Anatomy and physiology of ageing 10: the musculoskeletal system

Key points
- The age-related degeneration of the musculoskeletal system makes older people prone to frailty, falls and fractures
- Sarcopenia is produced by the atrophy and shrinkage of skeletal muscles, coupled with a reduction in the speed and force of their contraction
- Osteoporosis and osteoarthritis commonly occur in old age as a result of bone changes
- To have a healthy musculoskeletal system, it is essential that older people keep as physically active as possible

Skeletal muscles allow the body to move and maintain posture; by contracting, they also aid the venous return of blood to the heart and generate heat that helps maintain body temperature. Bones support the body, protect vulnerable regions and allow physical movement via a system of levers and joints; they also store fat and minerals, and house the red bone marrow responsible for blood cell production. With age, these components of the musculoskeletal system progressively degenerate, which contributes to frailty and increases the risk of falls and fractures. Part 10 in our series on the anatomy and physiology of ageing explores the age-related changes that occur in skeletal muscles and bones.

Changes in skeletal muscles
Older people often experience a loss of strength that can be directly attributed to anatomical and physiological changes in skeletal muscles (Papa et al, 2017; Freemont and Hoyland, 2007) (Box 1). With age, skeletal muscles atrophy and decrease in mass (Fig 1), and the speed and force of their contraction reduce (Choi, 2016). This phenomenon, known as senile sarcopenia, is accompanied by a decrease in physical strength. Sarcopenia can impair the ability to perform everyday tasks such as rising from a chair, doing housework or washing oneself (Papa et al, 2017).

Maximal muscle mass and strength are reached in the 20s and 30s. This is followed by a gradual decline through middle age. From the age of 60, the loss of muscle tissue accelerates. In late old age, the limbs may lose so much muscle tissue that people with reduced mobility appear to be little more than skin and bone. Deep furrows may develop between the ribs because of intercostal muscle atrophy, while the loss of facial muscle tissue contributes to a general loosening of the features.

This considerable loss of muscle tissue often seen in later years (senile sarcopenia), is associated with increased frailty. While frailty is multifactorial, musculoskeletal deterioration and sarcopenia are central to it, and are both associated with increased...
weakness, fatigue and risk of adverse events such as falls, which can all increase morbidity (Fragala et al, 2015).

Skeletal muscles are composed of two main types of fibres:
- Slow-twitch fibres (type 1), used for endurance activities, such as walking long distances;
- Fast-twitch fibres (type 2), used in short 'explosive' activities such as sprinting.

Sarcopenia is associated with changes in the number and physiology of fast-twitch fibres, while slow-twitch fibres are relatively unaffected by age (Bougea et al, 2016). Indeed, recent studies show that slow-twitch fibres maintain and even increase the concentrations of some metabolic enzymes, perhaps to counteract the decrease in fast-twitch muscle fibre activity (Murgia et al, 2017).

Sarcopenia is also thought to be driven by the loss of motor neuron fibres (denervation) and loss and degeneration of neuromuscular junctions (the synapses connecting motor neurons to skeletal muscles); as a consequence, muscles are less stimulated and lose mass (Stokinger et al, 2017; Power et al, 2013).

Sarcopenia is exacerbated by the reduction in the levels of circulating anabolic hormones – such as somatotropin (growth hormone), testosterone and testosterone-like hormones – which decline from middle age onwards. As skeletal muscles are metabolically very active, sarcopenia is a major factor contributing to the age-related reduction in metabolic rate. On average, we lose 3.8% of lean muscle mass per decade from the age of 30, which compounds the decline in basal metabolic rate that starts from around the age of 20. If caloric intake stays the same as in younger years, there is a much greater risk that excess calories will be stored in the form of fat. This may be exacerbated in older people who are insulin-resistant, as their skeletal muscles are less able to take up glucose and the amino acids used to generate new muscle fibres (Cleasby et al, 2016; Fragala et al, 2015).

The loss of skeletal muscle mass leads to a progressive reduction in the support afforded to the bones and joints, which in turn contributes to the postural changes observed in older age (Fig 2). It also increases the risk of joint pathologies, particularly osteoarthritis, as well as the risk of falls and fractures.

Aged muscles are more prone to injury and take longer to repair and recover. This slower recovery may be due to a reduction in the number of progenitor (satellite) cells – undifferentiated stem cells that can develop into new muscle cells or myocytes – combined with progressive cellular senescence (Bougea et al, 2016).

Changes in bones
Bone mostly consists of:
- The inorganic component calcium phosphate (hydroxyapatite);
- The organic component type I collagen.

Calcium phosphate crystals form the bone matrix and give bones their rigidity. The skeleton acts as a calcium reservoir: it stores around 90% of all the calcium in the body (Lau and Adachi, 2011). Insufficient levels of calcium or vitamin D (essential for calcium absorption) can lead to a reduction in bone density and increase predisposition to osteoporosis and fractures. In older people, the gut absorbs less calcium and vitamin D levels tend to decrease, which reduces the amount of calcium available for the bones.

