

38. Osteoporosis

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Summary

The incidence of osteoporosis related fractures has increased in the past decades (1). Accordingly, it is important to try to prevent osteoporosis and current preventative endeavours are focusing on a number of avoidable risk factors. One example is physical activity, a vital ingredient for reinforcing and maintaining bone tissue. The effects of physical activity on the bone tissue are most noticeable when the activity is of a weight-bearing nature, intensive and frequent, around 2–3 times a week. In addition to the effects on the skeleton, weight-bearing exercises will have a positive effect on fitness, muscle strength and coordination and this in turn leads to a reduced risk of fractures and an improved quality of life. Suitable activities include dancing, gymnastics, jogging, strength training, ball and racket sports, brisk walks and walking up and down stairs.

The purpose of the physical activity carried out by individuals with osteoporosis is not just to affect the bone tissue, but also to prevent falls and subsequent fractures. Balance, strength and coordination exercises are therefore a good complement to walking, for example. In the event of a vertebral compression with continued pain, the physical activity should also emphasize pain relief.

Disease definition

Osteoporosis is defined as a systematic skeletal disease characterised by low bone mass (BMD, bone mineral density) and microarchitectural deterioration of bone leading to greater bone fragility and a consequent increase in fracture risk (2).

Prevalence/Incidence

Osteoporosis is common in Sweden. Approximately one out of three women aged 70–79 is diagnosed with osteoporosis in the hip (3).

Cause

Depending on its cause, osteoporosis can be divided into two types:

- *Primary osteoporosis* is either postmenopausal or related to age or lifestyle factors such as a lack of physical activity, smoking, alcohol consumption and inadequate nutrition.
- *Secondary osteoporosis* is caused by certain diseases or as a result of medical treatments.

Risk factors

The risk factors of osteoporosis can be divided into unavoidable (family history, old age, female gender, early onset of menopause, personal history of fractures and height) and avoidable (smoking, physical inactivity, inadequate nutrition, low weight, poor health, secondary osteoporosis and medical treatment).

Pathophysiology

Bone tissue composition and renewal (remodelling)

The remodelling or renewal of bone tissue constitutes 25 per cent of trabecular and 2–3 per cent of cortical bone renewal each year. Bone is a very dynamic tissue that remodels constantly throughout life depending on external demands such as physical activity. This remodelling means that the osteoclasts, i.e. bone resorption cells, resorb bone from the bone surface while the osteoblasts, i.e. bone-forming cells, replace the loss of bone with new bone tissue (Figure 1).

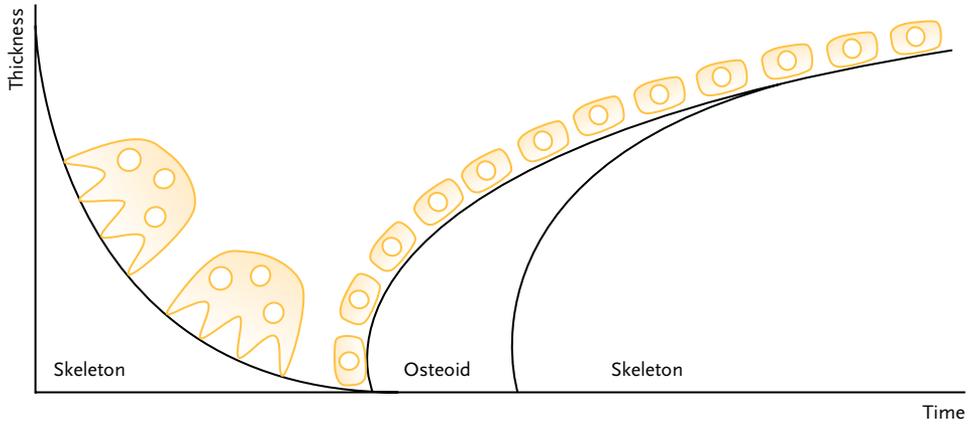


Figure 1. Schematic illustration of bone renewal (remodelling) with osteoclasts breaking down (resorbing) the bone (left in the picture) and osteoblasts producing new bone tissue (right in the picture). Modified according to Ericson (4).

