The effect of ageing and strength training on skeletal muscle

Ray Marks

The present review describes several age related changes in the neuromuscular system of humans and animals which may underlie the marked strength decline of ageing muscle. Studies describing the effects of resistance training on the muscle strength of ageing humans and animals are also reviewed. From a survey of these studies, the strength decline with age appears to be due, in part, to a loss of muscle mass. While not conclusive, the effect of resistance training has been to attenuate the extent of the atrophy occurring with age, and to improve strength. A better understanding of the neuromuscular mechanisms of ageing, as well as the adaptive response of ageing mammalian muscle to resistance training, could enable physical therapists to critically evaluate the merits of strength training intervention for improving the physical ability of an ageing individual.

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R Marks, BSc, BScPT, MScPT, Clinical Associate, Department of Physiotherapy, St Michael's Hospital, Toronto, Ontario M5B 1W8, Canada

Supported by The Arthritis Society, Toronto, Canada.

Correspondence: 60 Charnwood Place, Thornhill, Ontario L3T 5H3, Canada

It has been stated that exercise training can attenuate or even reverse many adverse effects of ageing which reduce the physical activity level and the quality of life of elderly persons (Lampman 1987). One form of exercise that is often advocated for counteracting the physical decline of elderly persons is resistance training. This is because sedentary persons over the age of 70 often demonstrate about a 35 to 59 per cent decrease in maximal static (Davies and White 1983, Murray et al 1985, Young et al 1984), and dynamic strength (Borges 1989, Harries and Bassey 1990, Murray et al 1980, 1985), as well as a 25 to 33 per cent reduction in total muscle mass (Lexell et al 1983, Young et al 1984).

To examine whether exercise training is a viable modality for improving the strength and physical function of the elderly, the present review on ageing will focus on two questions. Firstly, on the nature of the ageing process in the neuromuscular system and how the age changes could contribute towards muscle weakness in the elderly, and on the efficacy of resistance training for increasing the strength of an ageing individual. Although human ageing and training adaptations may differ in many respects from those of other species due to differences in lifespan and muscle fibre composition (Fitts et al 1984, Schmalbruch and Kamieniecka 1974), it is hoped that by presenting material from both sources, a more composite understanding of the topic will be provided.

Mechanisms contributing to muscle weakness with age

Neurogenic adaptations – *Spinal cord and peripheral nerves*

Age related changes in the spinal cord have been studied since the 19th century using electrophysiological and morphological methods. In addition to a 32 per cent decline in the numbers of myelinated fibres in human spinal roots after the third decade (Gardner 1940), and an age related shift to smaller diameters in the calibre spectrum of nerve fibres in the dorsal and ventral roots Rexed (1944), Cottrell (1940) and Swallow (1966) found a decline in the numbers of myelinated fibres in human peripheral nerves with increasing age. As well, studies of rodents (Johnson and Herner 1972, Pestronk and Drachman 1978, Wright and Spink 1959) and humans (Brown 1972, Campbell et al 1973, Carlson et al 1964, McComas et al 1973, Scheibel 1979, Tomlinson and Irving 1977) have indicated that there is probably a progressive loss of motorneurones with advancing age.

Because muscles are activated by motorneurones and maximal force production requires activation of the whole motorneurone pool (Sullivan et al 1986), a progressive loss of motorneurones could impair maximal force production. In addition, coupled with an age related decrease of the trophic influence from the remaining motorneurones (Engel 1970), the

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demyelinisation of spinal and peripheral nerves may result in denervation of some muscle fibres with subsequent atrophy and weakness of the motor responses. Nevertheless, since denervated muscle fibres may be reinnervated and incorporated into viable motor units over time by means of axonal sprouting (Stahlberg and Fawcett 1982), strength assessed clinically may be maintained, as shown by Brown (1972), despite any age related motorneurone dysfunction. In old animals however, axonal sprouting may be deficient (Pestronk and Drachman 1978, Rosenheimer and Smith 1985), and therefore the capacity to innervate previously denervated muscle fibres may be impaired (Drahota and Gutmann 1961). In fact, some fibres may not be reinnervated at all, leaving them permanently denervated (Jay and Barald 1989). Furthermore, the extra burden placed on the remaining nerve cells by reinnervation processes may hasten their degeneration and, ultimately lead to muscle wasting and weakness when the pool of viable motorneurones is critically reduced (Vandervoort et al 1986).

