Journal of Pelvic, Obstetric and Gynaecological Physiotherapy, Autumn 2021, **129,** 9–21

OPINION

Effects of the menopause on the neuromusculoskeletal system: a narrative review

Y. Coldron
Sutton Community Musculoskeletal Physiotherapy Service, Sutton Health and Care, London, UK

Abstract
The ageing process alters the neuromusculoskeletal (NMS) system, and these changes increase as women go through the menopause and begin the next stage of their lives. This paper reviews research on this topic; however, in some areas, the published evidence is sparse and can be contradictory. Age-related changes and the effect of reduced levels of oestrogen on the spinal motor neurons, skeletal muscle, connective tissue and bone are discussed. The pelvic floor muscles, the role that these play in continence and the function of oestrogen in maintaining the integrity of the pelvic floor complex are addressed. Some management strategies for the NMS system are also outlined.

Keywords: menopause, neuromusculoskeletal system, osteoporosis, pelvic floor, sarcopenia.

Introduction

The present article is one of a trio of papers on the menopause published in this edition of the journal (see also Bird 2021; Lindsay 2021). These are derived from lectures that were presented as parts of the 2020 Pelvic, Obstetric and Gynaecological Physiotherapy (POGP) online course entitled "Menopause Management for Physiotherapists", which was run in partnership with PositivePause (www.positivepause.co.uk). This paper presents an outline of the changes to the neuromusculoskeletal (NMS) system that occur during and after the menopause, and also a discussion of some management modalities.

Women experience hormonal changes during the perimenopausal and menopausal periods that continue throughout the postmenopausal phase until the end of life. Alterations to various bodily systems, including the NMS structure, continue for many years: women may spend one-third or more of their life living through multiple systemic changes. Therefore, raising awareness of this process and coping strategies is incumbent on medical professionals who encounter women during and after the menopause. These changes occur throughout the body, and affect the peripheral nerves, skeletal muscle, connective tissues and bone. Thus, knowledge of age and menopausal changes to the NMS system should underpin all physiotherapy treatment programmes for middle-aged and older women, including, but not confined to, rehabilitation of the pelvic floor muscles (PFMs). Information about the NMS system as a whole during and after the menopause is limited, and further research is needed.

Correspondence: Dr Yvonne Coldron, c/o Andrew J. Wilson Publishing Services, 15 Bughtlin Loan, Edinburgh EH12 8UZ, UK (e-mail: yvonnecoldron@gmail.com).

Nervous system

Normal ageing of the nervous system

Since changes to the brain are beyond the scope of the present paper, this description of age-related changes begins with the spinal cord and motor neurons. Ageing of the NMS system is characterized by loss of spinal motor neurons as a result of a process called apoptosis, a form of cell death (Aagaard *et al.* 2010). Insulin-like growth factor-1 (IGF-1) signalling in the NMS system is normally responsible for muscle hypertrophy, but ageing causes a decrease in the production of this hormone. Loss of IGF-1 may lead to atrophy over time. In addition, there is a concurrent elevation in amounts of circulating cytokines, which may lead to a decrease in the regenerative capacity of nervous tissue. Furthermore, there is also some evidence of an increase in cell oxidative stress with ageing, and this may lead to further nerve degeneration.

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Following the loss of motor neurons, axons adapt to the decrease in stimulus, and this triggers maladaptive axonal sprouting. The net result is a decrease in the number of motor endplate terminals, which accelerates the loss of muscle fibres (Aagaard *et al.* 2010), and causes skeletal muscle dysfunction.

The changes to the nervous system may also incorporate a slowing of axonal conduction speed, and a consequent decrease in agonist muscle activation and antagonist muscle coactivation, with an associated decrease in capacity for force steadiness (Aagaard *et al.* 2010). In addition, Aagaard *et al.* (2010) also reported that there is some evidence that there are reductions in myelination and internodal length, and that the frequency of motor unit firing decreases. These changes negatively affect the function of the motor end plates, and therefore, signalling to the muscle fibres in the motor unit will be impaired. Furthermore, there appears to be a reduction in spinal inhibitory circuitry, and a decrease in the excitability of the corticospinal pathways. All these factors impair the capacity for axonal reinnervation of the denervated myofibres. The consequence of these changes is a decline in the number and size of muscle fibres, i.e. sarcopenia, which results in altered and compromised muscle performance.

Effect of the menopause on the nervous system

There is no definitive evidence that these changes to the NMS system are greater in women during and after the menopause. However, it is known that oestrogen and progesterone are neuroprotective and neurotrophic, and have regenerative effects (Aagaard *et al.* 2010; Singh *et al.* 2016). Therefore, it may be concluded that a reduction in oestrogen and progesterone hormones during and after the menopause may further alter or accelerate the changes to the peripheral nervous system that are associated with normal ageing. For example, there is a small amount of evidence that there is a greater incidence of carpal tunnel syndrome in postmenopausal women compared with those who have yet to go through it (Pascual *et al.* 1991; Kaplan *et al.* 2008).

