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Aging and Muscle Function

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Summary Most of the available data on the aging of muscle function are cross-sectional in type. Static and dynamic muscle strength seem well preserved to about 45 years of age, but performance deteriorates by about 5% per decade thereafter. There is a parallel loss of lean tissue. Because muscle biopsy specimens are not always representative of an entire muscle, it is unclear whether there is a general hypotrophy or atrophy of the muscle fibres or a selective hypoplasia and degeneration of type II fibres associated with a loss of terminal sprouting. Other influences may include a deterioration of end-plate structures with impaired excitation-contraction coupling and impaired fibre recruitment. Both contraction time and half-relaxation time are prolonged with aging, and there is a decrease of maximal contraction velocity, more marked in the legs than in the arms. On the other hand, endurance at a fixed fraction of maximal force is increased. Potential factors leading to the enhanced endurance include a poorer maximal effort and an increased proportion of type I fibres; however, there is little evidence that muscle capillarity is altered. The loss of function is generally less in the arms than in the legs, but it remains unclear whether this is an inherent biological difference, or merely a reflection of differential changes in activity patterns between the upper and the lower limbs. The male/female strength ratio does not seem to change with age, but substantial slowing or reversal of the aging process is possible through appropriate activity programmes. The preservation of muscle function has important implications for the quality of life in the frail elderly, counteracting dyspnoea, stabilising joints and extending the period of independent living by up to 20 years.

1. Muscle Function

1.1 Cross-Sectional Aging Patterns

There have been numerous cross-sectional studies assessing the effects of aging on muscle function since the early work of the Belgian biometrician Quetelet (1835). Some authors have suggested a relatively constant annual loss of strength. However, most authors stress that there is apparently a plateau (or sometimes even an increase) of strength until the mid-40s, after which there is an accelerating loss of lean tissue, with associated decrements in static strength, dynamic strength and speed of movement (see the classical contributions of Asmussen 1965; Asmussen & Heeboll-Nielsen 1961; Cullumbine et al. 1950; Fisher & Birren 1947; Galton, 1884; Hettinger 1961; Quetelet, 1835; Reijs 1921; Ufland 1933; together with the more recent research of Aniansson & Gustafsson 1981; Aniansson et al. 1978, 1980a, 1981, 1983, 1986; Belanger et al. 1983; Borges 1989; Clarkson et al. 1981; Cunningham et al. 1987; Fugl-Meyer et al. 1980; Johnson 1982; Kavanagh & Shephard 1990; LaForest et al. 1990; Larsson 1978 1982; Larsson et al. 1979; Lennmarken et al. 1985; McDonaugh et al. 1984; Murray et al. 1980, 1985; Pearson et al. 1985; Petrofsky & Lind 1975a; Rikli & Busch 1986; Shephard 1991; Sperling 1980; Stalberg et al. 1989). Unfortunately, with the exception of the 7-year follow-up of Aniansson et al. (1986), there have been almost no longitudinal studies of ordinary city-dwellers of average fitness to address the potential problem of cohort differences in the physical demands placed upon the major muscles during adolescence and early adult life.

1.2 Cross-Sectional Area of Active Muscle and Strength

Larsson (1978, 1982) and Young et al. (1982, 1984) extended the classical findings of Ikai and Fukunaga (1968), noting that muscle strength was closely related to the cross-sectional area of *active* muscle, irrespective of the subject's age. This active area depends not upon the overall physical di-

mensions of the muscle, but rather upon the total available number of muscle fibres, the mean cross-sectional area per fibre, and the percentage of the available fibres that are activated. A further variable which can influence muscle strength is the type of fibre that is activated; at high velocities type II fibres develop a larger tension per fibre than do type I fibres.

The loss of muscle function with aging could thus be attributable to a decrease in the total number of fibres, a decrease in the dimensions of individual fibres, an impairment of excitation-contraction coupling and/or a decreased activation of high threshold motor units (MacLennan et al. 1980). Some authors have also suggested that aging leads to a selective loss or atrophy of fast-twitch (type II) fibres (Davies & White 1983; Davies et al. 1986; Froese & Houston 1985; Larsson 1978, 1983; Larsson et al. 1978, 1979) but others have disputed this (Essen-Gustavsson & Borges 1986; Grimby & Saltin 1983; Grimby et al. 1984; Lexell et al. 1988). If indeed such a change occurs, it remains unclear whether the cause is endogenous (for instance, a neurotrophic disturbance resembling denervation) or exogenous (particularly a decline in nerve-impulse activity related to progressive disuse and a selective decrease of high speed movements).

1.3 Variables Confounded with Aging

Whether cross-sectional or longitudinal data are examined, the aging process is inevitably confounded with any secular trend to a change of habitual physical activity. This problem is particularly evident in our on-going combined cross-sectional and longitudinal studies of muscle strength in the Inuit community of Igloolik (Rode & Shephard 1971; Shephard & Rode 1985), where most of the population, irrespective of age, have dramatically decreased their habitual physical activity over the past 2 decades.

In most studies, habitual activity is inversely related to age. It thus remains uncertain how far any age-related losses of muscle function are an inevitable consequence of aging, and how far they merely reflect the age-related decrease of physical activity. One recent cross-sectional study of masters athletes conducted by our laboratory suggested that lean tissue mass was well sustained into the seventh decade of life if subjects maintained a consistent level of physical activity (Kavanagh & Shephard 1990). Fiatarone and colleagues (1990), likewise, have noted the potential of an appropriate training programme to maintain or restore both muscle function and lean tissue even into the ninth decade of life.

The present review will examine the age-related deterioration of muscle function with particular reference to static and dynamic muscle strength, contraction speed, muscular endurance and possible variations in the rate of aging of muscle function between body parts, between sexes, and between active and inactive individuals, with a brief examination of the clinical significance of these observations.

2. Muscle Strength

2.1 Loss of Static and Dynamic Strength

Most of the classical studies examined the loss of static strength of major muscle groups, but more recently there have also been a number of cross-sectional studies of the effects of aging on isokinetic strength. Both follow a rather similar course. The overall functional loss of strength over the total span of adult life is typically 30 to 40%. However, there is little loss of strength to the age of 45 years, with an accelerating deterioration thereafter, so that there is about a 25% impairment of function by the age of 65 years (Asmussen 1980; Åstrand & Rodahl 1986; Grimby 1986; Grimby & Saltin 1983; Shephard 1987).

The loss of static and dynamic muscle strength occurs at about the same rate as the age-related decrease in overall lean tissue mass (Allen et al. 1960; Aniansson et al. 1980a 1981; Borkan et al. 1983; Fleg & Lakatta 1988; Forbes 1976; Grimby et al. 1982; Shephard 1991; Steen et al. 1977; Tzankoff & Norriss 1977). This suggests a possible causal relationship between weakening of the muscle and the decrease in mass, although the loss of strength

and tissue mass could reflect a decrease of fibre number, a decrease of average fibre size, or both.

2.2 Biochemical Changes

Muscle biochemistry has been studied more in the young-old (65 to 75 years) than in middle-old (typically 75 to 85 years) and old-old (over 85 years) individuals. In people aged 65 to 75 years, there are only minor changes of enzyme activities (table I), suggesting that there has been little change of fibre composition (table II). Nevertheless, there does seem a small trend to an increase in the activity of oxidative enzymes, and a decrease in the activity of glycolytic systems (Larsson 1978), possibly presaging the senile pattern of cellular organisation, with clusters containing a preponderance of type I fibres (Grimby & Saltin 1983; Grimby et al. 1982, 1984; Jennekens et al. 1971a,b; Johnson et al. 1973; Larsson 1983; Lexell et al. 1984, 1986; Nygaard & Sanchez 1982; Tomonaga 1977).

2.3 Fibre-Typing

Needle biopsy specimens reveal only minor changes in either muscle fibre size or the ratio of type II to type I fibre areas in the vastus lateralis, at least until the seventh decade of life (fig. 1). However, in those over the age of 70 years, there is a progressive reduction in the area of muscle occupied by type II (particularly type IIb) fibres (Aniansson & Gustafsson 1981; Aniansson et al. 1978, 1980b, 1981, 1986; Essen-Gustavsson & Borges 1985, 1986; Grimby et al. 1982, 1984; Larsson 1978, 1982, 1983; Larsson et al. 1978; McCarter 1978; Scelsi et al. 1980; Stalberg et al. 1989; Tomonaga 1977).

To date, most muscle biopsy specimens have

Table I. Changes in the enzyme activity of the vastus lateralis muscle with age. Data for untrained healthy men, with tendencies in parentheses for women

Reference	Age	n	Oxidative				Glycolytic							Mg^{++} ATP
			SDH	HAD	CYTOX	CS	PFK	PHOSP	LDH	MK	HK	TPDH	CPK	
Aniansson et al. (1980b)	16-78	113	↔				↔	↔						
Aniansson et al. (1981)[a]	66-76	47							↔	↔				↔
Aniansson et al. (1986)	73-83	22							↔	↔				
Borges & Essen-Gustavsson (1989)	20-70	14		↔		↔			↔	⇓			↔	
				(↔)		(↔)			(↔)					
Essen-Gustavsson & Borges (1986)	20-70	34		↔		↓			↔		↔	↓		
				(↔)		(⇓)			(↔)		(↔)	(⇓)		
Grimby et al. (1982)[a]	78-81	12		↔		↔			↔		↔			
Larsson (1978)	22-65	55		⇑	↑	↑	↔		↓	↔				↔
Örlander & Aniansson (1980)[a]	70-75	5		↑	↔	↔	↔		↓					

a Based on comparisons with a group of young or middle-aged subjects of the same sex (Lithell et al., 1981; Örlander et al., 1978; Saltin et al., 1980; Sjøgaard, 1981) using identical procedures for measurement of enzymatic activities.

Abbreviations and symbols: CPK = creatine phosphokinase; CS = citrate synthase; CYTOX = cytochrome oxidase; HAD = 3-hydroxyacyl-CoA-dehydrogenase; HK = hexokinase; LDH = lactate dehydrogenase; MG^{++} ATP = Mg^{++} stimulated adenosine triphosphatase; MK = myokinase; PFK = phosphofructokinase; PHOSP = phosphorylase; SDH = succinate dehydrogenase; TPDH = triose phosphate dehydrogenase; ↔ = unchanged activity; ↑, ↓ = tendency to increased or decreased activity; ⇑, ⇓ = significantly ($p < 0.05$) increased or decreased activity.