A selective loss of large motorneurones which innervate fast-twitch (type II) fibres would be correlated with a loss of maximal strength, since these fibres are recruited for high strength activities (Larsson 1982). As well, because nerves control the contractile properties of skeletal muscles (Buller et al 1960), reinnervation of fast-twitch fibres with nerve endings from remaining small anterior horn cells to produce groups of slow-twitch (type I) fibres (Jennekens et al 1971, Stalberg et al 1989) may result in further weakness since the fibres have a low peak force.

In addition, vascular insufficiency due to a generalised arteriosclerosis may produce disturbances in the neural microcirculation of older persons further diminishing the reinnervative capacity of the ageing motorneurones (Vandervoort et al 1986). Also, because proteosynthesis may be missing or delayed as a result of ageing (Gutmann et al 1968), surviving motorneurones may have a limited capacity to reinnervate denervated fibres, resulting in their permanent replacement by non-contractile connective tissue (Lexell et al 1983). It has also been established (Drahota and Gutmann 1961) that even after reinnervation has started, protein metabolism in senile muscle may be very slow, potentially impairing the turnover of myofibrillar proteins and enzymes required for force production.

Table 1.
Morphology in ageing human muscles.

Authors	Sample	Muscles	Findings
Aniannson et al (1986)	23 women (73-83yrs)	Vastus lateralis Biceps brachii	FT fibre atrophy
Grimby et al (1982)	24 men, women (78-81yrs)	Vastus lateralis	FT fibre atrophy
		Biceps brachii	Neuropathy
Jennekens et al (1971)	8 men, women (65-92yrs)	Deltoid Biceps brachii Rectus femoris Gastrocnemius	FT fibre atrophy ST fibre atrophy Neuropathy Myopathy
Larsson et al (1978)	55 men (22-65yrs)	Vastus lateralis	FT fibre atrophy FT fibre loss
Lexell et al (1983)	6 men (70-73yrs)	Vastus lateralis	Fibre loss Muscle atrophy
Tomonaga (1977)	79 men, women (50-90yrs)	Deltoid Vastus lateralis Gluteus medius Gastrocnemius	Neuropathy Myopathy FT fibre atrophy ST fibre atrophy

Abbreviations:
FT = fast twitch
ST = slow twitch

Neuromuscular junction

Profound abnormalities at the neuromuscular junction have been observed in ageing rodents (Gutmann et al 1968, Kelly 1978, Pestronk and Drachman 1978, Robbins and Fahim 1985, Rosenheimer and Smith 1985) and in humans (Oda 1984, Tomonaga 1977). The degenerative changes are probably caused, in part, by diminished axonal transport of proteins and enzymes to the ageing end-plates (Robbins and Fahim 1985). This may result in a gradual withdrawal of the terminal axon (Gutmann and Hanzlikova 1972, 1973) and a reduction in nerve terminal area (Tomonaga 1977) and number (Tucek and Gutmann 1973). An impairment of short-term synaptic functions concerned with neuromuscular transmission may also occur (Carlson et al 1964). In addition, long-term non-impulse trophic functions concerned with synthesis, transport, and release of macromolecules and/or transmitters such as acetylcholine (Gutmann and Hanzlikova 1965, Oda 1984, Smith 1979) and choline acetyltransferase may undergo profound age related changes (Tucek and Gutmann 1973, Verkhratsky 1970).

However, in spite of these changes, physiological data has shown no deficit and some increases in neurotransmitter release with age have been reported (Banker et al 1983, Rosenheimer and

Table 2.
Morphology in ageing rodent muscles.

Authors	Model	Age (months)	Muscle	Findings
Caccia et al (1979)	Rat	30	Extensor digitorum longus, Soleus	15% fibre loss Neuropathy
Daw et al (1985)	Rat	27	Extensor digitorum longus	4.2% fibre loss
			Soleus	5.6% fibre loss
Larsson and Edstrom (1986)	Rat	20-24	Extensor digitorum longus	No atrophy No fibre loss
			Soleus	FT fibre loss FT fibre atrophy
Hooper (1981)	Mouse	16	Sternomastoid	Compensatory hypertrophy
			Biceps brachii, Tibialis Anterior	Fibre loss Compensatory hypertrophy
Rowe (1969)	Mouse	25	Sternomastoid	FT fibre atrophy Fibre loss
			Tibialis anterior	Compensatory hypertrophy

Abbreviation:
FT = fast twitch

Smith 1990). Older persons also seem to retain the ability to fully activate their motor units (Brown et al 1990, Vandervoort and McComas 1986). The short-term tetanic responses and the amplitude of miniature end-plate potentials of muscle may not change appreciably with age (Banker et al 1983), suggesting that profound structural changes at the neuromuscular junction are not functionally significant or are well compensated. Nonetheless, findings of an age related reduction of large end-plate areas, coupled with a reduction in acetylcholine storage and release suggests age changes at the neuromuscular junction could contribute towards progressive denervation of the fast muscle fibres (Gutmann and Hanzlikova 1965, Verkhratsky 1970). Ultimately this may result in a loss of larger motor units. According to Campbell et al (1973) it is the loss of larger motor units which causes weakness with age.