It is known that there is a higher incidence of peripheral neuropathy in menopausal women, and that low levels of oestrogen and progesterone are associated with lower motor nerve conduction velocity (MNCV). Singh *et al.* (2016) studied MNCV in postmenopausal women who presented with peripheral neuropathy, and compared them with an age-matched group of control subjects. The median, ulnar and common peroneal nerves were tested. It was found that levels of serum oestrogen and progesterone were significantly lower in the participants with peripheral neuropathy compared to those in the control group. Significant differences in the MNCV of all three nerves were found between the two groups in both their right and left limbs. Singh *et al.* (2016) concluded that, while postmenopausal women are at greater risk of peripheral neuropathy, it is the greater decline in serum oestrogen levels among individuals that is critical in the development of this condition.

Further research needs to be undertaken in order to study the effects on the nervous system of the changes in nerve function during and after the menopause.

Physical activity can induce adaptive training in the nervous system, including elevated electromyographic (EMG) amplitudes and an upsurge in maximal motor neuron firing (Aagaard *et al.* 2010). It is possible that an increase in muscle activity augments the supply of trophic substances (e.g. nerve growth factor), and thus, lengthens the lifespan of neurons. Therefore, it follows that women need to continue to exercise during and after the menopause in order to maintain the health of their nervous system.

The muscular system

Sarcopenia

Sarcopenia is defined as "a muscle disease (muscle failure) rooted in adverse muscle changes that accrue across a lifetime; sarcopenia is common among adults of older age but can also occur earlier in life" (Cruz-Jentoft *et al.* 2019, p. 16). The condition is characterized by a reduction in the number of muscles fibres/cells (quantity), and atrophy of the remaining fibres, which results in decreased muscle mass (quality) (Cruz-Jentoft *et al.* 2019). There is a loss of contractile proteins, and a reduced rate of myofibrillar protein synthesis. Over time, the muscle becomes infiltrated with noncontractile tissue such as fat and connective tissue.

On average, peak muscle mass values are reached between the ages of 25 and 35 years, and maintained, with slight reduction, between the ages of 40 and 49 years (Lindle *et al.* 1997; Lynch *et al.* 1999). From approximately 40 to 70 years of age, adults lose 8% of their muscle mass per decade, and 15% per decade after this (Avola *et al.* 2020). A diagnosis of sarcopenia is based on the presence of both low muscle mass

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and poor muscle function. The parameters necessary for a diagnosis and establishing the degree of severity are muscle strength, muscle quality/quantity and physical performance (Cruz-Jentoft *et al.* 2019).

Sarcopenia triggers changes in both kinds of muscle fibres: type II (i.e. the fast-twitch and force-generating fibres) are more affected than type I (i.e. the slow-twitch and endurance fibres), which appear to undergo relative hypertrophy. Therefore, there is a shift from the fast to the slow fibre type during ageing. This process is different from disuse atrophy, which causes a shift from type I to II fibres. Older people experience a loss of strength per unit of muscle mass (Rutherford & Jones 1992; Goodpaster *et al.* 2006), and these changes may occur as early as the fourth decade of life (Lynch *et al.* 1999). This knowledge influences the type of rehabilitation programmes prescribed for older people, which need to include strengthening and force-generating components.

Causes of sarcopenia

Ageing is associated with hormonal fluctuations that cause neurodegenerative changes and a reduction in MNCV, and these factors affect muscle recruitment and activity. As described above, the impairment in the mechanical function of the muscles is accompanied and partly caused by age-related losses in the NMS system. Consequently, muscle recruitment and activity will be affected, as will agonist muscle activation and coactivation, force steadiness, muscle mass, and strength (Aagaard *et al.* 2010). Other muscle ageing factors include mitochondrial dysfunction, DNA mutations and decreased antioxidant capacity. Endocrine factors such as altered regulation of the body's endogenous corticosteroids and IGF-1, and a reduction in thyroxine and insulin levels are important factors in the development of skeletal muscle dysfunction. The role of inflammation in maintaining muscle health is only just beginning to be understood, but it appears that regulation of various inflammatory factors (e.g. cytokines, oxidative stress, hypoxaemia and tumour-derived factors) is also important for maintaining muscle health.

Decreased activity, including a sedentary lifestyle and bed rest, are known causes of muscle mass reduction, but since sarcopenia also results in the loss of muscle fibres, ageing women are at risk of impaired muscle performance (Aagaard *et al.* 2010). Other general health factors contributing to impaired muscle function include poor nutrition, decreased protein intake, problems with malabsorption and anorexia.

Muscle quality was defined by Lynch *et al.* (1999) as specific tension, which refers to the peak torque per unit of muscle mass, and therefore, this is a better indicator of muscle function than strength alone. Two hundred and twenty-four men and 278 women (age range = 19–90 years) were assessed for isokinetic concentric and eccentric peak torque of the elbow and knee flexors and extensors using an isokinetic dynamometer. With advancing age, female muscle quality declined more in the leg than the arm; however, the rate of deterioration of arm muscle quality was the same as in men. Interestingly, women appear to lose strength at an earlier age than men, but the total percentage loss of strength is slightly less than that in men overall. Lynch *et al.* (1999) investigated the relationship of muscle quality to performance, but the distribution of muscle fibre types, muscle mass, neural activation and muscle pennation angles were not addressed. There was a greater loss of age-related concentric strength than eccentric strength/muscle quality in both sexes. In a study of muscle performance in very old age, Lindle *et al.* (1997) found that women appear to preserve their eccentric strength better than men. The European Working Group on Sarcopenia in Older People have recently recommended that low muscle strength, and detection of low muscle quality and quantity confirms a diagnosis of sarcopenia (Cruz-Jentoft *et al.* 2019), with severe sarcopenia further defined as including poor physical performance.