Table II. Changes in fibre-type distribution and total number of human muscle fibres with age

Reference	Muscle	Sex	Age (y)	Fibre type distribution (%)					Fibre no.
				I	II	IIA	IIB	IIC	
Aniansson & Gustafsson (1981)	VL	M	69-74	51.0	49.0	33.8	15.4		
Aniansson et al. (1978)	VL	M	70	49.4	50.6	34.4	15.2		
		F	70	53.9	46.1	40.8	3.9		
Aniansson et al. (1980b)	VL	M	16	54	46	32	13		
			25	54	46	32	14		
			61	53	47	29	18		
			70	48	52	36	16		
			78	60	40	23	17		
		F	16	52	48	33	15		
			24	51	49	33	16		
			70	50	50	44	5		
	G	M	48	54	46	22	24		
			61	59	41	20	21		
Aniansson et al. (1981)	VL	M	66-76	47.9	52.1	33.7	17.6	0.5	
		F	61-71	53.9	46.1	40.8	3.9	1.3	
Aniansson et al. (1986)	VL	M	73-83	51.8	48.2	30.6	16.9	0.9	
	BB	M	73-83	51.2	48.8	28.3	19.9	0.6	
Borges & Essen-Gustavsson (1989)	VL	M	20-30	62.1	37.8				
			70	62.8	37.1				
		F	20-30	46.2	53.8				
			70	42.0	58.0				
Essen-Gustavsson & Borges (1986)	VL	M	20	57	43	25	12		
			30	60	40	23	15		
			40	49	51	34	18		
			50	59	41	32	9		
			60	60	40	29	11		
			70	62	38	21	13		
		F	20	52	48	29	16		
			30	55	45	26	18		
			40	46	54	34	18		
			50	50	50	37	12		
			60	54	46	30	15		
			70	48	52	34	15		
Grimby et al. (1982)	VL	M	78-81	54.5	45.5	34.6	11.5	0.7	
		F	78-81	45.9	54.1	28.9	24.5	1.0	
	BB	M	78-81	58.5	41.5	26.4	15.5	0.4	
		F	78-81	53.3	46.7	23.7	23.0	0.1	
Grimby et al. (1984)	VL	M	70-89	52	48	29	17		
		F	66-75	55	45	27	17		
			76-85	54	46	26	19		
			86-100	58	42	26	14		
Larsson (1978)	VL	M	20-29	41	59	35	21	4	
			60-65	55	45	28	14	3	
Larsson et al. (1979)	VL	M	20-29	40.5	59.5				
			30-39	36.8	63.2				
			40-49	48.2	51.8				
			50-59	51.7	48.3				
			60-65	55.0	45.0				

Table II. Contd

Reference	Muscle	Sex	Age (y)	Fibre type distribution (%)					Fibre no.
				I	II	IIA	IIB	IIC	
Lexell et al. (1983a)	VL	M	19-37	51	49				478 000
			70-73	54	46				364 000
Lexell et al. (1986)	VL	M	15-35	49	51				614 000
			49-56	52	48				582 000
			71-83	51	49				387 000
Lexell et al. (1988)	VL	M	15-22	50	50				648 000
			26-37	50	50				599 000
			49-56	52	48				579 000
			70-75	52	48				380 000
			80-83	55	45				323 000
Sato & Tauchi (1982)	V	M	18-49	34	66				86
			50-59	39	61				89
			60-69	41	59				73
			70-79	39	61				53
			80-97	38	62				43
Sato et al. (1984)	PM	F	26-39	60	40				156
			40-49	60	40				161
			50-59	60	40				158
			60-69	60	40				131
			70-80	62	38				118
Scelsi et al. (1980)	VL	M+F	65-70	64.0	36.0				
			71-80	70.2	29.8				
			81-89	74.6	25.4				

Abbreviations: BB = biceps brachii; G = gastrocnemius; PM = pectoralis minor; V = vocalis; VL = vastus lateralis.

been taken from the vastus lateralis (fig. 1), but there is evidence from several empirical studies that the extent of the age-related change varies from one muscle group to another. For instance, the reduction in type II fibre size with age is apparently more pronounced in vastus lateralis specimens than in specimens taken from the biceps brachii (fig. 1; Aniansson et al. 1986; Brooke & Engel 1969; Edstrom & Nystrom 1969; Grimby 1986; Grimby et al. 1982; Jennekens et al. 1971b; Nygaard & Sanchez 1982). Likewise, the loss of muscle strength appears to develop somewhat more slowly in the upper than in the lower extremity (Asmussen 1980; Asmussen & Heeboll-Nielsen 1961; Simonson 1947). McDonagh et al. (1984) found that the maximal voluntary force of the male triceps surae dropped from 1895N at an age of 26 years to 1141N at 71 years, a decrease of 40%, but the corresponding figures for the elbow flexors (330 and 263N) represented a decrease of only 20%. If fast twitch muscular activity is indeed better conserved in the upper limbs than in the lower limbs as a person ages, this still leaves unanswered the question whether there is an inherent difference of aging rates between the two regions, or whether there is merely a differential disuse of type II fibres in the lower limbs.

2.4 Compensatory Hypertrophy

In principle, the altered type II/type I ratio of figure 1 could reflect either atrophy of type II fibres or a selective compensatory hypertrophy of type I fibres (Drahota & Gutmann 1962).

The latter explanation is not supported by biopsies of the human vastus lateralis (Aniansson et al. 1980b; Essen-Gustavsson & Borges 1986; Grimby et al. 1984; Larsson 1978). On the other hand, Sato

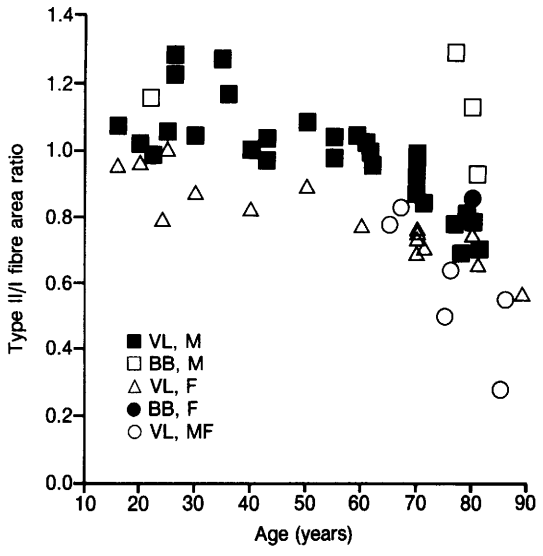

Fig. 1. Fibre area ratio (type II/type I) in relation to age. Both sexes, vastus lateralis and biceps brachii (from data by Aniansson & Gustafsson 1981; Aniansson et al. 1980b, 1981, 1986; Borges & Essen-Gustavsson 1989; Essen-Gustavsson & Borges 1986; Grimby et al. 1982, 1984; Larsson 1978; Larsson et al. 1978; Nygaard & Sanchez 1982; Polgar et al. 1973; Scelsi et al. 1980; Tomonaga 1977).

et al. (1984) observed that in surgical specimens of the female pectoralis minor, type I fibres were decreased in number but increased in size after the age of 60 years.

Studies of the rat soleus and extensor digitorum longus, and the biceps brachii, tibialis anterior, rectus femoris and sternomastoid of mice have also suggested that if there is a selective fibre loss, then a proportion of the remaining fibres, dominantly type I, hypertrophy in an attempt to sustain function (Alnaqeeb & Goldspink 1980, 1987; Hooper 1981; Silbermann et al. 1983).

2.5 Loss of Terminal Sprouting

One explanation of senile muscle atrophy and the associated changes in the proportion of type I and type II fibres is a loss of terminal sprouting, with resultant axonal withdrawal. Terminal sprouting is a normal process of end-plate repair and reconstruction throughout the lifespan (Barker & Ip 1965; Cardasis & Padykula 1981; Lewkowicz 1979; Tuffery 1971; Wernig & Herrera 1986; Wernig et al. 1980, 1984), but the capacity of this process deteriorates in senescence (Cardasis 1983; Cardasis & LaFontaine 1987; Pestronk et al. 1980) with an associated muscular atrophy and a degeneration of both muscle fibres and end-plate structures. The magnitude of these morphological changes at the neuromuscular junction appears to be influenced by the level of muscular activity during aging (Andonian & Fahim 1987; Appell 1984; Rosenheimer 1985; Stebbins et al. 1985).

Stebbins et al. (1985) have further suggested that the loss of terminal sprouting is much greater in glycolytic (type II) fibres (for instance, in gastrocnemius biopsies) than in oxidative (type I) fibres (for example, in soleus biopsies). The altered fibre distribution may thus be a consequence of a differential denervation/reinnervation process.

2.6 Muscle Atrophy

Given that the size of the individual muscle fibres does not change until extreme old age, the decrease of muscle mass that is observed in late middle and early old age must reflect a decrease in the number of muscle fibres, as demonstrated in human vocal muscles (Sato & Tauchi 1982), pectorales minor (Sato et al. 1984), vastus lateralis (Lexell et al. 1983a, 1986 1988), and in various animal muscles (Alnaqeeb & Goldspink 1980, 1987; Caccia et al. 1979; Daw et al. 1988; Gutmann & Hanzlikova 1966; Hooper 1981; Ihemelandu 1980; Ishihara et al. 1987; Layman et al. 1980; Tauchi et al. 1971).

According to autopsy studies by Lexell et al. (1983a, 1986, 1988), the decrease in the total number of fibres in the vastus lateralis (table II) is sufficient to account for all of the decrease of muscle volume, without postulating any reduction in the size of individual fibres. Relatively few human studies are available because of the technical difficulties associated with such investigations. Most of the available estimates have been derived from a combination of computerised tomography and measurements of mean fibre size. A comparison of the calf muscles in young and 80-year-old men us-

ing such an approach supported the notion that the loss of muscle volume is closely linked to the decreased number of muscle fibres (Grimby & Saltin 1983). There is a parallel reduction in muscle compound action potentials (M waves) [table III], suggesting a decrease in the number of functioning motor units (Campbell et al. 1973; Vandervoort & McComas 1986).

2.7 Neurophysiological Changes

There is considerable controversy concerning age-related changes in the number and size of motor units. Stalberg and Fawcett (1982) and Stalberg et al. (1989) used signal averaging techniques to obtain multiple single-unit electromyograph (EMG) recordings (the macro EMG). With this technique, the recorded voltage is a function of the average distance separating the muscle fibres from the electrode, the size of the muscle fibres, the total number of fibres in a motor unit, the volume characteristics of the muscle and the properties of the electrical recording system.

Stalberg et al. (1989) found an increase of the median macro EMG signal, and since they found little evidence of fibre hypertrophy, they attributed this change to a process of denervation and reinnervation, with an increase in the number of muscle fibres within surviving motor units. Sato and colleagues (1984) observed some increase of fibre size. Changes in the EMG signal are particularly marked after the age of 60 years (Campbell et al. 1973; Sato et al. 1984; Sica et al. 1974; Stalberg & Fawcett 1982).

In contrast to the human observations, animal studies to date have suggested a decrease in the size of the motor unit, reflecting a loss of muscle fibres, with little or no change in the number of motoneurones (Gutmann & Hanzlikova 1966, 1972, 1972/3, 1976; Gutmann et al. 1968 1971; Hashizume et al. 1988; Ishihara et al. 1987).