During remodelling, some of the osteoblasts remain encased in bone matrix and are then called osteocytes. The osteocytes communicate with each other and the cells on the bone surface via long cell outgrowths that form a network of small channels. The osteocytes most likely play an important role as they detect and respond to the mechanical load put on the skeleton, thereby initiating the remodelling of relevant bone surfaces. Moreover, the mechanical load may be an important link between bone resorption and bone formation and lead to bone formation without preceding bone resorption (modelling). Hormones (systemic influence) and local growth factors are also likely to be involved in different levels in the remodelling process (Figure 2).

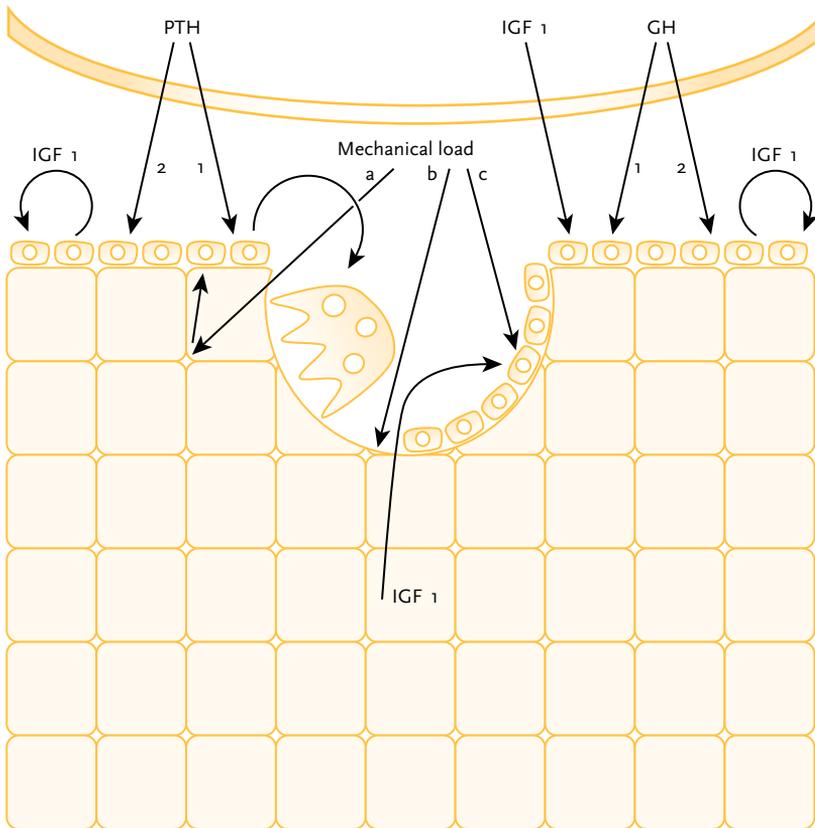


Figure 2. Schematic illustration of local and systemic influence on the bone cells. The mechanical load can intervene and affect various stages in the remodelling cycle (a–c) while endocrine, autocrine and paracrine regulators of the parathyroid hormone (PTH), growth hormone (GH) and insulin-like growth factor 1 (IGF-1) have the potential of modulating or supplementing the mechanical load response. Arrows 1 and 2 show direct and indirect effects of PTH and GH on bone cells. Modified according to Brahm (5).

It is vital for the resorbed bone to be replaced with the same amount of new bone or the bone renewal will be incomplete, resulting in a loss of bone. The loss of bone becomes particularly pronounced when the remodelling rate is high, such as during menopause.

Most common symptoms – what are the consequences of the disease?

Osteoporosis is a symptom-free disease, which only causes suffering in the event of a fracture. Approximately 50 per cent of women and 25 per cent of men aged 50 will at some point during the rest of their lives suffer a fracture because of osteoporosis (6).