Myogenic adaptations – *Muscle morphology*

Measurements of total excretion of creatinine from healthy young and older persons ranging in age from 20 to 97 years have provided indirect evidence that muscle mass is reduced by about one third over the age of 50, with a further reduction of about 15 per cent between the ages of 70 to 80 years as indicated by whole-body counting of potassium (Grimby and Saltin 1983). Significant reductions of this magnitude have also been reported by Young et al (1984) who used a technique of ultrasound scanning to measure cross-sectional areas of the quadriceps in young and older women. A decline in muscle mass could be indicative of either an age related loss of muscle fibres, a decrease in fibre size, or a loss of entire motor units (Aniansson et al 1981).

In specimens of surgically isolated vastus lateralis, the muscle cross-sections of older men, mean age 72 years, were 18 per cent smaller, and contained 25 per cent fewer fibres than those of younger individuals with a mean age of 30 years (Lexell et al 1983) shown in Table 1. With no significant age differences in fibre size or fibre type proportion, it appears that ageing atrophy in the vastus lateralis muscle of a group of healthy 72-year-old men was due to a loss of fibres. A reduction in fibre number of the order of 25 per cent would be consistent with studies reporting the magnitude of the quadriceps strength loss at 70 years (Vandervoort et al 1986).

However, a later study including 43 men, aged between 20 and 80 years (Lexell et al 1988) indicated that in addition to a loss of fibres, ageing atrophy may be caused by a reduction in fast-twitch fibre size. The findings concurred with those of Grimby et al (1982) who observed a 10 to 30 per cent fibre size decrease in vastus lateralis muscle samples from subjects ranging in age from 78 to 81 years, with the decline predominating in the fast-twitch fibre population. Nonetheless, a reduction in fibre size may be moderate in comparison to the muscle volume reduction with age (Grimby and Saltin 1983) and therefore may play a somewhat lesser role than a loss of fibres in decreasing muscle strength with age. Furthermore, the absence of any age related fibre size changes in samples of the biceps brachii (Grimby et al 1982) seems to suggest that the mechanism underlying the onset of weakness with age may be unrelated to a decrease in fibre size.

In examining the relationship between decreases in vastus lateralis fibre size and age changes in knee extensor muscle strength over a seven year period, Aniansson et al (1986) found no

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correlation between the 10 to 22 per cent reduction in muscle strength and the decreased fast-twitch fibre areas of the muscle samples. Stalberg et al (1989) reported that despite a decrease in mean quadriceps fibre size of about 16 per cent in males and about 26 per cent in females by the age of 70 years, the decrease accounted for only 4 to 9 per cent of their knee extensor strength loss. Aniansson et al (1981) actually observed a negative correlation between the torque measurements and relative areas of the fast-twitch oxidative fibres of the knee extensors of men, of mean age 70 years. The apparent discrepancy between the strength decline and the fast-twitch fibre size was thought to be the result of an altered motor recruitment pattern. However, perhaps the fast-twitch fibre density was greater in those muscles with smaller fast-twitch fibre areas which resulted in greater torque. At all ages, Lexell et al (1988) found the slow-twitch fibre size of vastus lateralis samples inversely related to the total number of muscle fibres.

With increasing age, histochemical reactions have revealed changes in fibre type distribution characterised by a decrease in the percentage of fast fibres (Scelsi et al 1980) and a concomitant high content of intermediate fast-twitch oxidative-glycolytic or type IIa fibres (Salviati et al 1984). Such changes in fibre composition, could cause a decrease in strength with age. However, Clarkson et al (1981) found no correlation between vastus lateralis muscle fibre composition and isometric knee extensor strength measurements of 15 subjects, having a reported mean age of 64.5 years. Similarly, Lexell et al (1988) and Aniansson et al (1986) found no significant change in vastus lateralis fibre composition with increasing age to account for the age-associated decrease in the strength of the muscle. Furthermore, although Aniansson et al (1981) found evidence of selective fast-twitch fibre atrophy in vastus lateralis biopsies of older women, as did Larsson et al (1979) in older men, the subjects' quadriceps strength did not correlate with the percentage of fast-twitch fibres or fast-twitch fibre subgroups of the muscle samples.