2.8 Altered Patterns of Recruitment

The relationship between static or dynamic strength and muscle cross-section is surprisingly consistent from young adults to old age (Young 1986; Young et al. 1982, 1984). However, the consistency is not necessarily absolute, since function may also be modified by altered contractile properties, motor unit recruitment or inhibition, and the inclusion of fat (Borkan et al. 1983; Cunningham & Paterson 1990; Paterson 1990) and connective tissue (Jennekens et al. 1971a; Lexell et al. 1983a,b; MacLennan et al. 1980) into the measured muscle bulk.

Ikai et al. (1967) demonstrated a 30% increase over maximal voluntary force during electrical stimulation, showing that a voluntary contraction was not successful in the maximal recruitment of all available motor units. Problems of recruitment seem particularly marked in sedentary individuals (Secher et al. 1978), and it is thus conceivable that an age-related decrease of habitual activity could contribute to a loss of muscle strength through a deterioration of recruitment patterns. On the other hand, Young et al. (1982) found a constant ratio of muscle force to cross-sectional area in the quadriceps in a comparison of young and elderly women, showing that at least in this well used muscle group there was no need to postulate any age-related changes in recruitment, excitation coupling or contractile properties.

Vandervoort and McComas (1986) examined the torque developed per unit area of muscle in response to both voluntary contraction and electrical stimulation. They noted a decrease of both data sets with age, and suggested that the primary explanation of the deterioration in muscle function was a selective loss or atrophy of type II fibres, rather than an alteration in recruitment patterns.

2.9 Conclusions Regarding Muscle Strength

We may conclude that the main reasons for the decrease in maximal voluntary force in older subjects are: (1) a progressive decrease in the number of muscle fibres, with a resultant reduction in the cross-sectional area of a given muscle group; and (2) denervation due to death of motoneurons with subsequent reinnervation of a proportion of the affected fibres. Issues such as altered patterns of

neural discharge and compensatory hypertrophy play a subordinate role to these main effects.

3. Contraction Speed

3.1 Contraction and Relaxation Times

Both contraction time (CT) and half-relaxation time (½RT) are prolonged with advancing age; this has been demonstrated for the dorsi-flexor and plantar-flexor muscles (Vandervoort & McComas 1986; table III), the extensor digitorum brevis (Campbell et al. 1973), the triceps surae (Davies & White 1983; Davies et al. 1983, 1986; Klein et al. 1988; McDonagh et al. 1984), and the elbow flexors (McDonagh et al. 1984).

Irrespective of the precise mechanism, the prolongation of the muscle twitch increases the efficiency of the older person in that a lower frequency of neural discharge is needed to induce a given torque (Nelson et al. 1984; Vandervoort & McComas 1986).

3.2 Maximum Velocity

There have been relatively few investigations of changes in the maximum speed of contraction of human muscles in relation to age. Although there seems to be some decrease in the contraction speed both for large muscles such as the quadriceps (maximum knee extension velocity) [Larsson 1978; Larsson et al. 1979] and for the small muscles involved in handwriting (Birren 1955), any changes in maximum knee extension velocity with age are less than the associated decreases in muscle strength. Larsson (1978), for example, noted a 7% decrease of maximal knee extension velocity compared with a 26 to 38% decrease of strength in 60 to 69 year old subjects. The slowing of maximal velocity apparently reflects a change in muscle quality (a decrease in the proportion of functional type IIb fibres) rather than a decrease of muscle quantity.

Aniansson et al. (1980a, 1981) found no difference of maximal knee extension velocity between 70-year-old men and women, although there was a large sex-difference in muscle mass. Likewise, animal experiments suggest that the speed of contraction is not influenced by a decrease in the total number of muscle fibres and thus muscle mass; the speed of contraction diminishes only when animals reach extreme old age (Brooks & Faulkner 1988; Carlsen & Walsh 1987; Eddinger et al. 1986; Edstrom & Larsson 1987; Fitts et al. 1984; Gutmann & Syrovy 1974; Larsson & Edstrom 1986; Syrovy & Gutmann 1970).

3.3 Fibre Type and Maximal Velocity

Several authors have argued that the quality of the muscle is the important determinant of maximal knee extension velocity, pointing to the statistically significant correlations between maximal knee extension velocity and the percentage of type IIB fibres in 22 to 65 year old men (Larsson 1978) or the overall percentage of type II fibres (17- to 37-year-old men) [Thorstensson 1976].

There are apparently no changes in the relative proportions of fibre types IIa, IIb and IIc with aging (Larsson 1978), but a number of reports suggest that the percentage of type I (slow twitch) fibres increases with advanced age (table II) [Larsson 1978; 1983; Larsson et al. 1978; 1979; Orlander et al. 1978; Scelsi et al. 1980).

3.4 Interobserver Disagreement

It may be premature to speculate on either mechanisms or the functional significance of any decrease in type II fibre representation in aging muscle, given a number of reports that find relatively normal fibre distribution in the elderly compared with either the literature or younger controls (table II, Aniansson & Gustafsson 1981; Aniansson et al. 1978, 1980b, 1981, 1984, 1986; Borges & Essen-Gustavsson 1989; Essen-Gustavsson & Borges 1985, 1986; Grimby et al. 1982, 1984; Lexell et al. 1983a, 1986, 1988; Sato & Tauchi 1982; Sato et al. 1984). The discrepancy between reports showing a marked change of fibre-type distribution and those showing little or no change is disturbing. One factor is undoubtedly subject age, given that type II fibre atrophy is probably a phenomenon of later

Table III. Effects of aging on muscle contractile properties

Reference	Muscle	Sex	Age (y)	MVC (N)	P_t (N)	CT (msec)	½RT (msec)	CV (m/sec)	M wave (mV)	PAP (/control)
Campbell et al. (1973)	EDB	M+F	3-58		?	64	53	48.1	5.7	
			60-96		?	93	109	44.2	2.7	
Davies & White (1983)	TS	M	21	1759	102	113	78			
			67-71	1152	89	148	99			
Davies et al. (1983)	TS	M	22	2109	137	121	76			
			69	1210	92	151	98			
Davies et al. (1986)	TS	M	22	1932	120	118	82			
			70	1199	96	147	100			
		F	22	1441	152	132	98			
			69	1043	118	143	126			
Klein et al. (1988)[a]	TS	M	19-32	1579	87	105	91			
			64-69	1085	64	128	92			
McDonagh et al. (1984)	TS	M	26	1895	133	119				
			71	1141	86	146				
	EF	M	26	330	39.1	71				
			71	263	27.2	76				
Vandervoort & McComas (1986)[b]	ADF	M	20-32	43.5	4.2	101	84		9.4	1.71
			40-52	37.2	4.5	111	100		9.7	1.45
			60-69	36.2	3.3	104	102		7.0	1.42
			70-79	31.6	3.3	115	122		7.8	1.31
			80-100	24.2	2.6	125	125		5.4	1.31
		F	20-32	26.6	2.7	96	84		9.1	1.75
			40-52	25.8	3.7	113	110		10.5	1.46
			60-69	23.8	2.8	115	120		7.9	1.35
			70-79	21.5	1.8	110	119		6.3	1.35
			80-100	16.7	1.7	128	131		5.2	1.28
	APF	M	20-32	171	15.5	144	109		20.7	1.52
			40-52	171	16.3	169	122		18.6	1.56
			60-69	136	13.4	170	117		13.3	1.29
			70-79	121	13.4	178	133		12.2	1.22
			80-100	94	11.9	186	144		9.5	1.15
		F	20-32	113	13.6	146	123		18.9	1.35
			40-52	127	14.5	179	139		15.0	1.20
			60-69	96	11.9	182	133		10.5	1.12
			70-79	94	13.0	183	143		8.8	1.12
			80-100	54	8.6	195	169		6.4	1.17

a Subjects were physically active.

b Data on MVC and P_t are expressed as N · m.

Abbreviations: ADF = ankle dorsiflexor; APF = ankle plantarflexor; CT = contraction time; CV = maximal impulse conduction velocity; EDB = extensor digitorum brevis; EF = elbow flexor; MVC = maximal voluntary contraction; PAP = postactivation potentiation; P_t = maximal twitch tension; ½RT = half-relaxation time; TS = triceps surae.

old age. There is often a problem of selective sampling of physically active subjects; this problem inevitably becomes more acute as the age of the sample is increased.

Another concern is whether a needle biopsy sample comprising 200 to 400 fibres is representative of the characteristics of an entire muscle. Duplicate samples, whether taken from the same leg (vastus lateralis) [Blomstrand et al. 1984; Campbell et al. 1979; Gollnick et al. 1972; Halkjaer-Kristen-

sen & Ingemann-Hansen 1981; Thorstensson 1976] or at corresponding sites on the opposite leg (Blomstrand & Ekblom 1982; Campbell et al. 1979; Essen-Gustavsson & Borges 1986; Halkjaer-Kristensen & Ingemann-Hansen 1981) show an inter-test coefficient of variation of 10 to 20%. Some authors have found no systematic differences of fibre type within the muscle (Edgerton et al. 1975; Elder 1977; Elder et al. 1982; Nygaard & Sanchez 1982), but others have found significant differences between the deep and superficial fibres of the vastus lateralis (Jennekens et al. 1971c; Johnson et al. 1973; Lexell et al. 1983a,b, 1984). Thus, it is important that muscle biopsies be taken at a comparable depth within a muscle (Essen-Gustavsson & Borges 1986). If the muscles are smaller in an older person, it becomes very difficult to decide how to control for this variable, and an inappropriate decision might account for the apparent effects of aging upon muscle fibre composition.

Several alternative tactics have been suggested to obtain more representative muscle specimens. One option is to obtain a well defined muscle sample by open surgical biopsy (Bylund et al. 1977; Sjostrom et al. 1980, 1982a,b). A second possibility is to use a noninvasive, magnetic resonance technique. Kuno et al. (1988) found a high correlation between magnetic relaxation times and the percentage of type II fibres (T1, $r = 0.924$; T2, $r = 0.889$; T1 and T2, $r = 0.929$); this relationship was not changed by strength training (Kuno et al. 1990). Possibly, such approaches will resolve the current controversy.

Grimby and Saltin (1983) have suggested that the lack of change in fibre composition with age in many studies may reflect the fact that fibres become reinnervated in accordance with the existing distribution of phasic and tonic nerve endings. In line with this, there are only minor changes in enzyme activity with age (table I) [Aniansson et al. 1980b; 1981; 1984, 1986; Borges & Essen-Gustavsson 1989; Essen & Gustavsson & Borges 1986; Grimby et al. 1982; Larsson 1978; Larsson et al. 1978; Orlander & Aniansson 1980; Orlander et al. 1978; Saltin & Gollnick 1983), although the data do not exclude the possibility of small changes in enzyme activity.