When reviewing the literature on muscle performance, it became apparent that parameters and subject groups differed between the studies, and a variety of methodologies had been used. Therefore, the results and conclusions were inconsistent, and at times, contradictory. Muscle strength, force, mass, quality and task specificity have all been studied, but there is no consensus about the best methods of assessing the muscle changes or hormone variations associated with ageing. Variables which could be studied include neuromuscular facilitation, contractile and mechanical properties, growth hormone factors, endurance and fatigability, maximum muscle strength, power, and rate of force development (Evans & Hurley 1995; Lindle *et al.* 1997).

Menopause, muscle performance and other characteristics

The implications of declining levels of sex hormones for muscle function in women still

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require further research. Tiidus (2011) reviewed the effect of female hormones on muscle performance. Declines in muscle force and mass accelerated in women compared to men of the same age, and the ability to generate muscle force in women declined after the menopause. Tiidus (2011) concluded that, while there was contradictory evidence, hormone replacement therapy (HRT) had significant beneficial effects overall on skeletal muscle mass, force generation, protection from damage and repair indices. The physical activities that showed significant improvements were an increase in jump height, running speed and postural balance. It was suggested that HRT prior to the menopause may be beneficial for retaining muscle properties. There were many variables to be taken into account, but overall, offsetting age-related muscle mass loss, HRT may help women to retain independent lifestyles for longer.

A review of oestrogen, muscle performance and injury risk noted that a deficiency of oestrogen over a 24-week period resulted in a 10% decrease in muscle strength, and an 18% reduction in muscle cross-sectional area (Chidi-Ogbolu & Baar 2019). Oestrogen improved muscle mass and strength, and increased the collagen content of other tissues. In the absence of oestrogen, muscle was more prone to injury, although the mechanism behind this was unclear. Muscle mass is maintained by a balance between myofibrillar protein synthesis and degradation. It appears that postmenopausal women show higher rates of both processes, but it is not yet understood if the protein synthesis is counteracted by an even greater rate of protein breakdown. If this is the case, then muscle mass and strength would decline. Chidi-Ogbolu & Baar (2019) noted that postmenopausal women show reduced sensitivity to anabolic stimulus. However, this reduced level of sensitivity returned to normal in women who were given oestrogen replacement therapy. Furthermore, this treatment was shown to increase myofibrillar protein synthesis in response to resistance exercise.

Carville *et al.* (2006) demonstrated that a group of women taking HRT generated greater power in an explosive leg extension than those who had never undergone the treatment. In a study involving female twins, Ronkainen *et al.* (2009) showed that HRT increased strength, balance and muscle mass. Oestrogen was shown to be beneficial to muscle strength because it improved intrinsic muscular quality (Lowe *et al.* 2010). With oestrogen present, myosin binds strongly to actin, and thus, enhances sliding filament muscle contraction. Furthermore, oestrogen has been shown to be a major regulator of human skeletal signalling in women (Laakkonen *et al.* 2017). Hormone replacement therapy may also influence muscle mass by increasing the expression of IGF-1 signalling, and thus, inducing muscle hypertrophy as well as increasing satellite cell activation (Tiidus 2011).

Conversely, some studies have not reported any increase in muscle mass or size, or improvement in balance with oestrogen supplementation. In a review paper, Elliott-Sale (2014) concluded that there is no unanimous consensus regarding the effect of oestrogen on muscle strength. Unfortunately, the exercise regimes, hormone treatments and outcome measures differed between the studies, and therefore, it is difficult to make comparisons and draw conclusions. Nevertheless, there does seem to be some evidence that the dose of HRT, activity levels, age and medications will all have an influence on muscle performance. In addition, nutrition (in particular, a high-protein diet and adequate levels of vitamin D) will affect muscle mass and functional strength. [N.B. The impact of nutrition is not addressed in the present paper because this was covered by other presenters at the POGP menopause online course (see pp. 66–74)].

The role of other sex hormones (i.e. progesterone and testosterone) in relation to sarcopenia in menopausal women has not been studied extensively. Smith *et al.* (2014) measured the basal rate of muscle protein synthesis and the expression of muscle growth-regulatory gene. They concluded that this process was stimulated by testosterone and progesterone, but not oestrogen. However, this study had a limited sample size, and neither muscle protein breakdown rates nor changes in muscle mass were studied. Therefore, no extrapolation can be made from these results. In two groups of surgically induced menopausal women, administration of 25 mg of testosterone to the treatment cohort significantly increased chest-press and loaded-stair-climb power compared to those receiving placebo (Huang *et al.* 2014). However, other performance-based measures, including gait speed, lift-and-reach, and unloaded-stair-climb speed and power, did not differ significantly between the two groups. The effect of testosterone supplementation on postmenopausal muscle performance is not clear, and further research needs to be undertaken.