Collagen provides anchorage for the calcium phosphate crystals, knitting the bone together to prevent fractures. Some people have genes leading to faulty collagen production, which results in brittle bone disease (osteogenesis imperfecta).

Like muscle, bone is a dynamic tissue continuously being deposited and broken down. This state of flux is mediated by the two major bone cell types:
- Osteoblasts, which deposit bone;
- Osteoclasts, which digest bone, releasing ionic calcium into the blood.

Osteoblasts are more active when the bones are under the stress imposed by the weight of an upright, active body. In young mobile adults, osteoblasts and osteoclasts work at a similar rate and bone density is maintained. Inactivity means a decrease in osteoblast activity that ultimately results in reduced bone density (Nigam et al, 2009). The age-related loss of skeletal muscle mass contributes to the reductions in load (both weight and contractile force) on the bones, which compounds decalcification. It is therefore essential that older people keep as mobile and active as possible.

Changes to bone density
Studies (predominantly in the US) show that around 90% of peak bone mass is achieved in men by age 20 and women by age 18. Increases continue in both sexes until around the age of 30 when peak bone strength and density is achieved (National Institutes of Health, 2015). Bone density decreases as middle age approaches.

Women are at particular risk of bone demineralisation and osteoporosis as they gradually lose the osteo-protective effects of oestrogen pre and post menopause. In a 10-year study, women lost 1.5-2 times more bone mass per year from their forearms than men (Daly et al, 2013). Bone loss in both sexes continues into old age, and 80-year-olds have approximately half the bone mass they had at its peak in young adulthood (Lau and Adachi, 2011; Kloss and Gassner, 2006).

**Box 1. Age-related changes in skeletal muscles**
- Reduction in protein synthesis
- Reduction in size and number of muscle fibres, particularly in the lower limbs
- Decrease in the number of progenitor (satellite) cells
- Reduction in muscle growth
- Reduction in the ability of muscles to repair themselves
- Replacement of active muscle fibres by collagen-rich, non-contractile fibrous tissue
- Reduction in the number of motor neurons and deterioration of neuromuscular junctions
- Increase in fat deposition at the expense of lean muscle tissue
- Accumulation of lipofuscin (an age-related pigment)
- Reduction in the number of mitochondria (although not all studies are in agreement)
- Less-efficient metabolism, particularly in fast-twitch muscle fibres
- Reduction in blood flow to the major muscle groups
Osteoporosis
The age-related loss of calcium from the skeleton commonly leads to the bones taking on the porous, sponge-like appearance indicative of osteoporosis. There are two recognised forms of this:

- Type I, seen in menopausal and post-menopausal women and thought to occur as a result of falling oestrogen levels;
- Type II, referred to as senile osteoporosis, which affects both men and women and appears to be caused by reductions in the number and activity of osteoblasts. Additionally, some pro-inflammatory cytokines (whose numbers increase with age) such as interleukin 6 stimulate osteoclasts, leading to bone demineralisation (Lau and Adachi, 2011).

The vertebrae are particularly vulnerable to osteoporosis and may develop micro-fractures resulting in them collapsing under the weight of the body and becoming compressed and deformed. This contributes to the stooping curvature of the spine often seen in older age (Fig 2). Many factors contribute to age-related bone loss and senile osteoporosis (Box 2).

Risk of fracture
The age-related decrease in bone density is associated with an increased risk of fracture in many bones including the femur, ribs, vertebrae and bones of the upper arm and forearm. Osteoporosis is linked not only to a loss of inorganic mineral content, but also with a loss of collagen and changes to its structure. As collagen helps to hold bones together, this further increases the risk of fracture (Boskey and Coleman, 2010; Bailey, 2002).

The risk of fracture is compounded by a lack of mobility, for example, due to a prolonged stay in hospital (Nigam et al, 2009).

Box 2. Factors contributing to age-related bone loss and senile osteoporosis

- Reduction in testosterone levels in men and oestrogen levels in women
- Reduction in growth hormone levels (somatopause)
- Reduction in body weight
- Reduction in levels of physical activity
- Reduction in calcium absorption and vitamin D levels
- Increase in the levels of parathyroid hormone
- Smoking

Not only are fractures more common in old age, but healing takes much longer (Lau and Adachi, 2011).

Population studies in the US show that around 5% of adults over the age of 50 have osteoporosis affecting the femoral neck (neck of the femur) (Looker et al, 2012). This region is particularly vulnerable to fracture, as the two femoral necks support the weight of the upright body. Costache and Costache (2014) found that femoral neck fractures – which are serious and potentially life-threatening injuries – become more frequent after the age of 60 years and that women are more affected than men.

