

Chapter

THE EFFECTS OF AGEING AND EXERCISE ON SKELETAL MUSCLE STRUCTURE AND FUNCTION

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ABSTRACT

Musculoskeletal ageing is associated with profound morphological and functional changes that increase fall risk and disease incidence and is characterised by age-related reductions in motor unit number and atrophy of muscle fibres, particularly type II fibres. Decrements in functional strength and power are relatively modest until the 6th decade, after which the rate of loss exponentially accelerates, particularly beyond the 8th decade of life. Physical activity is a therapeutic modality that can significantly attenuate age-related decline. The underlying signature of ageing, as manifested by perturbed redox homeostasis, leads to a blunting of acute and chronic redox regulated exercise adaptations. Impaired redox regulated exercise adaptations are mechanistically related to altered exercise-induced reactive oxygen and nitrogen species generation and a resultant failure to properly activate redox regulated signaling cascades. Despite the aforementioned specific impairment in redox signaling, exercise induces a plethora of beneficial effects, irrespective of age. There is, therefore, strong evidence for promoting regular physical exercise, especially progressive resistance training as a lifelong habitual practice.

Keywords: sarcopenia, atrophy, reduced function, redox signaling, exercise interventions

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INTRODUCTION

Ageing is associated with progressive functional decrements across all bodily systems. For example, indices of cardiovascular function such as maximal oxygen uptake ($\text{VO}_{2\text{max}}$) are progressively affected in aged individuals. Ageing also impairs the redox-regulated stress responses to exercise, with deleterious effects on skeletal muscle structure and function contributing to the decline in exercise capacity with advancing age, a process which is exacerbated by chronic habitual inactivity. Eventually this leads to a compromised ability to perform habitual activities of daily living and increased risk of falls, particularly in very old populations. This chapter explores the age-related physiological decrements of skeletal muscle structure and function, with particular emphasis on the following: 1) how ageing negatively affects skeletal muscle structure and function; 2) the age-related effects of how habitual physical activity levels influence redox homeostasis at rest and following both acute and chronic exercise training in humans; 3) how physical activity plays its role in helping to combat these deleterious effects.

Ageing and Functional Capacity

Ageing is a process in which motor units (MUs) undergo profound changes, leading to progressive skeletal muscle atrophy, and thus weakness (Hepple & Rice, 2015). It has been shown that muscle mass peaks at around 24 years of age, with only a small progressive decline (~10%) up until the 6th decade, after which there is an accelerated reduction, with an additional 30% decrease up to the age of 80 (Lexell et al., 1988). Similarly, the reductions in voluntary strength are relatively well maintained up to the age of ~ 60 years, before showing accelerated reductions exceeding 50% of the strength capabilities of younger populations (Vandervoort, 2002). Indeed, Frontera et al. (2000) conducted a longitudinal study on aged males (65.4 ± 4.2 yrs) over a 12 yr period and found significant decreases in muscle cross-sectional area (CSA) (~15%), isokinetic strength (20-30%), % Type 1 fibres and capillary-to-fibre ratio. Ageing also negatively impacts the ability of skeletal muscles to regenerate after injury due to the decreased regenerative capacity of aged satellite cells (Tintignac et al., 2015).

This progressive decrease in skeletal muscle functional capacity has been shown to adversely affect functional activities of daily living, leading to increased risks of serious adverse events occurring such as accidental falls (Deschenes, 2004). Events such as falls, which can have serious debilitating effects, are often due to individuals having insufficient 'functional' strength to prevent the fall, due to decreased strength and power capabilities of the skeletal muscles, particularly in the lower limbs (Hunter et al., 2004). It is estimated that up to a half of the bone fractures sustained in the elderly could be avoided by increased strength and coordination (Hollman et al., 2007). Apart from the debilitating implications associated with these adverse events, there are also very obvious socio-economic implications, especially as the world's population of over 65s is expected to triple from 600 million in 2000 to in excess of 2 billion by 2050 (Zembroń-Lacny et al. 2014). The population of elderly requiring long term care due to loss of functional independence is also set to quadruple by 2050 (Miljkovic et al., 2015).