Table 3.
Biochemistry of ageing human muscles.

Authors	Sample	Muscles	Findings
Aniannson et al (1981)	65 men, women (61-76yr)	Vastus lateralis	Myosin ATPase unchanged
Aniannson et al (1986)	23 men (73-83yr)	Vastus lateralis Biceps brachii	LDH, myokinase unchanged
Borges and Essen Gustavsson (1989)	10 men, women (70yr)	Vastus lateralis	Myokinase reduced
Grimby et al (1982)	24 men, women (78-81yr)	Vastus lateralis Biceps brachii	Anaerobic enzymes maintained
Larsson et al (1978)	55 men (22-65yr)	Vastus lateralis	ATPase, myokinase unchanged LDH reduced
Taylor et al (1984)	12 subjects (70-80yr)	Flexor digitorum superficialis	ATP, Pi, PC unchanged

Abbreviations:
ATP = adenosine triphosphate
Pi = inorganic phosphate
LDH = lactate dehydrogenase
PC = phosphocreatine

Age related signs of denervation, such as fibre type grouping and slow-twitch fibre predominance, as well as myopathic manifestations such as necrosis, myofibrillar disorganisation, and cell infiltration have been found in older persons who display muscle weakness (Jennekens et al 1971, Serratrice et al 1968, Tomonaga 1977) and in nonwasted subjects, aged 65-89 years, with no suspected neuromuscular disease (Scelsi et al 1980). However, the absence of neuropathic alterations in vastus lateralis muscle samples of 70 year old persons (Aniansson et al 1981), and the rarity of myopathic changes in the normally aged muscle (Aniansson et al 1981, Grimby et al 1982, Scelsi et al 1980) speaks against a denervation process or myopathy being the major cause for reduced muscle strength in the elderly.

Studies of senile animal muscles (see Table 2), have suggested that the predominant morphological changes occurring in ageing rodent muscle are due to neurogenic phenomena. However, based on samples of the extensor digitorum longus, soleus and diaphragm muscles of young (3 months) and senile (28-36 months) rats, Bass et al (1975) suggested that no generalisations on age change in older muscle should be made on the basis of a study limited to one type of muscle. Additionally, because there have been few attempts to correlate morphological alterations in animal senescent muscle with physiological data, it is difficult to arrive at a consensus regarding the mechanism responsible for the onset of muscle weakness with advancing age.

Eddinger and colleagues (1986) examined the mechanical and contractile properties of fibre bundles and skinned fibres of the extensor digitorum longus and soleus muscles of young adult (9 months) and senescent

Table 4.
Biochemistry of ageing rodent muscle

Author	Model	Age (months)	Muscles	Finding
Bass et al (1975)	Rat	28-36	Extensor digitorum longus	TPDH, LDH, GPDH reduced
			Soleus	TPDH, LDH, MDH, CS reduced
			Diaphragm	TPDH, GPDH reduced
Schmukler and Barrows (1966)	Rat	24-30	Not specified	LDH, MDH reduced
Syrovy and Gutmann (1977)	Rat	24-36	Extensor digitorum longus	Myofibrillar, myosin ATPase reduced
			Soleus	

Abbreviations:
TPDH = triphosphate dehydrogenase
LDH = lactate dehydrogenase
GPDH = c-glycerol-3-phosphate dehydrogenase
MDH = malate dehydrogenase
CS = citrate synthetase
ATPase = adenosinetriphosphatase

(30 month) Fisher 344 rats. Possible effects on the mechanical properties due to age related changes at the neuromuscular junction were averted by direct electrical stimulation of the fibre bundles and by addition of curare to the bathing solution. The isometric tension generated by the extensor digitorum longus fibres remained unchanged with age and there were no age-associated decreases in muscle fibre number or area, based on myosin-ATPase histochemistry. Contrary to expectation, the tension generated by the soleus bundles and fibres increased significantly with age, while showing an increased percentage of type I (slow-twitch) fibres and fibre area. Thus, in a situation in which one might have expected a decline in tension or increase in percentage of type II fibre area, since type II fibres produce more tension per cross-sectional area, the opposite was found. Although, an increase in strength with age would be consistent with an increase in type I fibres in the rat where fibre properties are reversed, Eddinger et al (1986) classified their type I fibres as slow-twitch. Despite this confusion, the findings concurred with those of Kugelberg (1976) and suggested that changes in the muscle, such as changes in the contractile proteins or in the mechanism of excitation-contraction coupling, are not responsible for the widespread finding of weakness in old age.