3.5 Suggested Mechanisms of Altered Fibre Type Distribution

Postulated mechanisms for an increased proportion of type I fibres include an increase in the number of type I fibres stemming from splitting or proliferation (that is, new fibre formation from satellite cells), a preferential atrophy and subsequent reduction in the number of type II fibres due to a loss of terminal sprouting, and a transformation of type II into type I fibres.

Some hyperplasia of muscle fibres has previously been reported after heavy resistance training such as a weight-lifting programme (Gonyea et al. 1986; Hoppeler 1986; Tesch 1988), although the observed response (about 10%) is close to the limits of measurement. Such a response could explain why fibre size was similar but muscle girths were greater in body builders than in untrained controls (MacDougall et al. 1982). Thus, a selective reduction in the number of type II fibres (Lexell et al. 1983a, 1986, 1988) and a transformation of type II into type I fibres seem plausible explanations of the age-related changes.

Conversion of muscle fibre types during aging is usually quoted as evidence for motor neuron death and consequent reinnervation by the remaining intact motor neurons. Fibre-type conversion could thus occur if type II fibres were reinnervated by the sprouting of nerve terminals normally innervating type I fibres. Type IIb may be converted to type IIa fibres (Hollozy & Coyle 1984; Katsuta et al. 1988), and a transformation of type II into type I fibres has also been observed by some authors following either repeated prolonged exercise such as endurance running (Green et al. 1984; Wada & Katsuta 1988) or chronic electrical stimulation at an appropriate discharge frequency (Howald 1982; Pette 1984). In this regard, Wernig and Herrera (1986) proposed an alternative explanation for its occurrence, based on the idea that such a change may not necessarily be initiated by motor neuron loss. According to their hypothesis,

fibre-type conversion may be the result of a continuous remodelling of motor units, whereby collateral and terminal sprouts from one neuron form synapses (probably in the end-plate area) on neighbouring muscle fibres, with or without loss of the pre-existing terminal. The concept of polyneural innervation is controversial; in their view, the enzyme patterns (histochemical parameters) of polyneuronally innervated fibres would generally be determined by the neuron with the higher activity (according to the new activity pattern). Alterations dependent on neuronal activity resemble age-dependent changes in that type I fibres are relatively more active than type II fibres, thereby causing a progressive change from type II to type I as the fibres are reinnervated (Grimby & Saltin 1983; Grimby et al. 1982 1984; Jennekens et al. 1971a, b; Johnson et al. 1973; Larsson 1983; Lexell et al. 1984, 1986; Nygaard & Sanchez 1982; Tomonaga 1977).

Further study is required to explain the detailed mechanisms of the altered fibre-type distribution.

3.6 Other Explanations of Slowed Contraction

Some authors such as Payton and Poland (1983) have suggested that in view of the relatively small changes of fibre composition with aging, impairments of membrane depolarisation and excitation-contraction coupling mechanisms may provide a more plausible explanation of the slowing of muscle contraction in older subjects.

4. Muscular Endurance

4.1 Normal Determinants

In young men, muscular endurance is a specific characteristic of muscle function. The endurance time for a fatiguing static effort is independent of the muscle group involved, of isotonic or isometric muscle strength (Myers & Sullivan 1968), or of the state of static or dynamic training (Hansen 1967; McGlynn 1968; Peterson et al. 1961). The critical factors influencing performance are the intensity of effort relative to the maximal voluntary force of the muscle concerned, the number of repetitions, if any, and the inter-contraction interval (Kilbom 1976; Royce 1958; Shephard 1974).

Thus, when well-motivated subjects of any age perform isometric exercise at a standard fraction (40 to 50%) of their maximal isometric strength, the endurance time will be the same as for other individuals exercising at the same relative load. The only limitation to this generalisation might arise if the heart of an older person were unable to increase the systemic blood pressure as easily as in a younger person.

4.2 Technical Factors

Reasons for any increase of muscular endurance in older individuals remain uncertain. Since endurance is related to peak effort, a poorer maximal contraction by an older person (whether due to the aging of muscle function or reduced cooperation) might result in the imposition of a relatively lighter task, allowing a greater endurance. However, Petrofsky and Lind (1975b) have rejected this hypothesis.

4.3 Influence of Superficial Fat

Petrofsky and Lind (1975b,c) suggested that inter-individual differences in muscular endurance were related to the percentage of body fat, and thus intramuscular temperature. Subjects with a high percentage of body fat had a higher muscle temperature and a reduced muscle endurance time.

Most studies have shown some increase of superficial body fat with aging (Heath et al. 1981; Irving et al. 1980; Kasch et al. 1985; Montegriffo 1968; Parízková 1963; Pollock & Wilmore 1990; Pollock et al. 1974; 1987; Shephard 1991). Moreover, a multivariate analysis by Petrofsky and Lind (1975b) found that age and body mass (the latter possibly serving as a surrogate marker of body fat) each made independent contributions to the inter-individual variance in isometric endurance.

However, it is unlikely that an increase of subcutaneous fat has a major influence upon muscular endurance, since handgrip isometric endurance

(Aniansson et al. 1978; Petrofsky & Lind 1975a,b; Sperling 1980) and knee-extensor isometric and dynamic endurance (Johnson 1982; Larsson 1978; Larsson & Karlsson 1978) are either unaltered or increase only slightly over the first half of adult life (when much of the increase of superficial body fat normally occurs).

4.4 Cellular Influences

One potential cellular explanation of enhanced endurance might be an altered fibre distribution, although as discussed above the evidence for such a change is conflicting.

Type II fibres have a higher activity of the lactate forming enzyme lactate dehydrogenase (LDH), a higher rate of lactate production and a more rapid onset of fatigue (Essen & Haggmark 1975; Mainwood & Renaud 1985; Meyer & Terjung 1979; Sjodin 1976; Thorstensson et al. 1977). The accumulation of lactate seems an important limiting factor in the dynamic and isometric endurance tests commonly used for comparative studies (Hulten et al. 1975; Tesch et al. 1978).

The lactate hypothesis is supported by Larsson (1978), who noted an association between increased dynamic endurance, decreased activity of the muscle-specific isoenzyme of LDH (M-LDH), and an increased cytochrome oxidase activity (table I), the enzyme changes favouring oxidation of pyruvate rather than its conversion to lactate (Brooks 1986a,b; Stainsby 1986).

4.5 Local Blood Flow and Endurance

A final factor that could change endurance would be an alteration in the capillarity of skeletal muscle. Capillarity can be expressed in terms of various indices, including capillary density [the number of capillaries in unit area of muscle cross-section (capillaries/mm^2)], capillary to fibre ratio (capillary density/fibre density), capillary/fibre contact ratio (the number of capillaries in contact with a typical fibre of a given type), or the capillary/fibre contact ratio per unit fibre cross-sectional area (a measure of the typical diffusion distance) [table IV].

In young adults (Anderson & Henriksson 1977; Aniansson et al. 1980b; Brodal et al. 1977; Ingjer 1979a,b; Ingjer & Brodal 1978; Nygaard 1981; Parízková et al. 1971), the capillary density is 270 to 369/mm^2, the capillary/fibre ratio 0.81 to 1.80, the capillary contact ratio 2.33 to 4.84, and the capillary contact per unit fibre area 0.78 to 1.26/$\mu m^2 \times 10^{-3}$ (table IV). These values are almost unchanged in 70 to 80 year old men and women (Aniansson et al. 1980b 1981 1986; Grimby et al. 1982; Parízková et al. 1971), to the point that the coupling between capillarity and the oxidative potential of the muscle fibres suggested by Romanul (1965) apparently persists throughout life.

The only potential adverse feature in an older person would be a selective loss of the smaller, type II fibres; in itself, this would increase the diffusion distance from capillary to mitochondria, but this is more than compensated by an increase of capillary/fibre contact ratio per unit fibre cross-sectional area (table IV). It thus appears unlikely that alterations of muscle capillarity make a major contribution to age-related changes in muscular capillarity make a major contribution to age-related changes in muscular endurance.

5. Variations in the Aging of Muscle Function

5.1 Regional

In confirmation of classical views, McDonagh et al. (1984) observed only a 20% decrease of elbow flexor strength from 20 to 70 years, whereas the triceps surae showed a 40% average decrease of maximal voluntary force. However, there was insufficient detail on the lifestyle of the 2 groups of subjects to determine whether such findings reflect regional differences of activity patterns or a more fundamental difference in the aging process between the upper and the lower limbs.

Asmussen and Heeboll-Nielsen (1961) and Simonson (1947) also support the view that function deteriorates less rapidly in the upper limbs.

Table IV. Influence of age upon muscle capillarity (data from Andersen & Henriksson 1977; Aniansson et al. 1980b, 1981, 1986; Brodal et al. 1977; Grimby et al. 1982; Ingjer 1979a,b; Ingjer & Brodal 1978; Nygaard 1981; Parizková et al. 1971)

Group		Capillaries (mm^{-2})	Capillaries ($fibre^{-1}$)	CC			CC relative to fibre area ($\mu m^{-2} \times 10^{-3}$)		
				type I	type IIA	type IIB	type I	type IIA	type IIB
Men	Young[a]	270-369	0.81-1.80	3.90-4.76	4.20-4.84	2.94-3.00	1.03-1.25	0.86-0.95	0.78-0.84
	Old[b]	247-347	0.59-1.61	3.70-4.56	3.08-3.92	2.64-3.20	0.90-1.54	1.03-1.72	1.07-1.38
Women	Young[c]	301-348	1.11-1.39	4.00-4.11	3.40-3.70	2.33-2.90	1.07	0.99	0.84
	Old[b]	296-358	1.10-1.40	3.40-4.38	2.60-3.80	2.75-2.90	1.11-1.31	1.05-1.30	0.80-1.26

a 18 to 34 years.
b 67 to 81 years.
c 18 to 40 years.
Abbreviation: CC = capillaries in contact with each fibre.

5.2 Sex-Related

In young adults, it is generally held that women have approximately 60% of the peak maximal voluntary force of men (Asmussen 1965), although this ratio varies from the upper to the lower limbs, and depends also on body size and cultural factors that have encouraged greater muscular training in men (Walter et al. 1988).

Much of the data for men and women in the seventh and eighth decades of life shows this same 60 to 65% ratio (Aniansson et al. 1980a; 1983; Borges 1989; Fugl-Meyer et al. 1980; Sperling 1980; Vandervoort & McComas 1986). On the other hand, Aniansson et al. (1980a, 1981) found no difference in peak knee extension velocity between 70-year-old men and women (mean values of 11.6 *vs* 10.8 rad/sec), despite large differences of muscle mass between the two sexes (29.2 *vs* 22.3kg).

5.3 Activity-Related

Grimby and Saltin (1983) have suggested that much of the difference in the rate of aging on muscle function between the arms and the legs is due to the persistence of faster and more forceful contractions in the arms. They note that both the arms and the diaphragm (Gutmann & Hanzlikova 1972) conserve fast twitch function more effectively than the legs.