Diagnosis

The diagnosis of osteoporosis is based on bone mineral density measurements (dual energy x-ray absorptiometry, DXA) and osteoporosis is defined by the World Health Organization (WHO) as a bone mineral density that is 2.5 standard deviations below the average bone mass of young adults of the same population (7).

Treatment principles

1. Lifestyle changes; physical activity, dietary habits and smoking.
2. Calcium and Vitamin D supplements.
3. Antiresorptive treatment; bisphosphonates, SERM.
4. Anabolic treatment; intermittent PTH.
5. Orthopaedic intervention; kyphoplasty/vertebroplasty (for vertebral compressions)

The effects of physical activity on bone resorption and renewal

A combination of factors are known to affect the dynamic bone tissue (see Figure 3).

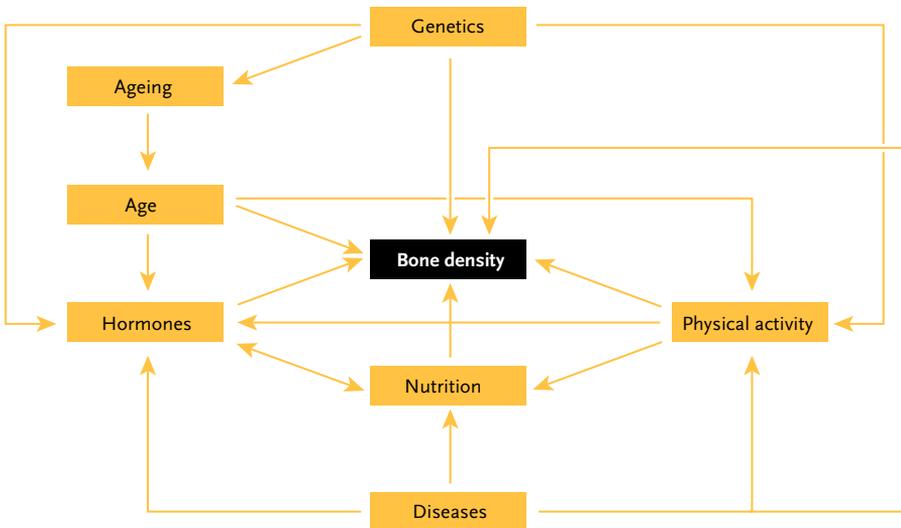


Figure 3. A summary of factors affecting the production and maintenance of bone tissue and the relationship between these factors. Modified according to Ziegler and colleagues (8).

An optimisation of these factors will aid the production, protection and maintenance of a healthy skeleton (9). It is not possible to influence heredity and age, but an improved nutritional intake (incorporating calcium and vitamin D), adequate levels of sex hormones (e.g. oestrogen substitutes during menopause) and increased physical activity can provide considerable benefits in the form of increased bone formation, for example.

In addition, physical activity affects the muscle mass and muscle strength whereupon the latter is closely connected to bone density. Hence, it is not unreasonable to see a strong link between these two factors, i.e. reduced muscle strength followed by reduced bone mass as a result of physical inactivity for example, even if the response from the bone tissue is somewhat delayed. Increased muscle strength would similarly precede an increase in bone mass, even though the load to which the bone is subjected is mainly muscle load and not weight load. Age-related bone loss is essentially linked to age-dependent changes in the muscle strength. However, the claims of numerous researchers that bone loss begins earlier in life than the loss of muscle strength contradict such a strong correlation. A number of studies also indicate that muscle strength is not just linked to the bone density of the “underlying” bone, but also to the bone density of the remainder of the skeleton without any relation to the muscle studied. Consequently, it is important to note that the effect of mechanical stimulus (physical activity) on bone tissue may also be dependent on the hormonal and metabolic environment, i.e. non-mechanical environment, with a potentially changing effect on the sensitivity of the bone cells.

Acute effects

Effects on bone density have been observed after 7–12 months of training (10–13).