Muscle biochemistry

The usable form of chemical energy required for force production, adenosine triphosphate (ATP) is provided through three interrelated biochemical processes. In addition to small amounts available within the muscle, the most readily available supply involves the breakdown of phosphocreatine and the reformation of the molecule from adenosine diphosphate. The second, anaerobic glycolysis, involves the metabolic breakdown of glucose into lactic acid, and the third system involves the complete oxidation of glucose (Sullivan et al 1986).

Animal studies have shown decreased levels of the substrate phosphocreatine (PC) (Syrovy and Gutmann 1977), as well as decreased levels of the enzymes lactate dehydrogenase, myosin ATPase, and myofibrillar ATPase (Bass et al 1975) with age. In humans, the enzymes myokinase (Borges and Essen-Gustaffson 1989) and lactate dehydrogenase (Larsson et al 1978) have also been found to decrease significantly with age, (see Tables 3 and 4). As indicated by abnormalities in the pattern and content of the energy-rich phosphogens in several clinical disorders, a decrease in phosphogen substrate and/or a decrease in enzymes involved in the breakdown of ATP might impair muscle force production (Moller et al 1980). According to Edwards (1978), muscle weakness will also occur if the contractile material is inadequately supplied with energy requirements.

However, Orlander et al (1978) examined the enzyme composition of vastus lateralis muscle homogenates of sedentary men aged 20 to 65 years and found no significant correlations with age for phosphofructokinase, lactate dehydrogenase, citrate synthetase or cytochrome oxidase, while 3-hydroxyacyl-Co A dehydrogenase, an oxidative enzyme, actually increased significantly with age. Based upon these observations, it was clear that the muscle strength loss occurring up to the age of 65 years, could not be adequately explained in terms of deteriorating skeletal muscle energy metabolism.

Likewise, Aniansson et al (1981) found no correlation between the phosphogen (ATP and CP) and the enzymatic (Mg^{2+}-ATPase, myokinase and lactate dehydrogenase) content of vastus lateralis samples of elderly men (66 to 76 years) and women (61 to 71 years) and their maximal isometric and isokinetic quadriceps torque

Table 5.
Strength training effect in ageing individuals.

Authors	Sample	Muscles trained	Training intensity and mode	Training sessions per wk	Training duration (wks)	Percent strength gain
Aniansson and Gustafsson (1981)	12 men (69-74yr)	Knee extensors	Moderate dynamic	3	12	14-22
Brown et al (1990)	14 men (60-70yr)	Elbow flexors	Moderate-high dynamic	3	12	48
Frontera et al (1988)	72 men (60-72yr)	Knee flexors, extensors	High dynamic	3	12	226
Kauffman (1985)	10 women (65-73yr)	Little finger abductors	High isometric	3	6	60
Larsson (1982)	6 men (56-65yr)	Knee extensors	Low dynamic	2	15	7
Moritani and deVries (1980)	5 men (67-72yr)	Elbow flexors	Moderate dymamic	3	8	22

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measurements. It was therefore concluded that factors other than changes in muscle biochemistry must account for the decline of force with age.

Taylor et al (1984) employed 1311 P nuclear magnetic resonance to compare intracellular pH and concentrations of ATP, PC, and inorganic phosphate of the elbow flexors of young adults with those of healthy elderly subjects aged 70 to 80 years. No group differences at rest were observed and dynamic aerobic exercise resulted in a similar reduction of PC in both groups with no change in ATP concentration. Furthermore, neither the fall in pH with exercise, nor the time to resynthesise ATP in the recovery period, as measured by time to replete PC, were significantly different in the two groups. Again the results suggested that ageing does not affect the metabolic properties of human skeletal muscle. Therefore the strength decrement demonstrated by elderly cohorts is unlikely to be attributable to alterations in ATP production or utilisation.

In a prospective study of ageing men conducted over a seven year period, two enzymes involved in the replenishment of ATP, lactate dehydrogenase and myokinase remained unchanged despite an overall 10 to 22 per cent reduction in torque (Aniansson et al 1986). The lack of correlation between the subjects' muscle torque and the muscle enzymatic activity suggested that the strength loss had occurred independently of a change in muscle metabolism.

In summary, age related neuromuscular changes in humans and rodents have been studied using a variety of anatomical, histological, and electrophysiological techniques. The results of these investigations are variable and the interpretation of the data difficult in some cases, because of the lack of correlative physiological information. However, the observations of Kugelberg (1976) suggest that before any loss of muscle fibres, there is a redistribution of muscle fibre type, with an increasing proportion of slow-twitch oxidative fibres. The onset of this process could be induced by a loss of motor axons (Caccia et al 1979); a change in neuromuscular transmission (Kelly 1978); or a change in motorneurone morphology (Larsson and Edstrom 1986). The poor correlation between body cell mass and fibre area, and between muscle enzymatic and phosphogen content, in contrast to the positive correlation between muscle strength and body cell mass, suggests that a reduced muscle fibre number produces muscle weakness with age (Aniansson et al 1981, Grimby et al 1982).