With regard to exercise programmes for women, the consensus seems to be that a progressive

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programme of resistance exercise is necessary to increase muscle mass, quality and other characteristics (Aagaard *et al.* 2010). This programme will need to take place over many weeks and months; for example, 6 months of exercise three times a week (Colón *et al.* 2018). Exercise regimes are discussed further in Table 1.

More research needs to be undertaken to assess the effect of both exercise alone, and exercise combined with HRT on menopausal muscle performance and characteristics. In addition, future research needs to define and quantify muscle performance.

Connective tissue

The menopause and connective tissue

The main types of collagen found in the musculoskeletal system are type I, which gives structures tensile strength, and type III, which allows tissues extensibility and elasticity. The evidence for the effect that oestrogen has on connective tissue is very limited and contradictory.

Some 60–85% of the dry weight of a tendon consists of collagen, and 60% of this is type I (Chidi-Ogbolu & Baar 2019). The function of tendons is to connect muscle to bone, transmit muscle forces, store energy and withstand tension during muscle contraction. The mechanical properties of tendons are collagen fibre density, cross-sectional area and diameter, and cross-linking of fibres. Oestrogen is a known regulator of connective tissue metabolism (Leblanc *et al.* 2017).

Oestrogen decreases tendon stiffness, and as a result, protects the attached muscle from injury (Chidi-Ogbolu & Baar 2019). As oestrogen levels decline during the menopause, there is also a reduction in tensile strength, collagen synthesis, fibre diameter and density, and an increase in tendon tissue degradation (Frizziero *et al.* 2014). Oestrogen receptors have been identified in human beings, and it is possible that oestrogen-deprived tenocytes have less viability, a lower cell migration speed and poorer-quality tissue repair.

After the menopause, tendons become increasingly stiff, and since these are attached to bone, muscle or tendon injury may result. This is because tendons have less compliance, and thus, do not respond as quickly to the demands of the muscles to which these are attached. The incidence of Achilles tendon rupture is lower in premenopausal women than men, but the risk of rupture is equal for postmenopausal women and men (Hansen & Kjaer 2014). Therefore, exercise needs to maximize tendon function both eccentrically and concentrically in order to allow the tendon fibres to align, and retain maximum strength and elasticity.

Hormone replacement therapy may encourage the incorporation of more collagen into tendons in postmenopausal women. Hansen *et al.* (2009) found that collagen incorporation into the patellar tendon was 47% higher in women who were undergoing HRT compared to control subjects. Exercise was not found to incorporate collagen further. Hormone replacement therapy may influence tendons by preserving collagen fibre diameter, and affecting tendon morphology and biomechanical properties (Frizziero *et al.* 2014). However, contradictory reports have found no correlation between tendon strength and HRT (Hansen *et al.* 2013). Nevertheless, it was concluded that HRT (oestrogen replacement) may be beneficial, and reduce the risk of lower-limb tendon injury in active postmenopausal women (Hansen & Kjaer 2014).

Approximately 75% of the dry weight of a ligament consists of collagen, and up to 85% of this is type I (Chidi-Ogbolu & Baar 2019). Stiff ligaments are necessary for joint stability, but anterior cruciate ligament stiffness alters during the menstrual cycle. It is thought that elevated oestrogen levels during the follicular and ovulatory phases of this cycle decrease ligament stiffness (Shultz *et al.* 2005; Adachi *et al.* 2008) and increase knee joint laxity, and thus, predispose ligaments to injury and rupture.

Studies of the effect of oestrogen on ligaments have reported contradictory results, but the consensus is that oestrogen may increase collagen synthesis or incorporation into ligaments, and thus, decrease overall stiffness (Chidi-Ogbolu & Baar 2019). Therefore, there may be increased ligamentous stiffness in menopausal and postmenopausal women, which could protect them from injury.

There is a greater proportion of type I than type III collagen in the fascial tissue of both women and men. The function of the fascia is to separate and link other connective tissue structures, and it provides scaffolding for the muscles. The fascia is a three-dimensional matrix of structural support that enables force transmission (Chidi-Ogbolu & Baar 2019). Collagen type I expression is related to age and menopausal status. The arcus tendineus fasciae pelvis (ATFP) provides support to the anterior vagina, and in non-menopausal women, is mainly made up of

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Table 1. Examples of exercise regimes for people with osteoporosis and sarcopenia (adapted from ROS 2020): (CR-10) Borg Category-Ratio Scale