Long-term effects

Like muscle strength, bone density is perishable and a change from a physically active life to a physically inactive life will result in a reduced bone density. However, there is evidence that athletes maintain a somewhat higher bone density than others when checked several years after their sports careers have finished (14).

Indications

Physical activity in order to affect the maximum bone mass – primary prevention

A maximum bone mass, known as peak bone mass, is achieved at 20–30 years of age following skeletal maturation throughout the adolescent years. The most important factors for an optimised maximum bone mass during this time in life are diet, hormonal status and weight-loading activity. Consequently, multifaceted physical education at school is likely to be of benefit to the skeleton, although it may be even more important that physical

education fosters continued physical activity in the generation growing up, whereby bone mass is maintained. The choice of lifestyle is therefore of utmost importance.

Prospective training studies of children between the ages of 6–10 with interventions such as jumping, aerobics, weight lifting and school gymnastics indicate an increase in bone density. The length of the training period varied from 7–24 months (10–13, 15). Prospective studies also show that self-chosen physical activities in the adolescent years have a positive effect on bone density (16–19). In addition, a large number of cross-sectional studies have compared the bone density of athletes with that of untrained individuals. These studies highlight the importance of weight-loading physical activity. Sports that involve rapid movements in different directions and/or a weight-bearing load have the greatest effect on bone density while an unloaded activity such as swimming has little or no effect. One study of the effect of loading activity compared the bone density in the dominant arm of female tennis players with the bone density in the non-dominant arm. The result of the study indicates that those who had started playing tennis before reaching puberty had a bone density side-difference of around 20 per cent while those who had started playing tennis after puberty had a difference of just under 10 per cent when comparing the dominant arm with the non-dominant arm (20). This may be seen as further evidence that regular physical activity in the primary school years is of greater benefit to the skeleton than the physical activity carried out in later years.

Physical activity in women between the ages of 20–50 (premenopausal)

A gradual loss of bone density takes place from around the age of 20, when the maximum bone mass is fully developed, until menopause. However, the results of activity studies carried out on women aged 30–50 are somewhat uncertain as to the effect of physical activity on aged-related bone loss. Still, most of the results indicate that weight-bearing activities (such as aerobics) are of the greatest significance – if not to increasing bone mass then at least to maintaining existing bone mass (21–24). A review article on the subject concluded that the quality of the relevant studies was relatively low. There is a large drop-out rate in the training studies, making it difficult to generalize the small positive effects that physical activity has on BMD (25).

Physical activity to reduce bone loss after menopause

Trabecular bone loss accelerates during and after menopause, but cortical bone is also affected. Cross-sectional and longitudinal studies show that the loss of bone taking place during this time in life cannot be prevented by physical activity, only slowed down.

The significance of walking, jogging, aerobics and strength training to the prevention of bone loss is not yet clear, although a number of randomised controlled studies indicate that such activities have a beneficial effect on bone tissue, either in the form of a slight increase or reduced resorption (26–28). Cross-sectional studies also indicate a lower incidence of fractures in postmenopausal women reported to be physically active (29, 30).

Physical activity in osteoporosis – secondary prevention

The effect of physical activity on the bone density of patients with osteoporosis is poorly documented. One study on women with osteopenia showed that 60 minutes of weight-bearing exercise three times a week for 12 months stabilised the BMD in the lumbar region, but did not have an effect on the hips (31). Home exercise programmes can result in an increased quality of life in patients with or without vertebral compression (32, 33). In addition, 10 weeks of physiotherapy incorporating balance exercises, strength training and lumbar stabilisation exercises lead to an improved daily functional capacity and quality of life with better balance and less pain (34). This indicates that the aim of physical activity need not be to increase bone density, but can be to improve balance in order to reduce the tendency to fall, resulting in a lower risk of fractures, less pain following a vertebral fractures and a better quality of life.

Prescription

General exercise advice based on what is known today

To obtain maximal effect on the skeleton, weight-bearing activities must be performed. The dynamic load put on the skeleton should consist of movements that compress, elongate, rotate and flex the skeleton. At a younger age, this would include activities such as jumping and running with quick starts and stops and changes in direction. This could be anything from normal play to an organised sports activity. Later in life, activities such as brisk walks, jogging, fitness training, aerobics, strength training or, if a more competitive form of exercises is preferred, ball and racket sports are recommended.

