Preserving muscle strength and function in old age

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For years it has been assumed that muscle wasting was an inevitable part of growing older, but John Starbrook, who at 87 completed the 2018 London Marathon is, together with several other masters athletes, forcing us to reconsider this view. While the media is full of how sedentary lifestyles and poor nutrition are making our youth ill, the research also seems to suggest that it may be an important factor influencing how our neuromuscular systems age.

LEARNING OUTCOMES
TO SUPPORT PHYSIO FIRST QAP

1. Understand that maintaining muscle mass and strength in old age is dependent on several variables.
2. Be aware of how different lifestyle choices, especially exercise, can influence neuromuscular aging.
3. Help patients to understand that they have a degree of control over how they age through knowledge sharing.

Introduction

Old age is commonly associated with a progressive loss of muscle mass, strength and performance. At its worst this can lead to physical disability, poor quality of life, and premature death.

This loss of muscle mass, also known as sarcopenia, and function is commonly regarded as a given, and researchers (Fujita *et al* 2007) often refer to these changes happening as a natural consequence of "healthy aging".

I would like to challenge this view. I believe that the current research is showing that sarcopenia and loss of muscle function is a result of the natural aging process of sedentary adults, not healthy ones. We know that a sedentary lifestyle is extremely unhealthy, contributing to a wide variety of illnesses including diabetes and heart disease.

While it is the case that a sedentary person may appear healthy, their physiology is affected in a very different way compared to that of active individuals. The research clearly shows that a sedentary lifestyle does not allow healthy aging of the neuromuscular system and it is my opinion that researchers should not refer to sedentary individuals as undergoing healthy aging (Passaro *et al* 2018).

There is currently no consensus with regard to a definition for sarcopenia. Most of the formulas used to detect it usually consider a person as being sarcopenic if their muscle mass deviates from the standard gender-specific mean muscle mass for young adults.

The prevalence of sarcopenia appears to vary greatly between different populations. In Taiwan, for instance, 26% of men and 19% of women over the age of 80 were found to be sarcopenic, while researchers in America have put the figures at 50% of men and 72% of women for a similar cohort (Mitchell *et al* 2012).

Differences in DNA, epigenetics, lifestyle, exercise and nutrition may all play a part in these differences as they have been shown to have an effect on muscle metabolism in old age.

Age-related changes in the neuromuscular system that affect performance

The following highlights some of the findings from a recent in-depth review of how aging affects skeletal muscle performance. For more detail on each of the topics, I recommend accessing the full Tieland *et al* (2018) research.

THE MUSCULAR SYSTEM

Studies have shown that, on average, muscle mass declines in the general population by around 0.37% per year in women and 0.47% in men. This increases to 0.64-0.70% and 0.80-0.98% respectively in people over the age of 75 (Mitchell *et al* 2012).

Several studies also show that the loss of muscle mass in old age is due to both a decrease in the total number of muscle fibres, and in the muscle fibre size (Tieland *et al* 2018). As it is mostly the Type 2 fast twitch, rather than the slow twitch Type 1 muscle fibres that appear to reduce in size, this may explain why elderly people in the general population often struggle to carry out movements that require quick, strong muscle contractions, e.g. climb stairs or get up off the floor.

Studies in rats have shown that there is also an increase in muscle fibrosis between muscles fibres which can influence the elastic properties of muscles. An increase

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in intermuscular adipose tissue also appears to be a symptom of old age, but the research has found that the amount can vary greatly between individuals (Tieland *et al* 2018).

A decrease in aerobic capacity of around 10% per decade has been observed. This is thought to be partly due to changes in the mitochondrial functioning and content of muscle cells (Tieland *et al* 2018).

CONNECTIVE TISSUE

The functional stiffness of tendons seems to decrease as we age, despite the tendon collagen becoming stiffer. The end result is a decrease in the spring-like properties of the muscle-tendon unit and a subsequent decrease in performance (Tieland *et al* 2018).

NERVOUS SYSTEM

It has been observed that, as we get older, the number of motor units in our muscles reduces. There also seems to be an increase in the number of muscle cells served by a single motor unit. This is thought to be due to compensatory sprouting of the surviving neurons (Tieland *et al* 2018) which the researchers suspect is the reason for the observed loss of Type 2, fast twitch, muscle fibres in some older people. This may be due to the denervation of these fibres and the subsequent re-innervation through axonal sprouting from slow motor neurons.