Muscle strengthening and ageing

Although several age related changes in the neuromuscular system may cause a loss of strength with age (see Figure 1), there is increasing evidence that voluntary exercise programs of six to 15 weeks duration will improve the maximal voluntary strength of persons ranging in age from 56 to 83 years of age (see Table 5). However, as a result of irreversible changes in the ageing neuromuscular system, the adaptive response of older individuals to strength training may be attenuated (Hettinger 1958, Kaufmann 1985, Liemohn 1975).

Moritani and deVries (1980) found that compared to young subjects who

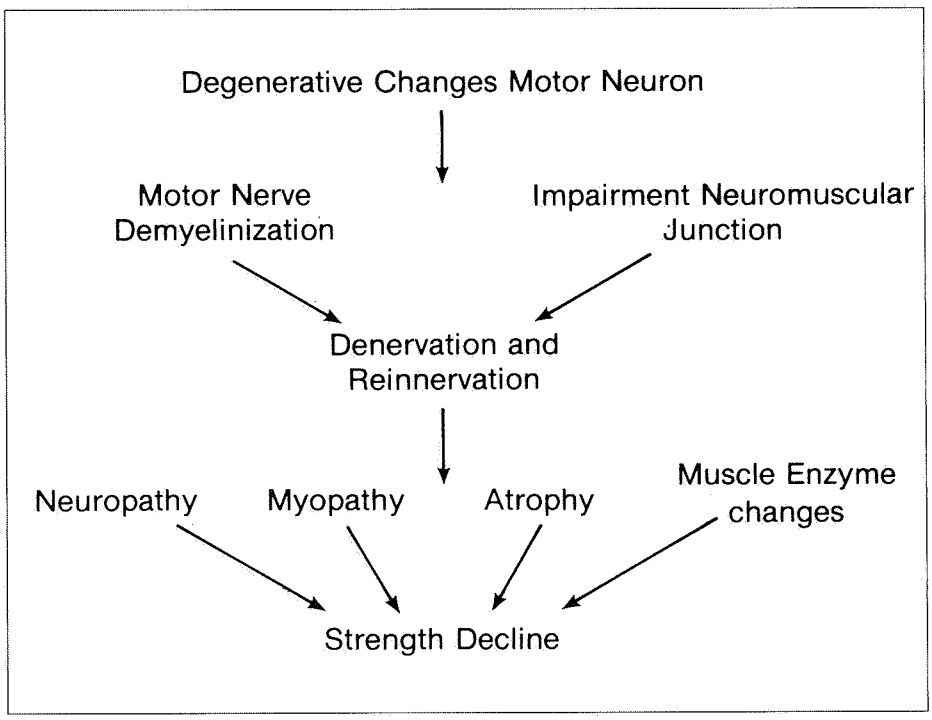

Figure 1.
Model of factors considered responsible for strength reduction with ageing.

demonstrated an isometric strength gain of 30 per cent following three-times-a week dynamic strength training for the elbow flexors, the strength increase recorded by older subjects carrying out the same protocol was only 22 per cent. Also, since the strength gain recorded by the older subjects was not accompanied by muscle hypertrophy, the investigators concluded that unlike younger individuals, the adaptive response to exercise in the elderly might rest entirely on neural factors. However, the work intensity of 66 per cent of maximal which would mostly recruit slow-twitch fibres, coupled with the short exercise period could explain why the older subjects failed to demonstrate muscle hypertrophy. Additionally, since the amount of intramuscular fat and connective tissue is known to gradually increase with age, a training-induced muscle fibre hypertrophy in old age could, correspondingly, take place at the expense of a decreased amount of fat and/or connective tissue without any visible change in the cross-sectional area of the muscle as was reported.

Alternatively, elderly persons may simply adapt more slowly than young persons to overload training. Aniansson and Gustafsson (1981) subjected nine men, aged between 69 and 74 years, to low intensity physical training which included strength training of the lower extremities for a 12 week period. They were able to demonstrate a 9 to 22 per cent increase in quadriceps strength as well as an increase in fast-twitch fibre number and relative area. The percentage increase in quadriceps strength in this study was reportedly the same as that observed after heavy strength training for seven to eight weeks in young men. Similar results were reported by Larsson (1982) who recorded a 7.5 per cent increase in the knee extensor strength of men, aged 56 to 65 years, following a twice-weekly quadriceps strengthening program. The regime was performed for 15 weeks and was accompanied by a reversal of age associated quadriceps muscle atrophy.