Sarcopenia: Mechanisms and Implications for Habitual Daily Living

Sarcopenia is not considered a disease, rather a collection of conditions that lead to progressive functional deficit (Peterson and Gordon, 2011). It is characterised by age-related changes in skeletal muscle in the form of: 1) reduction of muscular protein mass and CSA; 2) fat and connective tissue infiltration (Hollman et al., 2007). It can be defined as a pathological loss of muscle mass of more than two SDs below the mean skeletal mass index (SMI) of young adults, with its prevalence accelerating particularly sharply in very old populations. The prevalence of Sarcopenia may even be as high as 50% of populations over 80 years old (Zembroń-Lacny et al., 2014), although this is hard to determine as there are currently no standardized diagnostic criteria (Burton & Sumukadas, 2010). Sarcopenic individuals have also been shown to display greater incidences of obesity (Evans and Campbell, 1993), osteoporosis (Ferruci et al., 2002), insulin resistance (Boden et al., 1993) and arthritis (Roubenoff, 2000).

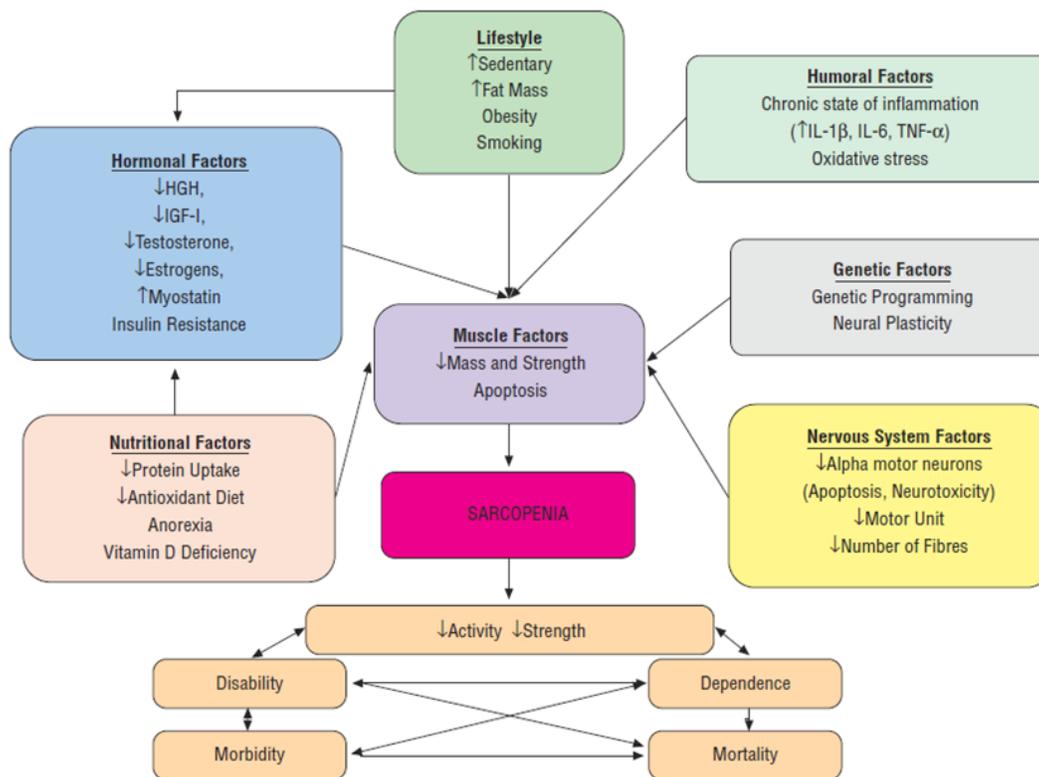


Figure 1. Scheme of the different etiological sarcopenia mechanisms and their consequences. *Used with permission from Beas-Jiménez et al., (2011).*