Although protein is synthesised less rapidly in an older individual (Zackin & Meredith 1989), a poor initial condition usually facilitates demonstration of a training response. Aoyagi and Katsuta (1990) suggested that the response depended on the age at which training was begun. They compared masters athletes aged 60 to 68 years who had engaged in endurance running since the age of 26, 45 and 56 years. At high contraction velocities not encountered in everyday living, the first 2 groups had a greater peak elbow flexion torque than either sedentary subjects or those who waited until they were older to initiate training. On the other hand, all 3 groups of trained subjects had higher peak knee extension torques than the untrained individuals at all velocities of contraction. It is less clear whether the training programme induced hypertrophy, prevented atrophy or slowed the decrease in the number of muscle fibres.

Larsson (1982) found only a limited response (less than 10% increase in strength) to a low resistance training programme with frequent repetitions. However, Moritani and deVries (1980) obtained substantial gains of strength (more than 20%) despite little hypertrophy after participation in a high resistance programme. Fiatarone et al. (1990) demonstrated a 10% increase in muscle area in 7 elderly subjects (mean 90 years) after 8 weeks of

strength training. Moreover, large increases of muscle strength were realised by as little as 12 weeks of 1-repetition maximum resistance training (Frontera et al. 1988). Mertens et al. (1978) demonstrated substantial gains of muscular endurance (44% over 1 year) following introduction of a simple weight training programme for 45 to 66 years of age patients with chronic obstructive lung disease. However, there is often a gap between gains of measured strength and gains of muscle bulk (Fried & Shephard 1970; Moritani & deVries 1980), much of the improvement of performance being attributable to a better coordinated contraction or even practice of the required skill.

Aoyagi and Katsuta (1990) found that intergroup differences in the peak velocity of elbow flexion and knee extension mirrored the differences in peak torque described above, although the advantage of contraction velocity gained from an early start to training was smaller than the advantage in strength. Given that the intergroup differences were seen in relatively early old age, it was suggested that regular training might sustain contraction velocity by preserving excitation-contraction coupling (Payton & Poland 1983), rather than by minimising the relative loss of type II fibres.

These observations may be compared with the earlier work of Aniansson and Gustafsson (1981) who found that in men aged 69 to 94 years, 12 weeks of strength training led to an increase of both static and dynamic strength in the quadriceps muscle of the same order as that anticipated in younger subjects. There was an associated increase in the proportion of type II fibres (particularly type IIa), but no significant changes of peak contraction velocity. The training-related increase of type II fibres is especially interesting, given that Aniansson and associates have tended to dismiss the possibility of a selective decrease in the number of type II fibres with aging. There were also some increases in the activity of myokinase and cytochrome oxidase, although they concluded that altered patterns of neural recruitment contributed substantially to the training response. In a related report, Orlander and Aniansson (1980) noted that while the glycolytic potential was increased by training, any increase of oxidative capacity took place within the existing mitochondrial volume, in contrast to the situation in young individuals where endurance training led to a preferential proliferation of subsarcolemmal mitochondria rather than interfibrillar mitochondria (Hoppeler et al. 1985; Rosler et al. 1985).

6. Clinical Significance of Loss of Muscle Function

The progressive deterioration of muscle function has many adverse clinical consequences for the frail elderly, including severe dyspnoea, unstable joints, and final dependence.

6.1 Dyspnoea

The weakened muscles contract at a larger fraction of their maximal force, so that there is more likelihood of a significant restriction of perfusion during aerobic activity (Kay & Shephard 1969). The resultant accumulation of H^+ions increases the ventilatory cost of effort, and particularly in patients who have some chronic obstructive lung disease, effort can be limited by severe dyspnoea (Mertens et al. 1978). This in turn creates a vicious cycle of unpleasant breathlessness, anxiety, restriction of daily activity, further muscle weakening and worsened breathlessness.

6.2 Unstable Joints

Arthritis leads to instability of major joints such as the knees in the frail elderly (Young 1986), exacerbating other causes of falls in this age group (Overstall 1980; Overstall et al. 1977; Tinetti et al. 1988). The strength of the surrounding muscles is a key factor in compensating for an unstable joint, and likewise the velocity of contraction is important to the restoration of balance if tripping should occur.

6.3 Dependence

As aging continues, the point is reached where muscle strength is insufficient to lift the body mass, on rising from a chair or toilet seat, and eventually

in attempting to get out of bed. Tasks such as carrying shopping become excessively arduous, and even the caps of tablet bottles become impossible to remove. The senior citizen then faces a period of dependence, with a major deterioration in the quality of life. The Canada Health Survey (1982) found that the period of dependence was greater in women than in men (in part because they lived longer, but possibly also in part because of weaker muscles at any given age). On average, Canadians showed partial dependence for 8 to 10 years, with total dependence during the final year of life.

6.4 Value of Training

Training to conserve muscle function can play an important role in both preventing and reversing such changes. Mertens et al. (1978) specifically demonstrated the beneficial effect of leg muscle training in improving the functional ability of dyspnoeic patients with chronic obstructive lung disease. Likewise, it is clear that a relatively fixed muscle force is needed to lift a given body mass from a chair; if strength deteriorates at 5% per decade, but training can increase strength by 10%, then this will set back by as much as 20 years the age at which muscle strength is insufficient to accomplish the tasks of daily living.

References

Allen TH, Anderson EC, Langham WH. Total body potassium and gross body composition in relation to age. Journal of Gerontology 15: 348-357, 1960

Alnaqueeb MA, Goldspink G. Interrelation of muscle fibre types, diameter and number in ageing white rats. Journal of Physiology 310: 56P, 1980

Alnaqeeb MA, Goldspink G. Changes in fibre type, number and diameter in developing and ageing skeletal muscle. Journal of Anatomy 153: 31-45, 1987

Andersen P, Henriksson J. Capillary supply of the quadriceps femoris muscle of man: adaptive responses to exercise. Journal of Physiology 270: 677-690, 1977

Andonian MH, Fahim MA. Effects of endurance exercise on the morphology of mouse neuromuscular junctions during ageing. Journal of Neurocytology 16: 589-599, 1987

Aniansson A, Grimby G, Hedberg M, Rundgren A, Sperling L. Muscle function in old age. Scandinavian Journal of Rehabilitation Medicine 6 (Suppl.): 43-49, 1978

Aniansson A, Grimby G, Rundgren A. Isometric and isokinetic quadriceps muscle strength in 70-year-old men and women. Scandinavian Journal of Rehabilitation Medicine 12: 161-168, 1980a

Aniansson A, Grimby G, Nygaard E, Saltin B. Muscle fiber composition and fiber area in various age groups. Muscle and Nerve 2: 271-272, 1980b

Aniansson A, Gustafsson E. Physical training in elderly men with special reference to quadriceps muscle strength and morphology. Clinical Physiology 1: 87-98, 1981

Aniansson A, Grimby G, Hedberg, Krotkiewski M. Muscle morphology, enzyme activity and muscle strength in elderly men and women. Clinical Physiology 1: 73-86, 1981

Aniansson A, Sperling L, Rundgren A, Lehnberg E. Muscle function in 75 year old men and women, a longitudinal study. Scandinavian Journal of Rehabilitation Medicine 9 (Suppl.): 92-102, 1983

Aniansson A, Hedberg M, Henning G-B, Grimby G. Muscle morphology, enzymatic activity, and muscle strength in elderly men: a follow-up study. Muscle and Nerve 9: 585-591, 1986

Aniansson A, Zetterberg C, Hedberg M, Henriksson KG. Impaired muscle function with aging: a background factor in the incidence of fractures of the proximal end of the femur. Clinical Orthopaedics 191: 193-201, 1984

Aoyagi Y, Katsuta S. Relationship between the starting age of training and physical fitness in old age. Canadian Journal of Sport Sciences 15: 65-71, 1990

Appell HJ. Proliferation of motor end-plates induced by increased muscular activity. International Journal of Sports Medicine 5: 125-129, 1984

Asmussen E. Muscular exercise. In Fenn & Rahn (Eds) Handbook of physiology: section 3, Respiration, Vol. 2, pp. 939-978, American Physiological Society, Washington, 1965

Asmussen E. Aging and exercise. In Horvath & Jousef (Eds) Environmental physiology: aging, heat and altitude, pp. 419-428, Elsevier, New York, 1980

Asmussen E, Heeboll-Nielsen K. Isometric muscle strength of adult men and women. Communications from the Testing and Observation Institute, Danish Infantile Paralysis Association 11: 1-44, 1961

Åstrand P-O, Rodahl K. Textbook of work physiology: physiological cases of exercise, 3rd ed., McGraw-Hill, New York, 1986

Barker D, Ip MC. The probable existence of a process of motor end-plate replacement. Journal of Physiology 176: 11-12P, 1965

Belanger AY, McComas AJ, Elder GBC. Physiological properties of two antagonistic human muscle groups. European Journal of Applied Physiology 51: 381-393, 1983

Birren J. Age changes in speed of simple response and perception and their significance for complex behaviour. In Old age in the modern world, chapter 8: Experimental studies of changes in performance with age, pp. 235-247, E & S Livingstone, London, 1955

Blomstrand E, Ekblom, B. The needle biopsy technique for fibre type determination in human skeletal muscle – a methodological study. Acta Physiologica Scandinavica 116: 437-442, 1982

Blomstrand E, Celsing F, Friden J, Ekblom B. How to calculate human muscle fibre areas in biopsy samples – methodological considerations. Acta Physiologica Scandinavica 122: 545-551, 1984

Borges O. Isometric and isokinetic knee extension and flexion torque in men and women aged 20-70. Scandinavian Journal of Rehabilitation Medicine 21: 45-53, 1989

Borges O, Essen-Gustavsson B. Enzyme activities in type I and II muscle fibres of human skeletal muscle in relation to age and torque development. Acta Physiologica Scandinavica 136: 29-36, 1989

Borkan GA, Hults DE, Gerzof SG, Robbins AH, Silbert CK. Age changes in body composition revealed by computed tomography. Journal of Gerontology 38: 673-677, 1983

Brodal P, Ingjer F, Hermansen L. Capillary supply of skeletal muscle fibers in untrained and endurance-trained men. American Journal of Physiology 232: H705-H712, 1977

Brooke MH, Engel WK. The histographic analysis of human muscle biopsies with regard to fiber types: 1. Adult muscle and female. Neurology 19: 221-233, 1969

Brooks GA. Lactate production under fully aerobic conditions: the lactate shuttle during rest and exercise. Federation Proceedings 45: 2924-2929, 1986a

Brooks GA. The lactate shuttle during exercise and recovery. Medicine and Science in Sports and Exercise 18: 360-368, 1986b

Brooks SV, Faulkner JA. Contractile properties of skeletal muscles from young, adult and aged mice. Journal of Physiology 404: 71-82, 1988