Frontera et al (1988) found left thigh girth and static knee extensor strength measurements were significantly greater at 12 weeks than the same measurements made following six weeks of training. The study involved 34 sessions with 24 repetitions/session at 80 per cent of the maximum force that the subject could exert on a single occasion (1 repetition maximum or 1RM) and the rate of dynamic strength gain per session was found to be similar to that reported in young men. It resulted in a 33.5 per cent increase in slow-twitch and a 27.6 per cent increase in fast-twitch fibre size. The findings suggested that adaptations to strength training occur for at least 12 weeks following the start of training. Therefore training sessions of six weeks duration may not provide sufficient time to effect an optimal adaptive response in elderly persons.

Contrary to the conclusions reached by Moritani and deVries (1980), Brown et al (1990) demonstrated that healthy men, between the ages of 60 and 70 years, retain the same potential for significant increases in strength performance and muscle hypertrophy of their elbow flexors in response to dynamic overload training as young men. On the basis of their findings, the investigators speculated that the rate of decline in strength and muscle mass in old age and the accompanying loss of independent functional capacity might be reduced or even reversed by appropriate resistance training programs.

Studies on animals have shown that old muscles can develop surgically-induced compensatory hypertrophy (Tomanek and Woo 1970) and that the diaphragm, a continuously active muscle manifests far fewer morphological changes than other skeletal muscles (Bass et al 1975). In ageing hamsters, Goldspink and Howells (1974), using a weight-lifting program, found a training-induced growth of several muscles. Nevertheless, after subjecting ageing rodents to strength training, Klitgaard et al (1989) found plantaris muscle weight loss was not completely counteracted by the exercise. Furthermore, despite the fact that twitch and tetanic tension remained

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unchanged as a result of strength training, the training became less effective in maintaining the contractile properties of the exercised muscle with increasing age.

Brown (1989) subjected 40 rats, ages 21, 24, 27 and 30 months to three sets of 10 manually resisted chin-ups, twice daily for three months. Twenty controls, five in each age group, were followed for three months but were not exercised. Before the experimental period began, the rats' left palmaris longus was removed, weighed, frozen, sectioned and stained. After the three-month period, the right palmaris longus muscle was removed and the same procedure followed. Three of the four groups of exercised rats showed an increase in muscle weight post-exercise, despite a concomitant loss of body weight during the exercise period. Significant increases in fast-twitch fibre area occurred for 21, 24, and 30 month old rats that exercised. However the magnitude of the fibre enlargement was greater for the two younger groups. There was no histological evidence of exercise-related tissue damage. The findings supported the view of a more limited response to exercise with advancing age. However, they also indicated that as opposed to forced running on a treadmill (McCafferty and Edington 1974, Steinhagen-Thiesen et al 1980), beneficial improvements might be observed in older animals following volitional exercise.

Summary and conclusion

The mechanisms underlying the loss of muscle strength with age are complex and may involve different levels of the nervous system (Larsson and Edstrom 1986). For example, a number of studies in man and other species have demonstrated an age related decline in the number of spinal motorneurones (Tomlinson and Irving 1977); an increased incidence of spinal nerve root and peripheral nerve degeneration (Cottrell 1940, Gardner 1940, Swallow 1966); a loss of axonal sprouting ability (Pestronk and Drachman 1978) and degeneration at the motor end-plates (Tomonaga 1977). However, in humans, and in animal models in particular, it has not always been possible to identify whether the changes observed at the ageing motor nerve terminals and spinal cord are due to factors other than age, such as weight loss, pathology, inactivity or a change in muscle fibre diameter (Robbins and Fahim 1985).

Muscle weakness during old age has also been attributed to a loss of muscle fibres, as is the case in human muscles (Lexell et al 1983), and in muscles of different animals (Caccia et al 1979, Hooper 1981). A progressive fall in the number of functioning motor units (Brown 1972, Campbell et al 1973, Sica et al 1974), a reduction in fibre size (Grimby et al 1982), and/or a selective loss of fast-twitch fibres (Larsson et al 1978) may also result in an age related loss of muscle strength. In addition, neuropathic manifestations such as fibre type grouping, small atrophic fibres, angulated fibres, and target fibres, as well as myopathic manifestations such as fibre necrosis, and cell infiltration described in the highest age groups could contribute to an age related reduction of muscular strength (Grimby et al 1982, Serratrice et al 1968, Tomonaga 1977). Again, in both humans and animals, it has not always been possible to isolate the morphological changes occurring in muscle due to age from those due to inactivity or pathology. It has also been shown that no generalisations can be made from the findings of a single biopsy of ageing muscle, and that changes in one muscle of an individual may differ significantly from those of other muscles within the same individual. Thus, the relative importance of the various age related changes occurring in the neuromuscular system to the loss of strength with age cannot be ascertained at the present time.