The underlying mechanisms of sarcopenia are still poorly understood, although it appears to be a multifactorial etiology as outlined in Figure 1 (Beas-Jimenez et al., 2011). Sarcopenic muscle is characterized by muscle fibre atrophy; decreased muscle fibre number; increased heterogeneity of fibre size; increased Type I to Type II fibre ratio; increased grouping of fibre types; increased prevalence of mixed muscle ‘hybrid’ fibres (expressing slow and fast

isoforms of myosin heavy chain); presence of centralised myonuclei; and infiltration of non-muscle cells such as adipocytes (Tintignac et al., 2015). There is evidence that the reduction in strength in elderly populations is not entirely accounted for by muscle atrophy alone, with previous findings of reductions in strength prior to significant muscle loss. The reasons for this would appear to result from: 1) neurological impairments, such as a decreased ability to recruit the same proportion of the agonist muscles compared to younger populations, combined with increased antagonist muscle co-activation (Degens et al., 2009); 2) decreased intrinsic force generating capacity of muscle fibres due to cellular and molecular changes to muscle fibres (Miljkovic et al., 2015). The following sections only briefly outline some of the underlying mechanisms of sarcopenia. For a more in-depth overview, please refer to a recent review on ageing skeletal muscle by Hepple & Rice (2015).

Motor Unit Loss and Fibre Atrophy

The remodeling of MUs and denervation of fibres with ageing due to repeated cycles of skeletal muscle fibre denervation-reinnervation throughout adulthood are known to induce changes to the pre and postsynaptic components of the neuromuscular junction (Oda, 1984). This contributes to impaired neuromuscular junction signaling and instability, resulting in: MU losses; preferential atrophy and angular shaping of type II fibres; hybridization and fibre type grouping (Hepple & Rice, 2015).

Because muscle activation follows a size order of recruitment (Henneman et al., 1965), i.e., smaller MUs have lower, and larger MUs have higher threshold levels of activation respectively, the smaller type I fibres, especially those comprising the postural muscles, are subject to more regular habitual activation than the larger type II fibres. A likely consequence of a sedentary habitual existence is that activation of the higher threshold type II MUs are chronically reduced for extended periods of time. This situation is likely exasperated as we age due to physical activity levels typically decreasing. The chronic inactivation of type II MUs in particular, likely expedites the process of fibre atrophy, MU losses and the resulting denervation of their muscles fibres. Age-related MU remodeling is also associated with a reduction in calcium release from the sarcoplasmic reticulum (Deschenes, 2004) and actomyosin crossbridge speed (Vandervoort, 2002). These consequences, combined with the preferential atrophy of type II fibres, have obvious significant implications on muscle force and contraction speed. This helps explain the slower, weaker movements, particularly in the lower limbs, characterised in very elderly populations.

Muscle Fibre Grouping

The repeated cycles of denervation & reinnervation throughout adult life, likely results in denervated fibres meeting one of three fates: 1) reinnervation by the original MU; 2) reinnervation via axonal 'sprouting' of adjacent MUs (where motoneurons enlarge their MU territory by capturing neighbouring fibres); 3) remain denervated, eventually leading to progressive atrophy, change in shape and permanent loss (Hepple & Rice, 2015). The fibres that do get reinnervated by adjacent fibres cause other 'active' MUs to increase in size as they now have to innervate more muscle fibres (Vandervoort, 2002). This process helps promote the phenomenon of fibre type grouping or 'clustering' in ageing muscle, where fibres of the same MU group together, as opposed to the sporadic distribution of fibre types typical in young muscle (Figures 2,3 – Anderson, (2003)). The sprouting of axons that connect to

previously innervated neighbouring fibres increases the presence of ‘hybrid’ muscle fibres, i.e., fibres that express more than one myosin chain isoform as a result of reinnervation by MUs containing different myosin chain isoform (Deschenes, 2004). The combination of MUs increasing in size and the grouping of fibre types within whole muscle is likely to negatively affect the efficiency of movement and coordination in ageing individuals.