Bylund A-C, Bjuro T, Cederblad G, Holm J, Lundholm K, et al. Physical training in man. Skeletal muscle metabolism in relation to muscle morphology and running ability. European Journal of Applied Physiology 36: 151-169, 1977

Caccia MR, Harris JB, Johnson MA. Morphology and physiology of skeletal muscle in aging rodents. Muscle and Nerve 2: 202-212, 1979

Campbell CJ, Bonen A, Kirby RL, Belcastro AN. Muscle fiber composition and performance capacities of women. Medicine and Science in Sports 11: 260-265, 1979

Campbell MJ, McComas AJ, Petito F. Physiological changes in ageing muscles. Journal of Neurology, Neurosurgery and Psychiatry 36: 174-182, 1973

Canada Health Survey. Ottawa: health and welfare, Canada, 1982

Cardasis CA. Ultrastructural evidence of continued reorganization at the aging (11-26 months) rat soleus neuromuscular junction. Anatomical Record 207: 399-415, 1983

Cardasis CA, LaFontaine DM. Aging rat neuromuscular junctions: a morphometric study of choline-esterase-stained whole mounts and ultrastructure. Muscle and Nerve 10: 200-213, 1987

Cardasis CA, Padykula HA. Ultrastructural evidence indicating reorganization at the neuromuscular junction in the normal rat soleus muscle. Anatomical Record 200: 41-59, 1981

Carlsen RC, Walsh DA. Decrease in force potentiation and appearance of a-adrenergic mediated contracture in aging rat skeletal muscle. Pflugers Archiv - European Journal of Physiology 408: 224-230, 1987

Clarkson PM, Kroll W, Melchionda AM. Age, isometric strength, rate of tension development and fiber type composition. Journal of Gerontology 36: 648-653, 1981

Cullumbine H, Bibile S, Wikramanayake TW, Watson RS. Influence of age, sex, physique and muscular development on physical fitness. Journal of Applied Physiology 2: 488-511, 1950

Cunningham DA, Morrison D, Rice CL, Cooke C. Ageing and isokinetic plantar flexion. European Journal of Applied Physiology 56: 24-29, 1987

Cunningham DA, Paterson DH. Discussion: exercise, fitness and aging. In Bouchard et al. (Eds) Exercise fitness and health: a consensus of current knowledge, chapter 60, pp. 699-704, Human Kinetics Publishers, Champaigne, 1990

Davies CTM, White MJ. Contractile properties of elderly human triceps surae. Gerontology 29: 19-25, 1983

Davies CTM, White MJ, Young K. Electrically evoked and voluntary maximal isometric tension in relation to dynamic muscle performance in elderly male subjects, aged 69 years. European Journal of Applied Physiology 51: 37-43, 1983

Davies CTM, Thomas DO, White MJ. Mechanical properties of young and elderly human muscle. Acta Medica Scandinavica (Suppl. 711): 219-226, 1986

Daw CK, Starnes JW, White TP. Muscle atrophy and hypoplasia with aging: impact of training and food restriction. Journal of Applied Physiology 64: 2428-2432, 1988

Drahota Z, Gutmann E. The effect of age on compensatory and 'post-functional hypertrophy' in cross-striated muscle. Gerontologia 6: 81-90, 1962

Edgerton RV, Smith JL, Simpson DR. Muscle fibre type populations of human leg muscles. Histochemical Journal 7: 259-266, 1975

Eddinger TJ, Cassens RG, Moss RL. Mechanical and histochemical characterization of skeletal muscles from senescent rats. American Journal of Physiology 251: C421-C430, 1986

Edstrom L, Larsson L. Effects of age on contractile and enzyme-histochemical properties of fast- and slow-twitch single motor units in the rat. Journal of Physiology 392: 129-145, 1987

Edstrom L, Nystrom B. Histochemical types and sizes of fibres in normal human muscles: a biopsy study. Acta Neurologica Scandinavica 45: 257-269, 1969

Elder GCB. The heterogeneity of fibre type populations in human muscle. Medicine and Science in Sports 9: 64-65, 1977

Elder GCB, Bradbury K, Roberts R. Variability of fiber type distributions within human muscles. Journal of Applied Physiology 53: 1473-1480, 1982

Essen B, Haggmark T. Lactate concentration in type I and II muscle fibres during muscular contraction in man. Acta Physiologica Scandinavica 95: 344-346, 1975

Essen-Gustavsson B, Borges O. Enzyme profiles in Type I and II fibre populations of 70 year old men and women. Acta Physiologica Scandinavica 123: 23A, 1985

Essen-Gustavsson B, Borges O. Histochemical and metabolic characteristics of human skeletal muscle in relation to age. Acta Physiologica Scandinavica 126: 107-114, 1986

Fiatarone MA, Marks EC, Ryan ND, Meredith CN, Lipsitz LA, et al. High-intensity strength training in nonagenarians. Effects on skeletal muscle. Journal of the American Medical Association 263: 3029-3034, 1990

Fisher MB, Birren JE. Age and strength. Journal of Applied Psychology 31: 490-497, 1947

Fitts RH, Troup JP, Witzmann FA, Holloszy JO. The effect of ageing and exercise on skeletal muscle function. Mechanisms of Ageing and Development 27: 161-172, 1984

Fleg JL. Lakatta EG. Role of muscle loss in the age-associated reduction in $V0_{2max}$. Journal of Applied Physiology 65: 1147-1151, 1988

Forbes GB. The adult decline in lean body mass. Human Biology 48: 161-173, 1976

Frontera WR, Meredith CN, O'Reilly KP, Knuttgen HG, Evans WJ. Strength conditioning in older men: skeletal muscle hypertrophy and improved function. Journal of Applied Physiology 64: 1038-1044, 1988

Fried T, Shephard RJ. Assessment of a lower extremity training program. Canadian Medical Association Journal 103: 260-266, 1970

Froese EA, Houston ME. Torque-velocity characteristics and muscle fiber type in human vastus lateralis. Journal of Applied Physiology 59: 309-314, 1985

Fugl-Meyer AR, Gustafsson L, Burstedt Y. Isokinetic and static plantar flexion characteristics. European Journal of Applied Physiology 45: 221-234, 1980

Galton, F. Record of family faculties; consisting of tabular forms and direction, Macmillan, London, 1884

Gollnick PD, Armstrong RB, Saubert IV CW, Piehl K, Saltin B. Enzyme activity and fiber composition in skeletal muscle of untrained and trained men. Journal of Applied Physiology 33: 312-319, 1972

Gonyea WJ, Sale DG, Gonyea FB, Mikesky A. Exercise induced increases in muscle fiber number. European Journal of Applied Physiology 55: 137-141, 1986

Green HJ, Klug GA, Reichman H, Seedorf V, Weihrer W, Pette D. Exercise-induced fibre type transitions with regard to myosin parvalbumin and sacroplasmic reticulum in muscles of the rat. Plugers Archives 400: 432-436, 1984

Grimby G. Physical activity and muscle training in the elderly. Acta Medica Scandinavica (Suppl. 711): 233-237, 1986

Grimby G, Aniansson A, Zetterbeurg C, Saltin B. Is there a change in relative muscle fibre composition with age? Clinical Physiology 4: 189-194, 1984