The good news is that the research has shown that healthy, active older adults can preserve their ability to activate their muscle motor units optimally (Tieland *et al* 2018).

The active, older population

Wroblewski *et al* (2011) investigated muscle mass and body composition in masters athletes. They found that chronic intense exercise, i.e. more than four sessions a week, preserved lean muscle mass into old age and, despite the age-related increase in total body fat, this did not lead to fatty infiltration into the muscles themselves.

The research concluded that the decline in muscle mass previously thought to be a natural part of aging was more likely to be due to lifestyle choices, e.g. a sedentary lifestyle and poor nutrition.

Mckendry *et al* (2018) supported these findings in their recent review and meta-analysis. They concluded that the current evidence suggests that chronic exercise training preserves physical function, muscular strength and body fat levels in old age, similar to that of young, healthy individuals. Interestingly, Piasecki *et al* (2016), found that masters athletes experience a drop in motor unit numbers similar to that of the elderly, sedentary population.

There is still a paucity in research investigating physically fit older people and it's not clear how many of the previously mentioned neuromuscular changes also applies to this group. What the current research does suggest is that, if fit older adults are not spared from these neuromuscular changes, it may be possible to combat their negative affects through other strategies such as exercise and nutrition.

Inflammation

Ageing is often accompanied by inflammatory disorders. Slight elevations in circulating pro-inflammatory mediators and decreases in anti-inflammatory cytokines result in chronic low-grade inflammation which can add to muscle loss in older people (Zhai & Xiao 2017). Strategies that decrease chronic, low-grade inflammation may thus be important to combat sarcopenia (Dalle *et al* 2017).

Researchers found that, in rats, low-grade inflammation attenuated their muscle synthesis response to dietary protein, leucine and that antioxidant supplements that reduced the inflammation were effective in improving the anabolic response to leucine.

Other strategies that might be considered in combating inflammatory disorders include Omega 3 fatty acids that have been shown to alleviate systemic inflammation and increase muscle volume and muscle strength in the elderly (Dalle *et al* 2017), and vitamin D which is important for the maintenance of skeletal health and the immune system and prevents muscle wasting (Dalle *et al* 2017). Supplements may be of use to those who are at risk of deficiency in either of these elements.

Building new muscle: effects of nutrition and aging

It has been established that the balance between skeletal muscle breakdown and synthesis is strongly influenced by the levels of amino acids circulating in the blood. The exact science involved in the process is still unclear, but results thus far suggest there is an intricate relationship between amino acid availability, skeletal muscle synthesis and aging.

Ingesting small quantities, i.e. between 7-10g, of essential amino acids can

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stimulate skeletal muscle synthesis in young individuals, but not in older adults. However, larger quantities, i.e. between 20-40g, of amino acids do stimulate muscle protein synthesis to the same degree in all age groups (Dickinson *et al* 2013).

Dickinson *et al* (2013) conclude that the data indicates that the "threshold" at which amino acids stimulate muscle synthesis is increased with age, and so this process could be enhanced by ingesting adequate quantities.

As previously mentioned, leucine has been identified to be important for muscle synthesis, having been shown to be a potent stimulator for muscle growth. In studies where 2g of leucine concentrate was included in just 7g of protein, it was found that both the younger (28/30yrs), and older (66/70yrs) subjects showed the same level of muscle synthesis despite the small dose of protein ingested (Katsanos *et al* 2006; Paddon-Jones & Rasmussen 2009). Thus, increasing the amount of leucine within their diet may promote muscle synthesis in older adults.

This, however, contradicts prior research in which no difference in muscle synthesis was found in response to 20g amino acid ingestion between age groups (Pennings *et al* 2010). This disparity may be due to cultural differences (Dickinson *et al* 2013) that lead to societal discrepancies in daily physical activity and lifestyle. Exercise has been shown to enhance older adults' anabolic response to amino acids and it could be that the results for the subjects in Pennings' study were due to them having more active lifestyles.

Both of these studies are also at odds with Verhoeven *et al* (2009) who found no increases in muscle strength or mass in healthy elderly men who had taken 2.5g leucine supplement for three months.

The subjects in this Verhoeven *et al* (2009) study consumed mixed meals, i.e. containing protein, carbohydrates and fat, which may have influenced how their muscles responded to the leucine, and which is further explored in this article in the section on insulin.