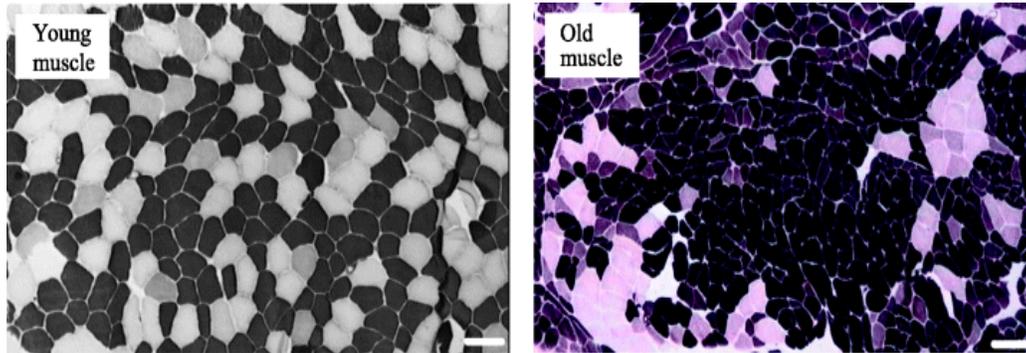


Figure 2. Grouping of muscle fibre types in ageing muscle. ATPase staining of a young muscle (22 y/o) and old (87 y/o) muscle. Note the random distribution of the fibre types in the young muscle vs. the pronounced grouping of the fibre types in the old muscle. Dark fibres = Type 1 fibres; White fibres = Type IIA; Grey fibres = Type IIX (or I/IIA) fibres; Bar = 100 μ m. ATPase staining (pH 4.6). *Used with permission from Anderson (2003).*

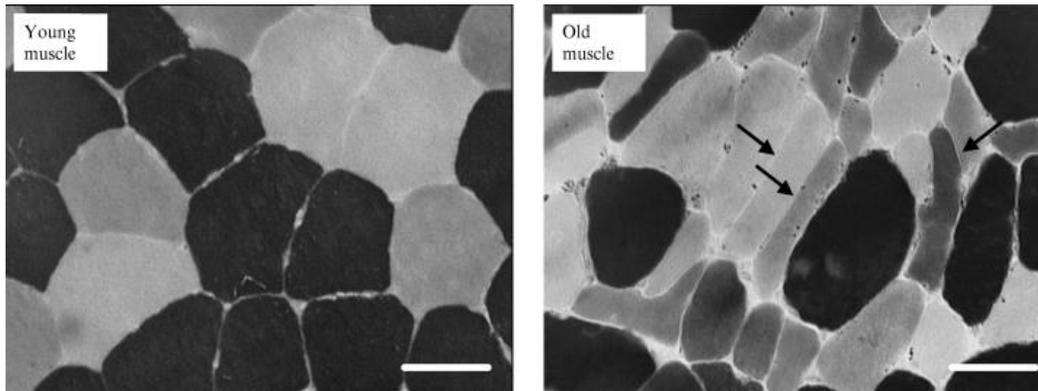


Figure 3. Fibre type shapes are often different in young and very old human skeletal muscle. Muscle fibres in the young muscle most often appear angular with four to 6 “angels” or “corners”, whereas many fibres in the elderly muscle appear as if they have been “flattened” or “crushed”. This flattening of the fibres is much more pronounced among the Type II fibres than the Type I fibres. Dark fibres = Type 1 fibres; White fibres = Type IIA; Grey fibres = Type IIX (or I/IIA) fibres; Bar = 50 μ m. ATPase staining (pH 4.6). *Used with permission from Anderson (2003).*

Ageing and Skeletal Muscle Adaptation

Ageing is associated with a progressive decline in skeletal muscle function, eventually leading to a compromised ability to perform tasks of everyday living in elderly populations. The implementation of chronic habitual physical activity is recommended for its beneficial

therapeutic effects in order to attenuate many of the age-related decrements in skeletal function that manifest in sedentary ageing populations. However, whilst exercise is known to promote a plethora of beneficial effects in ageing populations, it also appears that ageing is associated with compromised adaptive responses to acute and chronic exercise stressors compared to young adult populations (Cobley et al., 2014; Radak et al., 2013). At a molecular level, there appear to be many factors that help explain the age-associated blunted responses to both acute and chronic exercise. Prior to discussing the implementation and benefits of physical activity on skeletal muscle structure and function in relation to ageing, a brief overview of how ageing affects the adaptive responses to exercise will be reviewed.