	Osteoporosis			
Variable	Most people with osteoporosis	People with spinal fractures	People who are frail and unable to do exercise	Muscle strengthening (including most people with osteoporosis)
Intensity	Moderate impact × 50 repetitions	Low impact	Avoid prolonged sitting or lying Stand up Walk	8–12 repetitions per exercise Build up to three sets of each exercise CR-10: levels 4–6
Frequency	Daily	Most days	Stand up for a few minutes every hour	2–3 (non-consecutive) days of resistance exercise per week Gradually increase resistance
Duration	20–30 min per session	20 min	A few minutes with hourly repetitions	20–30 min per session
Type of exercise	Moderate impact		Low impact	Strengthening
Examples of exercise	Jogging/running Dancing Racket sports Skipping Balance Hopping Vigorous heel-drops Stamping Jumping Wall or floor press-ups		Walking Marching Sit-to-stand exercise Steps and step-ups Gentle stamping Wall or chair press-ups Balance exercises Postural exercises Heel-drops	Trunk: abdominal recruitment with upper or lower limb loading in functional positions (i.e. sitting and/or standing) mat exercises (if appropriate), such as: bridging trunk extension Pilates Lower limb: gluteal exercises leg-press exercises mini-squats modified lunges toe- and heel-raises hamstring curls Upper limb: shoulder and arm weights resistance bands wall press-ups
Other considerations	Multi-joint exercise Balance (possibly use support for weight-bearing exercises at first) Younger clients: CR-10 level 6 (i.e. breathless but able to talk while doing exercise) Older clients: normal breathing pattern Exercise in pain-free range of motion Use machines, weights and resistance bands			

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type I collagen, small amounts of elastin and smooth muscle. However, in postmenopausal women who are not undergoing HRT, the quantity of type I collagen decreases and the ratio of type I to III collagen also decreases, and this process may compromise tensile strength and increase their susceptibility to anterior vaginal wall dysfunction (Moalli *et al.* 2004). An increase in type III collagen in the ligaments of prolapsed uteruses has been demonstrated. However, there is some evidence that HRT may reverse such changes in levels of type III collagen. Sex hormone (i.e. oestrogen and relaxin) receptor expression has been found in fascial fibroblasts, which inhibits fibrosis and inflammatory activities. The reduction in levels of oestrogen and relaxin after the menopause may explain how hormonal factors relate to myofascial pain, i.e. by sensitizing fascial nociceptors (Fede *et al.* 2016).

Oestrogen has been shown to increase mRNA gene transcription of type I and III collagen in the connective tissues of the pelvic floors of rhesus macaques that had undergone bilateral oophorectomies, which indicates that oestrogen increases collagen synthesis in these tissues (Clark *et al.* 2005). Therefore, lower levels of oestrogen will affect pelvic floor collagen synthesis, which will result in an increase in the ratio of type III to type I collagen, and thus, decreased pelvic floor tensile strength.

These studies show that oestrogen influences the tensile strength of connective tissue, and it is possible that HRT may be beneficial in the maintenance of connective tissue function after the menopause. However, there is a paucity of evidence in relation to the effect of HRT on connective tissue, and this should be the subject of further research.

Pelvic floor complex

Pelvic floor muscles and fascia

The menopause affects all striated skeletal muscles, nerves and connective tissue. The functions and characteristics of the pelvic floor complex, and the different roles that this plays as women age will be of particular interest to readers. Any discussion of the PFMs must also incorporate the fascial attachments and nerves, as well as the muscles themselves. As a result of the limitations of both the original online presentation and the present paper, the female pelvic floor complex is discussed in outline only. The terminology used for the muscular components of the pelvic floor varies, and therefore, these are described in relation to the levator ani muscle group, which is formed by the puborectalis, pubococcygeus, iliococcygeus and ischiococcygeus (Lee 2009). Clinically, the levator ani is often considered as a single unit.

The function of the pelvic floor complex as a whole is to support the internal organs of the bladder, vagina and rectum. In addition, it plays a substantial role in controlling continence. Support is provided by connective tissue integrity and muscle stiffness, and the latter is divided into (1) active stiffness (neuromuscular control) and (2) passive stiffness (structural integrity of muscle components).

Connective tissue components

When considering the pelvic floor complex, the role of the connective tissue needs to be taken into account as well as that of the active muscular components. The diverse structures that support the female genital tract were described by Ashton-Miller & DeLancey (2007). Connective tissue support is provided by the cardinal and uterosacral ligaments, attachment of the levator ani is performed by the ATFP and endopelvic fascia, and the urogenital diaphragm and perineal body form the fusion components of the pelvic floor. In addition, the linea alba ("white line") is a connective tissue structure over the obturator internus muscle and the medial aspect of the ischial spines that has connections to the muscles of the levator ani and the vaginal walls. Together, these structures support the pelvic floor complex like a cat's cradle, and resist downward force as a result of raised intra-abdominal pressure (Lee 2009).

The paravaginal tissue is surrounded by the vaginal wall, the pubocervical fascia and the retrovaginal septum (Hinata *et al.* 2014). In a cadaveric study by Hinata *et al.* (2014), each of the above fasciae was shown to be independently connected to the superior fascia of the levator ani, which contains smooth muscle.

The endopelvic fascia is a tense layer that stretches like a hammock from the linea alba over the obturator internus muscle laterally to the vaginal wall medially. The endopelvic fascia is important in maintaining urinary continence because the urethra lies anterior to and above it, and thus, gets compressed against this tissue during increased intra-abdominal pressure (Ashton-Miller & DeLancey 2007). Therefore, weakness of this layer can lead to stress urinary incontinence (SUI). The common attachment of this

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fascial layer and the levator ani muscle complex to the linea alba means that contraction of the PFMs can elevate the bladder neck by pulling and tightening the endopelvic fascia.