Another randomised control trial, conducted over three weeks, looked at the effect of 20g protein supplement on muscle mass post total knee replacement surgery. The researchers found that the control group showed attenuated muscle atrophy in all the muscles of the operated and the un-operated leg (Dreyer *et al* 2013). At two weeks post-surgery the placebo group showed a -14.3 ± 3.6% change in muscle mass from baseline, and at six weeks this had increased to -18.4 ± 2.3% compared to -3.4 ± 3.1% and -6.2 ± 2.2% for the control group's operated legs.

The variability in these results underlines how complex the mechanisms behind sarcopenia are, and that a multi-faceted treatment approach is needed, and Dickinson and colleagues (2013) warn that it is important to remember that exercise has been shown to play an important role in muscle synthesis, and that physical activity or base level fitness may be another important reason for differences in results.

I fully agree with this statement since the subjects in Dreyer's study were asked to administer their supplements an hour after physical therapy, while Verhoeven and colleagues (2009) did not report on the activity levels of their subjects. Why the timing of food intake may be important is discussed later in this article.

The role of insulin

There is evidence that a meal that includes 40g carbohydrates and 40g protein interferes in the ability of amino acids to elicit a protein synthesis response in older adults (Dickinson *et al* 2013).

This puzzled researchers since a meal containing carbohydrates causes an increase in circulating insulin, and insulin is known to stimulate muscle growth. Further investigation suggested this effect may be due to the inability of insulin to stimulate an increase in muscle blood flow in older adults. Artificially increasing the blood flow in the muscles of older adults caused the difference in muscle synthesis between age groups to disappear.

These results are supported by those of Fujita *et al* (2007) who found that a bout of aerobic exercise before eating a mixed meal could restore the normal muscle protein synthesis reaction to insulin.

Building new muscle: effects of exercise and aging

RESISTANCE EXERCISE

Early studies showed that resistance exercise could increase muscle size and strength in older individuals, but not to the same level as for younger individuals (Peterson *et al* 2011). This may not be entirely true, however, as in more recent research Deutz *et al* (2014) have shown that older individuals may be able to achieve similar muscle gains.

There is evidence that more protein is incorporated into muscle if resistance exercise is performed before a meal (Pennings *et al* 2010). Drummond *et al* (2008) also found that, by performing a bout of resistance exercises before ingesting 20g of essential amino acids, older athletes experienced a similar anabolic response to the younger ones.

This is thought to be due to the exercise decreasing the insulin resistance in the muscle cells and increasing the blood flow, allowing for a normal anabolic response to the circulating amino acids.

AEROBIC EXERCISE

Harber *et al* (2012) showed that a 12-week cycling programme was effective in increasing muscle mass in both young and old individuals. The researchers suggest that the secret may again lie in the ability of aerobic exercise to overcome age-related insulin resistance. It is important to note that the individuals studied were not trained athletes, so it may also be the case that the improvement was because of their initial low baseline muscle mass prior to undergoing the exercise programme.

In an earlier investigation, Fujita *et al* (2007) examined the effect of aerobic exercise on muscle protein synthesis and found that, 20 hours after a single bout of walking for 45-min on a treadmill, at 70% of maximum heart rate, the physiological anabolic response of muscle protein to insulin was restored in older individuals.

Dalle *et al* (2017) also argues that chronic aerobic exercise further promotes muscle synthesis by decreasing chronic low grade inflammation.

Conclusion

Several factors may influence muscle loss in older age. Sedentary older adults appear to have a blunted muscle synthesis response to dietary amino acids when compared with young controls, which is likely to be a result of insulin resistance. However, when older adults perform a bout of aerobic exercise or resistance training before a meal they experience a similar muscle synthesis response to young controls. This may explain why masters athletes do not exhibit the same declines in muscle mass and function as sedentary older adults.

It is clear, therefore, that physical fitness is a key factor for managing health during all stages of life and should be promoted. As healthcare providers we should take every opportunity available to educate and inform our patients of the long-term benefits of physical fitness.

Generic advice on exercise should be avoided in favour of helping our patients with fitness plans that are tailored to their lives, and that they truly feel comfortable with. By empowering patients to take responsibility for their own health we may one day be able to reduce the detrimental effects of chronic illnesses on our society.

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