Even if walking in itself is not the activity that has the greatest positive effect on bone tissue, together with other daily activities (such as taking the stairs instead of the lift) it may nonetheless be of significance. Svenska Osteoporossällskapet [The Swedish Osteoporosis Society] recommends a brisk 30-minute walk five times a week as a general exercise to prevent osteoporosis, which should be feasible and practical for the population at large. However, this activity must be done on a regular basis as bone is a replenishable tissue.

Because osteoporosis is a risk factor for fractures, the objective of the physical activity is also to prevent falls and consequent fractures. Leg training exercises combined with balance and coordination training are recommended, such as in the form of dancing or Thai Chi.

The pain associated with a vertebral compression is often alleviated with lumbar stabilisation and mobility training which reduces muscle tension.

Functional mechanisms

The effect of physical activity on the skeleton

When bone tissue is subjected to a dynamic load, a temporary deformation and subsequent fluid flow induction takes place in the canalicular network surrounding the osteocytes. This is likely to have an effect on intracellular calcium levels and also on local osteoblasts and osteocytes. This effect increases with the size and speed of the load and whether the load has an unusual or variable direction.

Bone tissue responds immediately with a cellular reaction characterised by an acute local release of prostaglandins, which has been shown *in vivo* in humans (35). This leads to a local production of growth factors and subsequent bone renewal in response to the original load. However, a general ‘hormonal’ tissue reaction might also be expected as a consequence of the physical activity. Many of the hormones and growth factors known to affect the skeleton are influenced by physical activity such as growth hormone (GH), parathormone (PTH) and insulin-like growth factor 1 (IGF-1) among others (36). Perhaps the most crucial evidence of the skeleton health benefits gained through loading is the rapid bone resorption that takes place in the absence of weight-bearing load as seen during space missions and immobilisation (37–40).

The Mechanostat, *i.e.* the theoretic model describing the effect of mechanical load on bone tissue can be compared with a thermostat that limits load-related bone tissue deformations. A skeleton deformation is an indication that there is a risk of fracture whereupon the production of bone tissue starts and continues until the risk has been removed. Accordingly, the load put on the skeleton determines how much bone tissue is renewed and resorbed, and the individual in question only gets as much new bone tissue as he or she ‘deserves’.

Reduced muscle strength, balance and coordination constitute risk factors for falls. By training these functions, falls and, consequently, the risk of fractures can be prevented.

Functional tests/need for health check-ups

What tests should be carried out prior to training?

- Assess the DXA measurement to gain an understanding of how fragile the patient is before recommending a load.
- Test balance and muscle strength to assess the risk of falling and evaluate the effects of physical activity.
- Ask if the patient has a tendency to fall.
- Carry out a pain assessment.
- Test lumbar mobility and make an assessment of kyphosis.

How and when should the effects gained be assessed?

Increased bone density should be possible to measure with a DXA bone densitometry scan after 7–12 months.

Assess muscle strength, balance, pain, posture and the number of falls after a period of 2–6 months.

Interactions with drug therapy

Antiresorptive treatment with bisphosphonates have shown to be less effective on patients that are immobilised due to a spinal cord injury (41). Physical activity in combination with medication is recommended to achieve maximum effect.

Contraindications

Acute illness with pain and general deterioration.

Risks

General side-effects such as:

- Load injuries caused to the locomotor system if the intensity and frequency of physical activity is not gradually increased and incorrect footwear used.
- Risk of falling and increased risk of fracture.
- Weight loss due to insufficient nutritional intake.
- Anorexia nervosa with amenorrhea.

It is important that the exercise programme put together for an untrained individual or osteoporosis sufferer with a fragile skeleton incorporates activities that are enjoyable and can be performed with caution so as to avoid injury owing to an unusual load.

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