The anatomy of the urogenital tract is discussed by Lindsay (2021) in this edition of the journal (see pp. 22–32). The vagina is supported by its attachment to the endopelvic fascia, perineal body, levator ani and perineal muscles. Tears developing in the endopelvic fascia as a result of vaginal delivery may lead to a central defect, which may manifest as a central prolapse in turn. Disruption of the lateral endopelvic attachment leads to lateral detachment of the vagina from the side pelvic wall, and this may need to be managed surgically. Posterolaterally, the vagina is attached to the endopelvic fascia over the pelvic diaphragm and sacrum by the rectovaginal septum, which extends into the perineal body caudally and into the peritoneum of the pouch of Douglas cranially.

The ATFP appears to be more resistant to damage during a vaginal delivery than the uterosacral ligaments. After a combination of a vaginal delivery, a reduction in sex hormones and ageing, the basic construction of the fascial matrix may be destroyed, and replaced with several bundles of fascia and veins (Hinata *et al.* 2014). A venous plexus appears to replace the fascial connections in multiparous women, and the veins become embedded in the loose but elastic paravaginal tissues.

Muscular components

The levator ani is a broad, U-shaped sheet of muscles that originates from the back of the pubic bone, the linea alba over the obturator internus muscle and the medial aspect of the ischial spines. It stretches backwards and inwards from either side of the pubis to meet in the midline, encircling the urethra, vagina and the rectum, and reaching the coccyx (Ashton-Miller & DeLancey 2007). The morphological structure of the levator ani has been studied in cadavers, and its muscle fibres have been shown to be skeletal, but more medially, these fibres separate into two layers: the deeper stratum consists of smooth muscle and the superficial one is made of striated fibres (Shafik *et al.* 2002). Approximately two-thirds of the striated muscle fibres of the levator ani are type I collagen, and this configuration is affected by the ageing process, during which there is a shift from type III to type I collagen, as discussed above. This is one reason for the need to ensure that women perform strengthening and resistance training as part of their pelvic floor exercise regime.

Neural components

There is some difference of opinion regarding the innervation of the PFMs, but the consensus seems to be that it is innervated by branches of the S2–S4 sacral nerves and the pudendal nerve (Lee 2009). Paravaginal nerves have been found to be distributed evenly between the fasciae in nulliparous women, but the venous plexus found in multiparous women separates these into superior and inferior groups, which are then bundled into several fasciculi containing both sympathetic and parasympathetic nerves (Hinata *et al.* 2014). The paravaginal tissue contains the distal part of the pelvic autonomic plexus and its branches, i.e. the cavernous, urethral and internal anal sphincter nerves (Hinata *et al.* 2014). The sympathetic nerves arise from the lumbar splanchnic nerves, and the parasympathetic nerves originating in the pelvic plexus. Micturition is partly controlled by these autonomic nerves. The bilateral hypogastric nerves are joined by those of the pelvis in the anterior sacrum, and form part of the pelvic plexus. The changes to the NMS system caused by ageing and the menopause are described above, but together with the potential damage to the pudendal nerve associated with vaginal delivery, the nerve supply to the muscles of the levator ani may be compromised during the menopausal period and afterwards. Before the menopause, the pelvic floor nerve supply may be compromised as a result of vaginal delivery, but the integrity of the pelvic floor complex may be enough to maintain the function of the levator ani. However, if the damaged nerve supply is then further compromised by ageing and the menopause, neuropathy may ensue, and control of the levator ani muscles may decrease, leading to urinary incontinence; ultimately, this may be a factor in pelvic organ prolapse (POP) (Tindall 1987).

Pelvic floor complex dysfunction

The integrity of the pelvic floor complex depends on the nerve supply and connective tissue supporting components remaining intact, as well as the contractile mechanisms of the component parts of the levator ani muscles. The function of the pelvic floor complex is to support the internal organs, and it plays a substantial role in continence. This involves several mechanisms including control over urethral position and

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closing using the levator ani to enhance the role of the puborectal, uterosacral and cardinal ligaments. If the levator ani muscles maintain proper closure of the genital hiatus, the ligaments and fascial structures supporting the pelvic organs are under minimal tension. The fasciae simply stabilize the organs in their positions above the levator ani. With NMS damage to the pelvic floor, and a consequent loss of ability to close the muscles properly around the urethra and vagina, the supporting connective tissue structures will stretch and may eventually fail, which will result in POP (Chidi-Ogbolu & Baar 2019).

Pelvic floor dysfunction (PFD) in women increases in frequency with advancing age as a result of, for example, impaired neuromuscular function and resultant sarcopenia. Decreased EMG activity in the PFMs has been observed in menopausal and postmenopausal women who were compared with nulliparous subjects (Pereira *et al.* 2016), resulting in decreased active stiffness of the PFMs. Lower EMG values have been shown to be linked with SUI (Burti *et al.* 2015).

Another factor that may contribute to an increase in PFD in postmenopausal women is abdominal adiposity because women tend to carry more fat in this area than in the buttocks and thighs. It is possible that this increased abdominal adiposity might be connected with increased intra-abdominal pressure, and thus, present a constant strain on the pelvic floor musculofascial complex and decrease the physiological stiffness of the levator ani.