In fact, in spite of the age-associated abnormalities observed in the peripheral nervous systems of humans, a common finding is that persons over the age of 60 retain the ability to fully activate their remaining motor units (Brown et al 1990, Vandervoort and McComas 1986), suggesting that degenerative changes in the peripheral neural pathways are fully compensated for and that the decline in strength with age cannot be ascribed to altered motivation or central factors. Older persons also appear to exhibit few changes in energy supply enzymes or substrates which might undermine the contractile process.

Edgerton (1976) has summarised the results of several studies which indicate that the motorneurone cell body, which shows declining function with advancing age, may adapt favourably to overload. Although one cannot extrapolate directly from these studies of normal animals, it is known that an increase in motorneurone excitability, as evidenced by surface electromyography and percutaneous nerve stimulation will occur in humans during voluntary effort (Sale et al 1983).

Additionally, a study of motor end-plate regeneration in cat hind limbs has shown terminal sprouting which might favour reinnervation if denervated muscle is stimulated by an increased functional work load (Tuffery 1971). Again although the experiment was performed on normal animals, it is known that changes in terminal branch number would also be associated with corresponding alterations in the release of the neurotransmitter acetylcholine (Smith 1984). An increase in acetylcholine production would improve synaptic transmission, and pattern of motor recruitment (Rosenheimer 1985) which has been observed in aged men following overload training (Moritani and deVries 1980).

Training resulting in end-plate enlargement (Appell 1984) and an increased recruitment of fast motor units may lead to an age-associated increase in muscle volume (Larsson et al 1978). Although work induced hypertrophy due to muscle fibre splitting (Gonyea 1977, 1980) has not been observed in humans (MacDougall et al 1976), an increase in fast and

slow-twitch fibre cross-sectional areas after strength training (Fontera et al 1988) might be an additional factor accounting for the finding of increased total muscle area in older persons post-exercise. According to Herbison et al (1979) improvements in muscular activity as a result of increases in muscle area could, in turn, stimulate motor end-plate and lower motorneurone metabolism. This might delay or prevent further deterioration of descending nerve pathways due to age related trophic changes (Gutmann and Hanzlikova 1965, 1966, Gutmann 1970), thereby attenuating any loss of strength.

However, as a result of age differences, previous level of training, muscle studied, mode and intensity of training, method of measuring strength, and type and duration of training, the magnitude of the age related strength loss that may be overcome by training may vary considerably (see Table 5). It is also known that denervated or reinnervating muscle may be particularly sensitive to exercise and that excessive exercise may actually interfere with reinnervation (Herbison et al 1983). Nevertheless, in spite of significant increases in type IIa fibre areas, a low intensity training stimulus may not result in significant increases in static and dynamic muscle strength of aged muscle, as suggested by Larsson (1982). In contrast, the findings of Frontera et al (1988) suggest that a vigorous strength training program for higher age groups will cause a marked gain in static and dynamic strength of the exercised muscles which is accompanied by muscle hypertrophy of both fibre types and an increased rate of actomyosin turnover.

Unfortunately, although the ultimate goal of resistance training is to improve physical function and well-being, the hypothesis that increases in skeletal muscle strength in response to voluntary exercise will actually lead to such improvements has not been substantiated. In fact, neither the postulated importance of physical activity with respect to the age related decline in strength and the maintenance of posture and movement, nor the theorised relationship between muscle strength and function in older persons has been clearly demonstrated. There is also a paucity of information on the value of strength training for the frail elderly.

Physiotherapists working in the field of geriatrics could make a significant contribution in this area. This could be achieved by conducting randomised controlled investigations of the efficacy of various strength training protocols and comparing the various strength training protocols for alleviating the strength decline and improving the function of healthy and frail elderly persons of different ages. This information can also be useful in the prevention and retardation of disability due to ageing. Longitudinal studies which examine the effects of particular forms of exercise on pertinent neuromuscular mechanisms known to decline with age might also be beneficial. Because mammalian muscle differs across species, further quantitative information that will lead to greater insight into neuromuscular ageing mechanisms in human subjects is also required. The material presented in this review could be applied towards these endeavours.

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