Grimby G, Danneskiold-Samsoe B, Hvid K, Saltin B. Morphol-

ogy and enzymatic capacity in arm and leg muscles in 78-81 year old men and women. Acta Physiologica Scandinavica 115: 125-134, 1982
Grimby G, Saltin B. Mini-review. The ageing muscle. Clinical Physiology 3: 209-218, 1983
Gutmann E, Hanzlikova V. Motor unit in old age. Nature 209: 921-922, 1966
Gutmann E, Hanzlikova V. Age changes in the neuromuscular system, Scientechnica (publications) Ltd, Bristol, 1972
Gutmann E, Hanzlickova V. Basic mechanisms of aging in the neuromuscular system. Mechanisms of Ageing and Development 1: 327-349, 1972/73
Gutmann E, Hanzlikova V. Fast and slow motor units in ageing. Gerontology 22: 280-300, 1976
Gutmann E, Syrovy I. Contraction properties and myosin-ATPase activity of fast and slow senile muscles of the rat. Gerontologia 20: 239-244, 1974
Gutmann E, Hanzlikova V, Jakoubek B. Changes in the neuromuscular system during old age. Experimental Gerontology 3: 141-146, 1968
Gutmann E, Hanzlikova V, Vyskocil F. Age changes in cross-striated muscle of the rat. Journal of Physiology 216: 331-343, 1971
Gutmann E, Melichna J, Syrovy I. Contraction properties and ATPase activity in fast and slow muscle of the rat during denervation. Experimental Neurology 36: 488-497, 1972
Halkjaer-Kristensen J, Ingemann-Hansen T. Variations in single fibre areas and fibre composition in needle biopsies from the human quadriceps muscle. Scandinavian Journal of Clinical and Laboratory Investigation 41: 391-395, 1981
Hansen JW. Effect of dynamic training on the isometric endurance of the elbow flexors. Internationale Zeitschrift für Angewandte Physiologie 23: 367-370, 1967
Hashizume K, Kanda K, Burke RE. Medial gastrocnemius motor nucleus in the rat: age-related changes in the number and size of motoneurons. Journal of Comparative Neurology 269: 425-430, 1988
Heath GW, Hagberg JM, Ehsani AA, Holloszy JO. A physiological comparison of young and older endurance athletes. Journal of Applied Physiology 51: 634-640, 1981
Hettinger T. Physiology of strength. C.C. Thomas, Springfield, 1961
Holloszy JO, Coyle EF. Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. Journal of Applied Physiology 56: 831-838, 1984
Hooper ACB. Length, diameter and number of ageing skeletal muscle fibres. Gerontology 27: 121-126, 1981
Hoppeler H. Exercise-induced ultra structural changes in skeletal muscle. International Journal of Sports Medicine 7: 187-204, 1986
Hoppeler H, Howald H, Conley K, Lidstedt SL, Classen H, et al. Endurance training in humans: aerobic capacity and structure of skeletal muscle. Journal of Applied Physiology 59: 320-327, 1985
Howald H. Training-induced morphological and functional changes in skeletal muscle. International Journal of Sports Medicine 3: 1-12, 1982
Hulten B, Thorstensson A, Sjodin B, Karlsson J. Relationship between isometric endurance and fibre types in human leg muscles. Acta Physiologica Scandinavica 93: 135-138, 1975
Ihemelandu EC. Decrease in fibre numbers of dog pectineus muscle with age. Journal of Anatomy 130: 69-73, 1980
Ikai M, Fukunaga T. Calculation of muscle strength per unit cross-sectional area of human muscle by means of ultra-sonic measurement. Internationale Zeitschrift für Angewandte Physiologie 26: 26-32, 1968
Ikai M, Yabe K, Ishii K. Muskelkraft und muskulare Ermudung bei willkurlicher Anspannung und elektrischer Reizung des Muskels. Sportarzt und Sportmedizin 5: 197-211, 1967
Ingjer F. Effects of endurance training on muscle fibre ATPase activity, capillary supply and mitochondrial content in man. Journal of Physiology 294: 419-432, 1979a
Ingjer F. Capillary supply and mitochondrial content of different skeletal muscle fiber types in untrained and endurance-trained men: a histochemical and ultrastructural study. European Journal of Applied Physiology 40: 197-209, 1979b
Ingjer F, Brodal P. Capillary supply of skeletal muscle fibers in untrained and endurance-trained women. European Journal of Applied Physiology 38: 291-299, 1978
Irving JB, Kusumi F, Bruce RA. Longitudinal variations in maximal oxygen consumption in healthy men. Clinical Cardiology 3: 134-136, 1980
Ishihara A, Naitoh H, Katsuta S. Effects of ageing on the total number of muscle fibers and motoneurons of the tibialis anterior and soleus muscles in the rat. Brain Research 435: 355-358, 1987
Jennekens FGI, Tomlinson BE, Walton JN. Histochemical aspects of five limb muscles in old age: an autopsy study. Journal of the Neurological Sciences 14: 259-276, 1971a
Jennekens FGI, Tomlinson BE, Walton JN. The sizes of the two main histochemical fibre types in five limb muscles in man: an autopsy study. Journal of the Neurological Sciences 13: 281-292, 1971b
Jennekens FGI, Tomlinson BE, Walton JN. Data on the distribution of fibre types in five human limb muscles: an autopsy study. Journal of the Neurological Sciences 14: 245-257, 1971c
Johnson T. Age-related differences in isometric and dynamic strength and endurance. Physical Therapy 62: 985-989, 1982
Johnson MA, Polgar J, Weightman D, Appleton D. Data on the distribution of fibre types in thirty-six human muscles: an autopsy study. Journal of the Neurological Sciences 18: 111-129, 1973
Kasch, FW, Wallace JP, Van Camp SP. Effects of 18 years of endurance exercise on the physical work capacity of older men. Journal of Cardiac Rehabilitation 5: 308-312, 1985
Katsuta S, Kanao Y, Aoyagi Y. Is exhaustive training adequate preparation for endurance performance? European Journal of Applied Physiology 58: 68-73, 1988
Kavanagh T, Shephard RJ. Can regular sports participation slow the aging process? Some further data on Masters athletes. Physician and Sportsmedicine 18 (6): 94-104, 1990
Kay C, Shephard RJ. On muscle strength and the threshold of anaerobic work. Internationale Zeitschrift für Angewandte Physiologie 27: 311-328, 1969
Kilbom A. Circulatory adaptation during static muscular contractions: a review. Scandinavian Journal of Work and Environmental Health 2: 1-13, 1976
Klein C, Cunningham DA, Paterson DH, Taylor AW. Fatigue and recovery contractile properties of young and elderly men. European Journal of Applied Physiology 57: 684-690, 1988
Kuno S, Katsuta S, Inouye T, Anno I, Matsumoto K, et al. Relationship between MR relaxation time and muscle fiber composition. Radiology 169: 567-568, 1988
Kuno S, Katsuta S, Akisada M, Anno I, Matsumoto K. Effect of strength training on the relationship between magnetic response relaxation time and muscle fibre composition. European Journal of Applied Physiology 61: 33-36, 1990
LaForest S, St-Pierre DMM, Cyr J, Gayton D. Effects of age and regular exercise on muscle strength and endurance. European Journal of Applied Physiology 60: 104-111, 1990
Larsson L. Morphological and functional characteristics of the ageing skeletal muscle in man: a cross-sectional study. Acta Physiologica Scandinavica (Suppl. 457): 1-36, 1978
Larsson L. Physical training effects on muscle morphology in sedentary males at different ages. Medicine and Science in Sports and Exercise 14: 203-206, 1982
Larsson L. Histochemical characteristics of human skeletal muscle

during aging. Acta Physiologica Scandinavica 117: 469-471, 1983
Larsson L, Edstrom L. Effects of age on enzyme-histochemical fibre spectra and contractile properties of fast- and slow-twitch skeletal muscles in the rat. Journal of the Neurological Sciences 76: 69-89, 1986
Larsson L, Grimby G, Karlsson J. Muscle strength and speed of movement in relation to age and muscle morphology. Journal of Allied Physiology 46: 451-456, 1979
Larsson L, Karlsson J. Isometric and dynamic endurance as a function of age and skeletal muscle characteristics. Acta Physiologica Scandinavica 104: 129-136, 1978
Larsson L, Sjodin B, Karlsson J. Histochemical and biochemical changes in human skeletal muscle with age in sedentary males age 22-65 years. Acta Physiologica Scandinavica 103: 31-39, 1978
Layman DK, Hegarty PVJ, Swan PB. Comparison of morphological and biochemical parameters of growth in rat skeletal muscles. Journal of Anatomy 130: 159-171, 1980
Lennmarken C, Bergman T, Larsson J, Larsson L-E. Skeletal muscle function in man: force, relaxation rate, endurance and contraction time - dependence on sex and age. Clinical Physiology 5: 243-255, 1985
Lewkowicz SJ. Motor innervation of the gastrocnemius muscle of wobbler and dystrophic mice. Journal of the Neurological Sciences 43: 405-419, 1979
Lexell J, Henriksson-Larsen K, Winblad B, Sjostrom M. Distribution of different fiber types in human skeletal muscles: effects of aging studied in whole muscle cross-sections. Muscle and Nerve 6: 588-595, 1983a
Lexell J, Henriksson-Larsen K, Sjostrom M. Distribution of different fibre types in human skeletal muscles: 2. A study of cross-sections of whole m. vastus lateralis. Acta Physiologica Scandinavica 117: 115-122, 1983b
Lexell J, Downham D, Sjostrom M. Distribution of different fibre types in human skeletal muscles: a statistical and computational study of the fibre type arrangement in M. Vastus Lateralis of young healthy males. Journal of the Neurological Sciences 65: 353-365 1984
Lexell J, Downham D, Sjostrom M. Distribution of different fibre types in human skeletal muscles: fibre type arrangement in m. vastus lateralis from three groups of healthy men between 15 and 83 years. Journal of the Neurological Sciences 72: 211-222, 1986
Lexell J, Taylor CC, Sjostrom M. What is the cause of the ageing atrophy? Total number, size and proportion of different fiber types studied in whole vastus lateralis muscle from 15- to 83-year old men. Journal of the Neurological Sciences 84: 275-294, 1988
Lithell H, Lindgarde F, Hellsing K, Lundqvist G, Nygaard E, et al. Body weight, skeletal muscle morphology, and enzyme activities in relation to fasting serum insulin concentration and glucose tolerance in 48-year-old man. Diabetes 30: 19-25, 1981
MacDougall JD, Sale DG, Elder GCB, Sutton JR. Muscle ultrastructural characteristics of elite powerlifters and bodybuilders. European Journal of Applied Physiology 48: 117-126, 1982
MacLennan WJ, Hall MRP, Timothy JI, Robinson M. Is weakness in old age due to muscle wasting? Age and Ageing 9: 188-192, 1980
Mainwood GW, Renaud JM. The effect of acid-base balance on fatigue of skeletal muscle. Canadian Journal of Physiology and Pharmacology 63: 403-416, 1985
McCarter R. Effects of age on contraction of mammalian skeletal muscle. In Kalkor & Dibattista (Eds) Aging in muscle, pp. 1-22, Raven Press, New York, 1978
McDonagh MJN, White MJ, Davies CTM. Different effects of ageing on the mechanical properties of human arm and leg muscles. Gerontology 30: 49-54, 1984
McGlynn GH. Strength and endurance gains and their relationships. Internationale Zeitschrift für Angewandte Physiologie 26: 323-329, 1968
Mertens DJ, Shephard RJ, Kavanagh T. Long-term exercise therapy for chronic obstructive lung disease. Respiration 35: 96-107, 1978
Meyer RA, Terjung RL. Differences in ammonia and adenylate metabolism in contracting fast and slow muscle. American Journal of Physiology 237: C111-C118, 1979
Montegriffo VME. Height and weight of a United Kingdom adult population with a review of the anthropometric literature. Annals of Human Genetics 31: 389-399, 1968
Moritani T, deVries HA. Potential for gross muscle hypertrophy in older men. Journal of Gerontology 35: 672-682, 1980
Murray MP, Gardner GM, Mollinger LA, Sepic SB. Strength of isometric and isokinetic contractions. Knee muscles of men aged 20 to 86. Physical Therapy 60: 412-419, 1980
Murray MP, Duthie EH, Gambert SR, Sepic SB, Mollinger LA. Age-related differences in knee muscle strength in normal women. Journal of Gerontology 40: 275-280, 1985
Myers SJ, Sullivan WP. Effect of circulatory occlusion on time to muscular fatigue. Journal of Applied Physiology 24: 54-59, 1968
Nelson RM, Soderberg GL, Urbscheit NL. Alteration of motor-unit discharge characteristics in aged humans. Physical Therapy 64: 29-34, 1984
Nygaard E. Skeletal muscle fibre characteristics in young women. Acta Physiologica Scandinavica 112: 299-304, 1981
Nygaard E, Sanchez J. Intramuscular variation of fiber types in the brachial biceps and the lateral vastus muscles of elderly men: how representative is a small biopsy sample? Anatomical Record 203: 451-459, 1982
Orlander J, Kiessling K-H, Larsson L, Karlsson J, Aniansson A. Skeletal muscle metabolism and ultrastructure in relation to age in sedentary men. Acta Physiologica Scandinavica 104: 249-261, 1978
Orlander J, Aniansson A. Effects of physical training on skeletal muscle metabolism and ultrastructure in 70- to 75-year-old men. Acta Physiologica Scandinavica 109: 149-154, 1980
Overstall PW, Exton-Smith AN, Imms FJ, Johnson AL. Falls in the elderly related to postural imbalance. British Medical Journal 1: 261-264, 1977
Overstall PW. Prevention of falls in the elderly. Journal of the American Geriatrics Society 28: 481-484, 1980
Parízková J. Impact of age, diet, and exercise on man's body composition. Annals of the New York Academy of Sciences 110: 661-674, 1963
Parízková J, Eiselt E, Sprynarova S, Wachtlova M. Body composition, aerobic capcity and density of muscle capillaries in young and old men. Journal of Applied Physiology 31: 323-325, 1971
Payton OD, Poland JL. Aging process: implications for clinical practice. Physical Therapy 63: 41-48, 1983
Pearson MB, Bassey EJ, Bendall MJ. Muscle strength and anthropometric indices in elderly men and women. Age and Ageing, 14: 49-54, 1985
Pestronk A, Drachman DB, Griffin JW. Effects of aging on nerve sprouting and regeneration. Experimental Neurology 70: 65-82, 1980
Peterson FR, Grandal H, Hansseb JW, Houd H. Effect of varying the number of contractions on dynamic muscle training. Arbeitsphysiologie 18: 468-473, 1961
Petrofsky JS, Lind AR. Aging, isometric strength and endurance, and cardiovascular responses to static effort. Journal of Applied Physiology 38: 91-95, 1975a
Petrofsky JS, Lind AR. Isometric strength, endurance, and the blood pressure and heart rate responses during isometric exercise in healthy men and women, with special reference to age and body fat content. Pflugers Archiv - European Journal of Physiology 360: 49-61, 1975b

