Executive summary

Poor bone health has a devastating impact in the UK, both in terms of disease morbidity and mortality as well as financial costs. Smoking has long been acknowledged to be a risk factor for poor bone health as it affects the metabolism of hormones, body weight, vitamin D levels, calcium absorption, blood circulation and increases oxidative stress thus disrupting healthy bone resorption and formation, leading to osteoporosis. Consequently, smokers have a 25% increase in fracture risk and are nearly twice as likely to experience hip fractures. Smoking also delays bone healing following operations to repair fractures. However, stopping smoking has been shown to partially reverse the risk of suffering fractures, and smoking cessation is therefore advised in national guidelines for the prevention and treatment of osteoporosis.

Key points

1. Bone health – the scale of the problem

- The main disease associated with poor bone health, osteoporosis, is diagnosed in about 1.2% of the overall UK population\(^1\) but prevalence increases steeply with age: one in two women and one in five men over the age of 50 are affected by the disease.\(^2\)

- Osteoporosis is characterised by low bone mineral density (BMD) and deterioration of bone tissue, which leads to progressive bone fragility and causes over 230,000 fractures per year in the UK, primarily of the spine, hip, wrist, humerus and pelvis.\(^3\)

- Fractures greatly affect morbidity and mortality; up to a third die during the first year following a hip fracture\(^4\), another third require nursing home placements and less than a third will regain a normal level of physical function.\(^5\) Fractures also result in increased back pain, height loss and physical disability.\(^6\)

- The costs of fractures are high, currently totalling more than £1.7 billion per year in the UK for treating hip fractures alone.\(^7\)
2. Smoking and bone health

2.1 Primary effects

- Bone health is primarily determined by peak bone mass achieved (usually around 30 years of age) and the rate of bone loss in the succeeding years. While the former is largely dependent on untreatable factors such as genetics, the later is not only determined by non-modifiable causes like age but also by modifiable risk factors such as physical inactivity.

- Among treatable causes of osteoporosis, smoking has long been established as a contributing risk factor as it affects the balance of the naturally occurring processes of bone resorption and bone formation, resulting in low BMD as the amount resorbed is not fully replaced.

- Smoking is thought to cause low bone density through various pathways (see Figure 1): (1) Smoking has been linked to changes in hormone household, leading to a decrease in parathyroid hormone (thus reducing calcium absorption) and oestrogen levels as well as to an increase in the level of cortisol and adrenal androgens, changes that have been linked to an increased risk of osteoporosis; (2) Smoking reduces body mass, which is postulated to provide an osteogenic stimulus and is linked to higher BMD; (3) Smoking reduces the level of Vitamin D in the body, which is required for good bone health; (4) Smoking increases free radicals and oxidative stress which affects bone resorption; (5) Smokers are more likely to suffer from peripheral vascular disease, reducing blood supply to the bones; (6) As smokers are weaker, have poorer balance and impaired neuromuscular performance, smoking may also increase the risk of falls; (7) Finally, there may also exist direct toxic effects of many of the constituents in tobacco smoke on bone cells.

- Meta-analyses have attempted to estimate the effect of smoking on bone health. While estimates vary (see Figure 2), there is a significant effect of smoking on overall fracture risk – in particular for the hip, spine and heel bone. Overall, risk of any fracture is increased by about 25% in current smokers and for hip fractures risk is increased between 40–84% and there is an increase in risk to over a 100% in those over 85 years of age.
**Figure 1. Potential mechanisms of increased fracture risk among smokers.**
*Adapted from Wong et al, 2007.*

SHBG – sex hormone-binding globulin

### 2.2 Secondary effects

- As a consequence of osteoporosis, those affected often require surgery to deal with the complications of fractures. There is convincing evidence to suggest that smoking is linked to impaired bone healing.\(^{28}\) This is most likely due to its potential impact on cellular differentiation and compromised microcirculation, both required for fracture repair.\(^{29}\)
3. Smoking cessation and bone health

- While life-time risk of fractures is increased in ex-smokers compared with never smokers, this risk is lower than among current smokers (Figure 2). Several studies have shown that BMD is intermediate between current and never smokers suggesting that the effects of smoking may be partially reversible\textsuperscript{31,32} and there is a dose-response relationship for the amount in pack-years smoked and fracture risk\textsuperscript{33}.

- Longitudinal studies have shown that smoking cessation reduces bone loss\textsuperscript{34,35} and smoking cessation is therefore recommended in osteoporosis guidelines\textsuperscript{36}.

*Figure 2. Fracture risk associated with current and past smoking.*

A) Data from Vestergaard and Mosekilde, 2003\textsuperscript{37}  
B) Data from Kanis et al, 2005\textsuperscript{38}
References