It is known that acute exercise disrupts cellular homeostasis and increases the generation of reactive oxygen and nitrogen species (ROS/RNS), both of which are associated with redox signaling and oxidative stress (Cobley et al., 2015a; Radak et al., 2013). Redox signaling is responsible for inducing chronic adaptations to exercise training, whilst also providing protection against exercise induced oxidative stress, particularly cellular damage to DNA and mitochondria (Jackson & McArdle, 2011; Cobley et al., 2015b; Powers and Jackson, 2008). It appears that ageing causes a disruption to redox signaling, which blunts the redox regulated adaptations to acute and chronic exercise, whilst also compromising DNA repair, especially in sedentary populations (Jackson & McArdle, 2011). The precise mechanisms are not fully understood, however, may be partially explained by the resting redox status of older individuals.

Ageing is associated with altered antioxidant enzyme levels and greater redox disturbance at rest, characterized by chronically elevated basal levels of ROS and RNS in sedentary aged compared to habitual activity matched young populations (Radak et al., 2011). The implications for adaptations to exercise are that ROS/RNS resting values appear to determine the magnitude of exercise-induced redox stress response (Margaritelis et al. 2014), resulting in an attenuation of ROS/RNS production in response to an exercise bout. The consequences are compromised up-regulation of key redox signaling pathways in response to an exercise stimulus, thus negatively affecting the adaptive responses to exercise (Vasilaki et al. 2002; Vasilaki et al., 2003). Chronically elevated habitual levels of ROS/RNS also increase DNA damage (Radak et al., 2011). It is the accumulation of mitochondrial and nuclear DNA damage that over time gradually compromises cellular function, and likely contributes to the pathogenesis of ageing (Radak et al., 2013). This increased nuclear DNA damage has been linked to a plethora of pathological conditions such as cardiovascular and neurodegenerative diseases (Halliwell & Gutteridge, 2007).

Lifelong training attenuates some of the aged related declines in exercise induced redox signaling, thus decreasing oxidative damage in quiescent skeletal muscle by promoting a less hostile chronic environment. The beneficial effects of chronic physical activity appear to be twofold: 1) enhanced protection of muscle cells by up-regulating DNA repair, albeit, to a lesser extent compared to younger adults (Franzke et al., 2014; Radak et al., 2011); 2) enhanced training adaptations in response to repeated bouts of exercise (Cobley et al., 2012; Egan and Zierath, 2013). However, the molecular mechanisms underpinning these beneficial exercise effects in ageing populations are yet to be fully elucidated. The more favorable redox stress response to exercise allied to its positive effects on health and wellbeing in ageing populations, provides strong justification for promoting regular physical activity as a lifelong practice. However, there is no conclusive evidence that engaging in lifelong habitual physical activity can extend lifespan in humans.

In conclusion, redox regulated adaptations to acute and chronic exercise are known to be blunted in sedentary ageing populations. Lifelong physical activity attenuates some of the aged related deleterious effects which are linked to premature mortality, however, cannot completely attenuate these, particularly in very old populations.

BENEFITS OF PHYSICAL ACTIVITY ON AGEING MUSCLE

Despite the blunted age-related molecular responses to exercise, regular physical activity is associated with a plethora of health benefits in ageing populations. The following sections provide an evidence based overview of the primary modes of voluntary and alternative exercises, which are utilized to help off-set the age-related functional deficits and ultimately the onset of sarcopenia.

Implications of Physical Inactivity

Lifelong habitual inactivity is attributable to a plethora of chronic diseases such as cardiovascular disease and type II diabetes, and can decrease average life expectancy by up to 30% compared to lifetime active individuals (Booth et al., 2011). Chronic habitual physical inactivity is known to accelerate the progression of age induced decrements in skeletal muscle structure and function, characterised by a loss of MU number and atrophy of muscle fibres, particularly type II fibres. Motor unit loss has been shown to be as high as 50-70% when comparing young and old populations (Campbell et al., 1973; McNeil et al., 2005; Tomlinson and Irving, 1977) with the greatest losses occurring from about the age of 60 years (Vandervoort, 2002). However, there are very large inter-individual variations as well as intramuscular variations. For example, regularly exercised or chronically active postural muscles (e.g., the soleus muscle which typically comprises > 80% type I muscle fibres), exhibit relatively small MU losses compared to other non-chronically active muscles or muscles containing higher portions of type II fibres. This supports the view that chronic activation of muscles, either through exercise or habitual activation, has protective effects by delaying the age associated loss in MUs (Hepple & Rice, 2015). However, for very old populations, i.e., populations in their 10th decade and above, even postural muscles which typically experience lifelong chronic activation begin to exhibit markedly higher MU losses, showing that MU loss can only be delayed with physical activity and not prevented (Hepple & Rice, 2015).