Petrofsky JS, Lind AR. The relationship of body fat content to deep muscle temperature and isometric endurance in man. Clinical Science and Molecular Medicine 48: 405-412, 1975c
Pette D. Activity-induced fast to slow transitions in mammalian muscle. Medicine and Science in Sports and Exercise 16: 517-528, 1984
Polgar J, Johnson MA, Weightman D, Appleton D. Data in fibre size in thirty-six human muscles: an autopsy study. Journal of the Neurological Sciences, 19: 307-318, 1973
Pollock ML, Wilmore JH. Exercise in health and disease: evaluation and prescription for prevention and rehabilitation, Saunders, Philadelphia, 1990
Pollock ML, Miller HS, Wilmore J. Physiological characteristics of champion American track athletes 40 to 75 years of age. Journal of Gerontology 29: 645-649, 1974
Pollock ML, Foster C, Knapp D, Rod JL, Schmidt DH. Effect of age and training on aerobic capacity and body composition of master athletes. Journal of Applied Physiology 62: 725-731, 1987
Prince FP, Hikida RS, Hagerman FC, Staron RS, Allen WH. A morphometric analysis of human muscle fibers with relation to fiber types and adaptations to exercise. Journal of the Neurological Sciences 49: 165-179, 1981
Quetelet A. Sur l'homme et le développement de ses facultés, Bachelier Imprimeure-Libraire, Paris, 1835
Reijs JHO. Über die Veranderung der Kraft wahrend der Bewegung. Pflugers Archiv – European Journal of Physiology, 191: 234-257, 1921
Rikli R, Busch S. Motor performance of women as a function of age and physical activity level. Journal of Gerontology 41: 645-649, 1986
Rode A, Shephard J. Cardiorespiratory fitness of an Arctic community. Journal of Applied Physiology 31: 519-526, 1971
Romanul FCA. Capillary supply and metabolism of muscle fibers. Archives of Neurology 12: 497-509, 1965
Rosenheimer JL. Effects of chronic stress and exercise on age-related changes in end-plate architecture. Journal of Neurophysiology 53: 1582-1589, 1985
Rosler K, Hoppeler H, Conley KE, Claassen H, Gehr P, et al. Transfer effects in endurance exercise: adaptations in trained and untrained muscles. European Journal of Applied Physiology 54: 355-362, 1985
Royce J. Isometric fatigue curves in human muscle with normal and occluded circulation. Research Quarterly 29: 204-212, 1958
Saltin B, Gollnick PD. Skeletal muscle adaptability. Significance for metabolism and performance.In Peacher et al. (Eds) Handbook of physiology, section 10: skeletal muscle, pp. 555-633, Americam Physiological Society, Washington, 1983
Saltin B, Nygaard E, Rasmussen B. Skeletal muscle adaptation in man following prolonged exposure to high altitude. Acta Physiologica Scandinavica 109: 31A, 1980
Sato T, Tauchi H. Age changes in human vocal muscle. Mechanisms of Ageing and Development 18: 67-74, 1982
Sato T, Akatsuka H, Kito K, Tokoro Y, Tauchi H, et al. Age changes in size and number of muscle fibers in human minor pectoral muscle. Mechanisms of Ageing and Development 28: 99-109, 1984
Scelsi R, Marchetti C, Poggi P. Histochemical and ultrastructural aspects of M. Vastus Lateralis in sedentary old people (age 65-89 years). Acta Neuropathologica 51: 99-105, 1980
Secher NH, Rorsgaard S, Secher O. Contralateral influence on recruitment of curarized muscle fibres during maximal voluntary extension of the legs. Acta Physiologica Scandinavica 103: 456-462, 1978
Shephard RJ. Some determinants of continuous and intermittent handgrip endurance. Spor Hekim Derglisi 9: 89-103, 1974
Shephard RJ. Physical activity and aging, 2nd ed., Croom Helm Publishing, London, 1987
Shephard RJ. Body composition in biological anthropology, Cambridge University Press, London, 1991
Shephard RJ, Rode A. Acculturation and the biology of aging. In Fortuine (Ed.) Circumpolar health '84, pp. 45-48, University of Washington Press, Seattle, 1985
Sica REP, McComas AJ, Upton ARM, Longmire D. Motor unit estimations in small muscles of the hand. Journal of Neurology, Neurosurgery and Psychiatry 37: 55-67, 1974
Silbermann M, Finkelbrand S, Weiss A, Gershon D, Reznick A. Morphometric analysis of aging skeletal muscle following endurance training. Muscle and Nerve 6: 136-142, 1983
Simonson E. Physical fitness and work capacity of older men. Geriatrics 2: 110-119, 1947
Sjodin B. Lactate dehydrogenase in human skeletal muscle. Acta Physiologica Scandinavica (Suppl. 436): 1-32, 1976
Sjogaard G. Muscle enzyme activities in relation to maximal oxygen uptake. Acta Physiologica Scandinavica 112: 12A, 1981
Sjostrom M, Angquist K-A, Rais O. Intermittent claudication and muscle fiber fine structure: correlation between clinical and morphological data. Ultrastructural Pathology 1: 309-326, 1980
Sjostrom M, Angquist K-A, Bylund A-C, Friden J, Gustavsson L, et al. Morphometric analyses of human muscle fiber types. Muscle and Nerve 5: 538-553, 1982a
Sjostrom M, Kidman S, Henriksson J, Larsen K, Angquist K-A. Z- and M-band appearance in different histochemically defined types of human skeletal muscle fibers. Journal of Histochemistry and Cytochemistry 30: 1-11, 1982b
Sperling L. Evaluation of upper extremity function in 70-year-old men and women. Scandinavian Journal of Rehabilitation Medicine 12: 139-144, 1980
Stainsby WN. Biochemical and physiological bases for lactate production. Medicine and Science in Sports and Exercise 18: 341-343, 1986
Stalberg E, Fawcett PRW. Macro EMG in healthy subjects of different ages. Journal of Neurology, Neurosurgery and Psychiatry 45: 870-878, 1982
Stalberg E, Borges O, Ericsson M, Essen-Gustavsson B, Fawcett PRW, et al. The quadriceps femoris muscle in 20–70-year-old subjects: relationship between knee extension torque, electrophysiological parameters, and muscle fiber characteristics. Muscle and Nerve 12: 382-389, 1989
Stebbins CL, Schultz E, Smith RT, Smith EL. Effects of chronic exercise during aging on muscle and end-plate morphology in rats. Journal of Applied Physiology 58: 45-51, 1985
Steen B, Bruce A, Isaksson B, Lewin T, Svanborg A. Body composition in 70-year old males and females in Gothenburg, Sweden: a population study. Acta Medica Scandinavica (Suppl. 611): 87-112, 1977
Syrovy I, Gutmann E. Changes in speed of contraction and ATPase activity in striated muscle during old age. Experimental Gerontology 5: 31-35, 1970
Tauchi H, Yoshioka T, Kobayashi H. Age changes of skeletal muscles of rats. Gerontologia 17: 219-227, 1971
Tesch PA. Skeletal muscle adaptations consequent to long-term heavy resistance exercise. Medicine and Science in Sports and Exercise (Suppl. 20): S132-S134, 1988
Tesch P, Sjodin B, Thorstensson A, Karlsson J. Muscle fatigue and its relation to lactate accumulation and LDH activity in man. Acta Physiologica Scandinavica 103: 413-420, 1978
Thorstensson A. Muscle strength, fibre types and enzyme activities in man. Acta Physiologica Scandinavica (Suppl. 443): 1-45, 1976
Thorstensson A, Sjodin B, Tesch P, Karlsson J. Actomyosin ATPase, myokinase, CPK and LDH in human fast and slow twitch muscle fibres. Acta Physiologica Scandinavica 99: 225-229, 1977
Tinetti ME, Speechley M, Ginter SF. Risk factors for falls among elderly persons living in the community. New England Journal of Medicine 319 : 1701-1709, 1988
Tomonaga M. Histochemical and ultrastructural changes in sen-

ile human skeletal muscle. Journal of the American Geriatrics Society 25: 125-131, 1977

Tuffery AR. Growth and degeneration of motor end-plates in normal cat hind limb muscles. Journal of Anatomy 110: 221-247, 1971

Tzankoff SP, Norris AH. Effect of muscle mass decrease on age-related BMR changes. Journal of Applied Physiology 43: 1001-1006, 1977

Ufland JM. Einfluss des Lebensalters, Geschlechts, der Konstitution und des Berufs auf die Kraft verschiedener Muskelgruppen: I. Mitteilung: über den Einfluss des Lebensalters auf die Muskelkraft. Arbeitsphysiologie 6: 653-663, 1933

Vandervoort AA, McComas AJ. Contractile changes in opposing muscles of the human ankle joint with aging. Journal of Applied Physiology 61: 361-367, 1986

Wada M, Katsuda S. Changes in myoproteins following long-term with high intensity endurance training in rats. Japanese Journal of Physical Education 32: 221-229, 1988

Walter SD, Hart LE, Sutton JR, McIntosh JM, Gauld M. Training habits and injury experience in distance runners: age- and sex-related factors. Physician and Sportsmedicine 16: 101-113, 1988

Wernig A, Carmody JJ, Anzil AP, Hansert E, Marciniak M, et al. Persistence of nerve sprouting with features of synapse remodelling in soleus muscles of adult mice. Neuroscience 11: 241-253, 1984

Wernig, Herrera AA. Sprouting and remodelling at the nerve-muscle junction. Progress in Neurobiology 27: 251-291, 1986

Wernig A, Pecot-Dechavassine M, Stover H. Sprouting and regression of the nerve at the frog neuromuscular junction in normal conditions and after paralysis with curare. Journal of Neurocytology 9: 277-303, 1980

Young A. Exercise physiology in geriatric practice. Acta Medica Scandinavica (Suppl. 711): 227-232, 1986

Young A, Stokes M, Crowe M. The relationship between quadriceps size and strength in elderly women. Clinical Science 63: 35P-36P, 1982

Young A, Stokes M, Crowe M. Size and strength of the quadriceps muscles of old and young women. European Journal of Clinical Investigation 14: 282-287, 1984

Zackin MJ, Meredith CN. Protein metabolism in aging: effects of exercise and training. In Harris & Harris (Eds) Physical activity, sport and aging, pp. 271-236, Center for Study of Aging, Albany, 1989

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