The menopause and the pelvic floor complex

Oestrogen is thought to influence the connective tissues around the PFMs, urethra and base of the bladder. It stimulates the growth of the urethral epithelium, and increases the vascularity of submucosal venous plexuses in the urethra.

The received wisdom maintains that there are oestrogen receptors in the PFMs, but literature on this subject is scarce. However, in a small study using pelvic floor biopsies, Smith *et al.* (1993) found that there were oestrogen and progesterone receptors in the levator ani complex; however, there were more in the connective tissue than in the striated muscle. In addition, receptors were also seen in the urogenital ligaments and myometrium. Oestrogen receptors have been observed in smooth muscle cells in the vagina, uterus and fallopian tubes (Chidi-Ogbolu & Baar 2019), and in the uterosacral ligaments and pubocervical fascia (Clarke *et al.* 2005). The uterosacral ligament was found to be significantly thinner, and to contain fewer oestrogen and progesterone receptors after the menopause (Reay Jones *et al.* 2003). Hence, lower levels of oestrogen following the menopause may compromise the function of and support for the pelvic floor complex. A reduced number of oestrogen and progesterone receptors have been found in menopausal women with POP (Bai *et al.* 2005). The risks of POP in menopausal and postmenopausal women are many and multifactorial, and therefore, beyond the scope of the present paper. A recent literature review by Odofin (2020) highlighted that individual pelvic floor anatomical differences, pregnancy and vaginal delivery were associated with POP in primiparous women. Anatomical changes after childbirth, the depletion of oestrogen affecting the connective tissue and muscle tissue, which causes sarcopenia, reduced EMG activity in the levator ani, and lifestyle alterations leading to reduced exercise and increased abdominal adiposity may all contribute to a less-functional pelvic floor complex, the net result of which may be POP, urinary incontinence and sexual dysfunction. Although the hormonal changes that occur during the menopause contribute to PFD, these are not wholly responsible for it.

During the menopausal and postmenopausal periods, PFD can be managed with strength and endurance training (Dumoulin *et al.* 2018). The increases in muscle mass associated with strength training will also influence the integrity of the supporting connective tissue structures. Since oestrogen supplementation is thought to increase muscle size, it is worth considering HRT because this has been shown to reduce urethral incontinence (Jármy-Di Bella *et al.* 2007). However, the effect of this treatment on continence is controversial, and a Cochrane review found that systemic HRT may increase the risk of incontinence (Cody *et al.* 2012). The authors recommended that postmenopausal women who are considering receiving systemic HRT for reasons other than continence should be warned that they may develop urinary incontinence, but that application of local oestrogen treatment may help to control this problem (Cody *et al.* 2012). Nevertheless, a combination of PFM resistance training and HRT may offer the best outcomes of any treatment for PFD.

Osteoporosis and bone loss

It is widely known that bone loss and osteopenia, or osteoporosis, increases significantly with age, especially in women. Nine million osteoporotic

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fractures are estimated to occur worldwide each year (Hillard *et al.* 2017). Women are more susceptible to bone loss. Franic & Verdenik (2018) found that the prevalence of osteoporosis in female subjects was 24% between 60 and 64 years of age, and 37.4% between 70 and 75. One in three women will suffer an osteoporotic fracture at some point in their lives. The radius is most commonly fractured at 50 years, vertebrae between 60 and 70, and the femoral neck in the late 70s.

Bone turnover and remodelling occur throughout our lives. Osteoclasts are recruited to the bone surface, where these dissolve or reabsorb bone (the resorption phase). The osteoclasts then undergo apoptosis (the reversal phase), and osteoblasts are subsequently recruited to the bone surface and deposit new collagen on the bone surface (the formation phase). This results in mineralization and the formation of new bone (the mineralization phase).

The menopause and bone

During the menopause, the normal cycling of remodelling is impaired, and osteoclastic reabsorption occurs without any corresponding increase in osteoblastic activity. Therefore, more bone is reabsorbed than new bone deposited. The net result of oestrogen deficiency after the menopause is accelerated and disproportional bone loss. The loss of trabeculae leaves bigger holes between these cross-struts, and causes cumulative fatigue and microdamage. The annual proportion of bone loss may be as much as 4–5% following the menopause.

Risk factors for osteoporosis

The risk factors for osteoporosis are many and varied, and not just confined to reduced levels of oestrogen. These include:

- being over 50 years old;
- a family history of osteoporotic fracture;
- ethnicity;
- a previous fragility fracture;
- early or surgical menopause;
- hypergonadism;
- a body mass index below 18.5;
- smoking;
- drinking more than two units of alcohol per day;
- a lack of physical activity in the 20s or 30s;
- a low intake of calcium and vitamin D;
- medication such as corticosteroids, chemotherapy, antiepileptics, antiretrovirals and heparin; and
- certain medical conditions that may predispose women to alterations in the bone turnover process, such as rheumatoid arthritis, malabsorption syndrome, hyperparathyroidism, hyperthyroidism and Cushing's syndrome (Hillard *et al.* 2017).