PHYSICAL ACTIVITY MODALITIES TO INCREASE FUNCTIONAL CAPACITY IN AGEING POPULATIONS

Cardiovascular (CV) Based Activities

Cardiovascular (CV) based activities such as running and cycling have been shown to be effective for slowing or reversing decrements in a plethora of indices relating to whole body function, such as aerobic capacity (Fujimoto et al., 2010). A recent review of the literature by Cadore et al. (2014) demonstrates the positive effects of CV based exercises on indices of aerobic function in frail elderly populations. However, for improving other indices such as reactive muscle strength, power and hypertrophy, CV based activities may have limited effects (Vigorito and Giallauria, 2014). That said, CV based activities are still likely to induce some hypertrophy, especially in the form of high intensity interval training (HIIT) (Zembroń-Lacny et al., 2014). These are important considerations, because as previously discussed, insufficiencies in indices of strength and power can have significant ameliorating implications on activities of daily living and risks of falls in frail elderly populations. It has also been shown that combining CV based exercises with resistance training (RT) is more effective than CV alone for improving certain indices relating to strength and power production (Cadore et al., 2014), especially as CV based activities alone may actually hinder improvements to some of these vital indices of functional capacity in ageing populations (Hunter et al., 2004).

Resistance Training (RT)

For certain populations such as the frail elderly, progressive RT is likely to be the most effective modality for stimulating muscle hypertrophy, strength and power, while also enhancing energy expenditure and favourably altering body composition (Hunter et al., 2004; Haykowsky et al., 2005; Dodds and Sayer, 2014). Progressive RT in older populations has been consistently shown to induce substantial increases in muscle strength, with more moderate increases (5-10%) generally found in muscle CSA (Vandervoort, 2002). This suggests that the majority of the strength gains result from neural adaptations, although decreased co-activation of antagonist muscles may also contribute (Degens et al., 2009). For a comprehensive overview of previous studies that have investigated the effects of RT on muscle activation and size in ageing populations, please refer to two recent reviews and meta-analyses by Peterson et al. (2011) and Arnold & Bautmans, (2014).

Another important consideration is that heavy RT is likely to induce greater regular activation of the higher threshold type II MUs, which are most susceptible to atrophy, cell death and fibre denervation due to extended periods of disuse in aged sedentary populations. Importantly, the preferential atrophy of type II fibres has the greatest implications on the ability of the muscles to generate power, (Hunter et al., 2004), which likely impacts greatest on activities of daily living, as most typically involve dynamic movements (Vandervoort, 2002). Some of the other purported benefits of RT include: increased oxidative phosphorylation (Parise et al., 2005a); decreased insulin resistance (Misra et al., 2008); decreased mitochondrial dysfunction (Johnston et al., 2008); increased antioxidant enzyme activity (Parise et al., 2005b). However, the discussion of these purported benefits is beyond the scope of this chapter.

Table 1. Sample 6-month progressive resistance exercise model for healthy, older adults. Used with permission from Peterson (2010)

Training dosage	Wk 1-8			
	Wk 1-2	Wk 3-4	Wk 5-6	Wk 7-8
Volume (number of sets/muscle group)	1	1	1-2	1-2
Intensity (training load)	Body weight - 30% 1RM	Body weight - 50% 1RM	Body weight - 50% 1RM	Body weight - 60 %1RM
Frequency/split	1-2/full body	1-2/full body	2/full body	2-3/full body
Training agenda	Acclimation/familiarization	Acclimation/familiarization	Acclimation/familiarization	Muscular endurance
Movement tempo	Slow	Slow	Slow	Slow
Rest break between sets (s)	N/A	N/A	60-90	60-90
Mode (exercise choices)	Body weight; postural/stabilization; selectorized machines	Body weight; postural/stabilization; selectorized machines	Body weight; postural/stabilization; selectorized machines	Body weight; postural/stabilization; selectorized machines
Volitional fatigue	No	No	No	No
Training dosage	Wk 9-16			
	Wk 9-10	Wk 11-12	Wk 13-14	Wk 15-16
Volume (number of sets/muscle group)	2	2	2	2-3
Intensity (training load)	60-70% 1RM	65-70% 1RM	70% 1RM	75% 1RM
Frequency/split	2-3/full body	2-3/full body	3/full body	3/full body
Training agenda	Muscular endurance and hypertrophy	Muscular endurance and hypertrophy	Muscular hypertrophy and strength	Muscular hypertrophy and strength
Movement tempo	Slow to moderate	Slow to moderate	Slow to moderate	Slow to moderate
Rest break between sets (s)	90	90	90-120	90-120
Mode (exercise choices)	Postural/stabilization; selectorized machines	Postural/stabilization; selectorized machines; free weights	Postural/stabilization; selectorized machines; free weights	Postural/stabilization; selectorized machines; free weights
Volitional fatigue	No	No	Near	Near

