcium uptake. The bioavailability of iron from a vegetarian diet is only 10% compared to 18% from a mixed diet (Hunt, 2003). An 80% increase in intake is therefore required to meet requirements (Amit, 2010), which is challenging without supplementation or careful dietary planning. Vegetarian girls are six times more likely than omnivores to have low haemoglobin concentrations and three times more likely to have reduced iron stores (Thane et al., 2003). Calcium uptake is poor from many plant sources, but some such as soya beans, broccoli and kale have relatively high bioavailability (Weaver et al., 1999). Inclusion of dairy produce alongside such foods in the vegetarian diet should maintain healthy calcium status. In the absence of dairy produce, the vegan adolescent will need to take supplements of calcium and vitamin B12 (Amit, 2010).

Overall health and health behaviours
Notwithstanding the previous concerns, the dietary intakes of vegetarian adolescents are considered healthier by virtue of their greater consumption of fruit and vegetables and complex carbohydrates. It is also reported that vegetarian adolescents are less likely to smoke, consume alcohol or use drugs (Robinson-O’Brien et al., 2009). In adolescence, vegetarianism may be associated with a number of negative health behaviours. Vegetarians are less likely than omnivores to be smokers, consume less alcohol and are less likely to be overweight and obese. However, there is a greater prevalence of underweight, and vegetarian girls are more likely to suffer mental health disturbances and require medication for depression (Baines et al., 2007). A vegetarian diet can be used as a cover to hide more serious restriction of the diet and disordered eating by both girls and boys (Martins et al., 1999). Forestell et al. (2012) reported that vegetarians have less food neophobia and experiment with a wider range of new food experiences than omnivores. However, their dietary choices were more motivated by weight control, and they were more likely to restrict their diet in an unhealthy manner. Vegetarian adolescents are also more likely to engage in binge eating with loss of control, supporting the idea that the vegetarian diet is a proxy for increased risk of disordered eating (Robinson-O’Brien et al., 2009).

Fertility and reproductive function
Vegetarian girls may exhibit a number of problems with reproductive function. Vegetarianism is associated with longer cycle length, greater prevalence of amenorrhea, anovulation and luteal phase defects. This may be partly explained by reduced body fatness and leptin secretion but may also relate to hypothalamic control over sex hormone secretion (Griffith and Omar, 2003). Vegetarians secrete lower levels of luteinizing hormone, even if cycles are regular. Diets rich in fibre and low in fat alter the profile of sex steroids that are synthesized within the ovary and increase cycle length (Goldin et al., 1994). Baines et al. (2007) also noted that vegetarians were less likely than omnivorous teenagers to use the contraceptive pill.
Adolescence is a critical phase in the development of the skeleton. Puberty is the main phase for the accrual of bone mineral under the influence of sex steroids, so lifestyle factors during this stage of life that may have a detrimental effect on bone growth may result in lower peak bone mass and future bone health. Adolescents as a group are most likely to initiate cigarette smoking, begin consuming alcohol and experiment with recreational drugs and other substances.

**Smoking**

There is a well-established relationship between smoking and bone health in adults, with a 30% greater lifetime fracture risk in smokers compared to non-smokers. Reports of lower bone mineral density are common, with the hip, spine and femoral neck all exhibiting lower BMD in smokers (Wong et al., 2007). The impact upon the skeleton during adolescence is less well documented, but Jones et al. (2004) found that girls who smoked were significantly more likely to suffer fractures before age 18 if they smoked (OR 1.43, 95% CI 1.05–1.95). Regular smoking was associated with lower BMD at the hip and femoral neck in the study of Dorn and colleagues (2011) and also slowed the accrual of bone at the hip between the ages of 13 and 19 (Dorn et al., 2013). Välimäki and colleagues (1994) reported that men aged 20–29 had lower bone mineral density if they had smoked in adolescence. Similar findings were noted in a comparison of 18–19 year-old men who smoked with non-smokers (Lorentzon et al., 2007). The mechanistic basis of these effects upon bone may lie in the metabolism of vitamin D and hence the availability of calcium for deposition of bone mineral. Serum 1,25 dihydroxycholecalciferol concentrations are lower in smokers compared to non-smokers, and this will inhibit intestinal uptake of calcium.

**Alcohol**

Alcohol has known effects upon the skeleton, specifically the inhibition of osteoblast activity, which slows bone healing and turnover. However, there is evidence that in adults the relationship between alcohol and BMD is J-shaped, with low-to-moderate intakes actually stimulating accrual of greater bone mass (Wosje and Kalkwarf, 2007). This benefit of alcohol appears to be absent, however, with chronically excessive consumption or episodic binge drinking. In adolescents, the impact of alcohol is not well documented. While there is evidence that alcohol consumption does not alter the rate of accrual of bone between the ages of 13 and 19 (Dorn et al., 2013), adolescents who are regular consumers of alcohol have lower BMD at the hip and femoral neck (Dorn et al., 2011). Korkor et al. (2009) found no significant effect of alcohol or smoking in a small cohort of US high school students.

**Other substances**

Adolescents are experimental by nature and can be drawn to use of recreational drugs and abuse of toxic substances. Dündaröz et al. (2002) found that a group of young people who abused solvents had lower bone mineral density than an age-matched control group, but could not dissect the possible confounding effects of cigarettes and alcohol. There are several reports that abuse of opioids is also associated with loss of bone (Milos et al., 2011). Although there are no reports in the literature of the effects of cannabis use on bone health, it is known that bone contains cannabinoid receptors. These allow the tissue to respond to endocannabinoids which regulate the activity of osteoclasts (Idris and Ralston, 2012). Blockade or genetic knockout of these receptors stimulates bone growth, so there is the possibility that use of cannabis during adolescence could impact negatively on future bone health.