Diagnosis of osteoporosis

A diagnosis of osteoporosis is often not made until a fracture has occurred, and the patient may then be referred for dual-energy X-ray absorptiometry. This scan measures bone mineral density (BMD), and is used for diagnosis when osteoporosis is suspected. The L5 vertebra and femoral neck are scanned because these are comprised of 50% trabecular and 50% cortical bone. A T-score is a measure of the differences in BMD between a patient and a normal healthy adult. It is calculated by comparing the patient's BMD with the mean value associated with a standard young adult population, and expressed in terms of the number of standard deviations (Aldieri *et al.* 2018). A T-score of +2.5 to –1.0 is normal. Osteopenia is present if a T-score is –1.0 to –2.5, and osteoporosis is diagnosed if it is –2.5 or below. Osteoporosis can also be diagnosed with bone turnover markers in the blood, or quantitative ultrasound of the calcaneus. These tests are often combined with Fracture Risk Assessment Tool scores, which assess the risk of fracture over the following 10 years. Risk is graded as low at 10%, moderate at 10–20% and high at more than 20%.

Management of osteoporosis

Medications such as bisphosphonates, selective oestrogen receptor modulators and HRT have a positive effect on BMD. Bone mass and function are improved by HRT, and it significantly reduces the incidence of osteoporosis (Franic & Verdenik 2018).

Lifestyle modification has a large role to play in the prevention and treatment of osteoporosis, particularly cessation of smoking and reducing alcohol intake. Increasing protein consumption may have a beneficial effect on BMD. There is some controversy about calcium and vitamin D supplements, but because the latter facilitates the absorption of calcium, women should discuss the efficacy of vitamin D supplementation with their general practitioner or menopause consultant. In the first years after the menopause, moderate excess bodyweight significantly reduces vertebral postmenopausal bone loss (Trémollières *et al.* 1993). However, being

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overweight has other risks and is generally not advisable.

Weight-bearing, resistance and strengthening exercises have a major role to play in the prevention and treatment of osteoporosis. Exercise has been shown to improve bone formation and decrease bone resorption biomarkers in people with osteoporosis (Marini *et al.* 2020). As physiotherapists, we need to consider weight-bearing, resistance, and moderate- or low-impact exercise regimes since these appear to increase BMD in postmenopausal women (Table 1). These need to take place over a long period of time: 4–6 months of progressive resistance training is regarded as the minimum amount of time needed to produce an effect on BMD (Layne & Nelson 2001), and if exercise ceases, some bone mass may be lost within a month (Dalsky 1988).

Rheumatological and connective tissue diseases

Apart from those mentioned in the discussion of NMS dysfunction above, further exploration of the effects of the menopause on rheumatological and connective tissue conditions is beyond the scope of the present paper. While patients with rheumatoid arthritis are at increased risk of osteoporosis, there does not appear to be any evidence that HRT increases the risk of developing this condition, and it does not induce flare-ups (Hillard *et al.* 2017). There is some risk that oestrogen increases susceptibility to lupus, and oral oestrogen elevates the risk of thromboembolic disease in those who are predisposed to this autoimmune disease.

There is a marked increase in the number of women who develop osteoarthritis after the menopause, and it could be that oestrogen is essential for maintaining the homeostasis of the articular cartilage, although evidence for this is scarce. However, there is some proof that articular cartilage degradation and the need for joint replacement surgery are decreased among users of HRT (Hillard *et al.* 2017).

Conclusion

Changes to nerves, muscles, connective tissue and bone occur with both ageing and the menopause. These may affect the ability of women to exercise and maintain a healthy lifestyle. With a reduction in neuromuscular function, they may be predisposed to falls and osteoporotic fractures. There is increasing evidence that resistance and strengthening exercises may offset some of the changes to the NMS system, and these need to be continued throughout a woman's life. Physiotherapists are in an ideal position to develop exercise programmes for women as they age, and help them become involved in social prescribing enterprises. Physiotherapists with anatomical, physiological and therapeutic knowledge of PFD can support menopausal and postmenopausal women who present with symptoms such as incontinence and POP. The role of sex hormone supplementation in preventing and treating NMS dysfunction remains understudied, but there is increasing evidence that HRT may help to slow changes to the NMS system in menopausal and postmenopausal women.

The key message is that all perimenopausal, menopausal and postmenopausal women should be given advice about how to exercise for life to maintain and improve their NMS health.

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Until recently, Yvonne Coldron PhD MSc GradDipPhys MMACP MPOGP worked as an Advanced Physiotherapy Practitioner (MSK) for Sutton Health and Care. From 2007 to 2020, she also worked in private practice as a women's health physiotherapist at North Downs Hospital, Caterham, Surrey. Yvonne has been involved with the development and teaching of the POGP short courses on pregnancy-related musculoskeletal conditions since 2008, and contributed to the booklet on pregnancy-related pelvic girdle pain and the good practice statement on transcutaneous electrical nerve stimulation. Her doctoral thesis was entitled "Characteristics of abdominal and spinal muscles in postpartum women". Yvonne now works as a regional advancing practice supervision and assessment lead (London) for Health Education England.

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