Table 1. (Continued)

Training dosage	Wk 17-24			
	Wk 17-18	Wk 19-20	Wk 21-22	Wk 23-24
Volume (number of sets/muscle group)	2-3	2-3	3	3
Intensity (training load)	75-80% 1RM	80-85% 1RM	85% 1RM	85% 1RM
Frequency/split	3/full body	3/full body	3/full body	3/full body
Training agenda	Muscular hypertrophy and Strength	Strength	Strength	Strength
Movement tempo	Moderate	Moderate	Attempt fast	Attempt fast
Rest break between sets (s)	120-300	120-300	120-300	120-300
Mode (exercise choices)	Selectorized machines; free weights			
Volitional fatigue	Near	Yes	Yes	Yes
Progression/manipulation in training volume, intensity, frequency, mode, repetition tempo, and rest interval are designed to coincide with improvements in muscular fitness and performance.				
N/A = not applicable; RM = repetition maximum.				

Volume: The number of RE sets for a given muscle group, per training session. **Intensity:** Resistance load that corresponds with a maximal number of repetitions (RM) (eg, 10RM: load that corresponds with approximately 10 allowable repetitions). **Frequency:** The number of times per week each muscle group should be trained. **Split:** The general partitioning of RE for specific body parts (e.g., Full Body: resistance exercises are performed for all major muscle groups in a given session). **Training Agenda:** The respective purpose (or goal) for a given period of RE (i.e., Familiarization: A period of time devoted to gaining familiarity with the resistance exercises, as well as general physiological adaptation). **Rest Period between Sets:** The minimum amount of time devoted to rest/recovery between successive sets of RE for a given muscle group. **Mode:** The type of RE movements and loading parameters. *Body weight* RE comprises movements in which the patient's body mass is used as resistance (eg, calisthenics including body weight chair stand, squat, lunge, supine hip extension raises, etc.). *Postural/Stabilization* exercises are specific isometric postural and dynamic exercises (eg, forward and lateral planks, trunk curl-ups, supine straight-leg hip flexion, etc.) intended to improve low back health, posture, and joint stabilization. *Selectorized machines* represent standard resistance exercise machines (eg, Cybex, Nautilus, FreeMotion, etc.). *Free weight* exercises take place through the use of free-moving implements (eg, barbell chest press, dumbbell biceps curl, etc.).

There are well established RT guidelines for ageing populations developed by the American College of Sports Medicine (ACSM) and American Heart Foundation (AHA) for increasing muscle strength and fitness (Nelson et al., 2007). However, Peterson and Gordon (2011) have recommended that progressive periodized training programmes that emphasize increases in muscle strength and hypertrophy are particularly beneficial for ageing populations. These authors emphasize the need to incorporate higher intensities and volumes in an incremental fashion to achieve the greatest absolute and relative improvements in strength and hypertrophy. For a sample 6-month progressive RT programme from Peterson (2010), see Table 1.

Alternative Exercise Modalities

Despite inactivity being known to exacerbate the age associated reductions in human functional capacity, there is still a very high prevalence of habitual inactivity among ageing populations. While this may be a voluntary lifestyle choice for many due to factors such as lack of motivation etc., for many others, their habitual inactivity may be a result of barriers to performing voluntary exercises due to immobility or other pathological conditions. This has led to investigations into alternative modalities of exercise such as neuromuscular electrical stimulation (NMES) and whole body vibration (WBV). (Zembroń-Lacny et al. 2014).