Joint changes
The articular cartilages in synovial joints play the role of shock absorbers, as well as ensuring the correct spacing and smooth gliding of bones during joint movement. The number and activity of chondrocytes, the cartilage-forming cells, decrease with age (Freemont and Hoyland, 2007), which can result in a reduction in the amount of cartilage in important joints, such as the knees (Hanna et al, 2005). A lack of cartilage results in aged joints becoming more susceptible to mechanical damage and increases the risk of painful bone-to-bone contact that is commonly seen in osteoarthritis.

Osteoarthritis
Osteoarthritis is the most common arthropy (joint pathology) in the world. Large-scale studies in the US have shown that around 10% of men and 13% of women over the age of 60 are affected by symptomatic osteoarthritis of the knee (Zhang and Jordan, 2010). In the UK, around 8.5 million people have joint pain due to osteoarthritis (National Institute for Health and Care Excellence, 2015). This places a great burden on health services as many patients will require expensive joint surgery, particularly to the knee, hip and lumbar spine.

The outer portion of a joint capsule is composed of elastic ligaments that bind the joint together, preventing dislocation while allowing free movement. With age, changes to the collagen and elastin components of ligaments decrease their elasticity (Freemont and Hoyland, 2007), resulting in stiffness and reduced mobility. Certain joints are particularly susceptible; for example, between the ages 55 and 85 years, women lose up to 50% of flexibility and range of motion in their ankles (Vandervoort et al, 1992). Although there are many risk factors associated with the disease (including genetic predisposition, gender, obesity and previous joint injury), age is by far the greatest.

Healthy musculoskeletal ageing
Many factors influence how our bones and skeletal muscles age; genetics, environmental factors and lifestyle all play a role, so there is much individual variation. Preserving the structural and functional integrity of the musculoskeletal system is
Dietary supplementation
Increasing the intake of calcium, vitamin D and lean protein can increase bone density and provide amino acids for muscle growth. This may offset the reduction in the efficiency of nutrient absorption seen in older age. We know that, in younger adults, increasing protein intake can enhance protein synthesis in skeletal muscles, but this seems to work less well in older people. Fragala et al (2015) found that dietary supplementation with creatine can increase muscle strength and performance, while the intake of protein drinks supplemented with the amino acid β-alanine increases muscle-working capacity and quality in older men and women.

Hormone replacement therapy
Hormone replacement therapy (HRT) improves bone health in older people: oestrogen (HRT) (with either oestrogen or oestrogen plus progesterone) does not have the same anabolic effect (Fragala et al, 2015). Women can use TRT, but they may be reluctant to do so because of unwanted effects such as facial and body hair growth and deepening of the voice.

Exercise
Unless regularly used and placed under load, muscle fibres and neuromuscular junctions degenerate, resulting in disuse atrophy (Kwan, 2013). Moderate exercise helps to maintain lean muscle mass, increase bone density and reduce fat accumulation. Exercise also increases the number of mitochondria in muscle fibres, enhancing energy release, metabolism and muscle power. In people who remain physically active, the efficiency of mitochondria in releasing energy appears to be maintained until at least the age of 75 (Cartee et al, 2016).

Progressive resistance training is considered to be the most effective method to increase bone density and promote muscle growth in older people with sarcopenia. Older people attending a single exercise class per week and doing some exercise at home can improve muscle strength by 27%, effectively reversing age-related decline (Skleton and McLaughlin, 1996). When it comes to keeping the musculoskeletal system healthy, the bottom line is the common colloquialism: use it or lose it. NT

References

For more on this topic go online...
● Osteoporosis: the clinical nurse specialist role
Bit.ly/NTOsteoporosis

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<table>
<thead>
<tr>
<th>Article</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Part 1: the cardiovascular system</td>
<td>Feb</td>
</tr>
<tr>
<td>Part 2: the respiratory system</td>
<td>Mar</td>
</tr>
<tr>
<td>Part 3: the digestive system</td>
<td>Apr</td>
</tr>
<tr>
<td>Part 4: the renal system</td>
<td>May</td>
</tr>
<tr>
<td>Part 5: the nervous system</td>
<td>Jun</td>
</tr>
<tr>
<td>Part 6: the eyes and ears</td>
<td>Jul</td>
</tr>
<tr>
<td>Part 7: the endocrine system</td>
<td>Aug</td>
</tr>
<tr>
<td>Part 8: the reproductive system</td>
<td>Sep</td>
</tr>
<tr>
<td>Part 9: the immune system</td>
<td>Oct</td>
</tr>
<tr>
<td>Part 10: the musculoskeletal system</td>
<td>Nov</td>
</tr>
<tr>
<td>Part 11: the skin</td>
<td>Dec</td>
</tr>
</tbody>
</table>