Neuromuscular Electrical Stimulation (NMES)

Unlike for normal voluntary activation of muscle, which follows a size order effect, with NMES the activation of MUs is non selective, with those in closest proximity to the electrodes being activated regardless of size (Gregory and Bickel, 2005). This is important as it is the larger type II MU's in ageing populations that undergo the greatest degree of atrophy due to disuse, especially as threshold levels are normally not reached during habitual sedentary activities. Whereas, type II MUs can easily be activated using NMES, even at low intensities, especially as type II fibres are often more superficially located within muscle, and thus located closer to the electrodes (Knight and Kamen, 2005).

The use of tetanic NMES (60 Hz @ 500 μ s, with a 3sec ON:OFF of 1:1) has recently been shown to acutely increase muscle protein synthesis in sedentary aged (70.3 ± 2.4 yrs) type 2 diabetic individuals when used for 60 min (Wall et al., 2012). There is also good evidence showing high intensity NMES to be effective for increasing strength in various populations such as healthy sedentary (Banerjee et al., 2005; Bax et al., 2005). However, because the recommendation for optimal increases in strength, power and hypertrophy using NMES, is for the intensity to be applied as high as is tolerably possible, this precludes its use in certain populations such as sarcopenic elderly, as such intensities cannot always be tolerated due to the associated discomfort (Maffiuletti, 2010). However, with advancements in NMES technology, greater muscle mass activation at a given intensity is now possible (Malone et al., 2014), which may help overcome this problem in the future, although more research is needed.

Whole Body Vibration (WBV)

Over the past two decades, WBV training has been incorporated into training programmes across a diverse range of populations including the elderly (Hawkey et al., 2015). In sarcopenic populations, this modality of exercise has been promoted as a time efficient and practical alternative for individuals who are unwilling or unable to engage in conventional physical activity (Kemmler & von Stengel, 2012). Whilst evidence of the positive effects of WBV on indices of strength and power across a range of populations is inconclusive, it appears that the elderly derive the greatest benefits from WBV exercise programmes for promoting indices of strength and power (Kemmler & von Stengel, 2012; Kemmler et al., 2014; Maffiuletti and Cardinale, 2011). However, findings from other studies are less convincing (Corrie et al., 2015; Gómez-Cabello et al., 2013). Therefore, more research is needed in this population group before a more definite consensus can be derived.

Possible Contraindications to Physical Activity

Whilst physical activity, especially RT, has been shown to promote beneficial effects on muscle structure and function in ageing populations, there is emerging evidence from animal studies to show that initiating physical activity in previously sedentary very old age populations where significant MU remodeling has already occurred, may in fact increase denervation and muscle atrophy (Hepple & Rice, 2015). This has yet to be fully verified in human populations, and therefore at present is still speculative. However, based on findings from animal models, initiating physical activity in very old populations where there is substantially decreased plasticity of the MU's, may result in an overwhelming of the remodeled surviving MUs, thus exacerbating their loss (Hepple & Rice, 2015). This therefore may add to the strong justification for promoting physical activity as a lifelong practice, rather than just trying to implement it late in life, when individuals have already succumbed to many of the maladies associated with ageing and a chronic habitual sedentary lifestyle.

CONCLUSION

Ageing is associated with a progressive decrease in skeletal muscle function, characterised by MU losses, muscle fibre atrophy (particularly type II) and fibre type grouping. Sarcopenic individuals display a compromised ability to perform everyday activities of daily living and are at increased risk of accidental falls, mainly due to decrements in muscle strength and power. The underlying signature of ageing, as manifested by perturbed redox homeostasis, leads to a blunting of acute and chronic redox regulated exercise adaptations. Impaired redox regulated exercise adaptations are mechanistically related to altered exercise-induced ROS/RNS generation and a resultant failure to properly activate redox regulated signaling cascades. Despite the specific impairments in redox signaling associated with ageing, physical activity, especially progressive RT or combined CV & RT, induces a plethora of beneficial effects. However, to derive the greatest long-term benefits of physical activity for slowing or reversing age-related decrements in skeletal muscle structure and function, it needs to be promoted as a lifelong habitual practice rather than just implemented in later life.

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