

Current Concepts

The Effects of Aging and Training on Skeletal Muscle

Donald T. Kirkendall,*† PhD, and William E. Garrett, Jr.,‡ MD, PhD

*From the *Department of Physical and Occupational Therapy and the ‡Division of Orthopaedic Surgery, Duke University Medical Center, Durham, North Carolina*

ABSTRACT

Aging results in a gradual loss of muscle function, and there are predictable age-related alterations in skeletal muscle function. The typical adult will lose muscle mass with age; the loss varies according to sex and the level of muscle activity. At the cellular level, muscles lose both cross-sectional area and fiber numbers, with type II muscle fibers being the most affected by aging. Some denervation of fibers may occur. The combination of these factors leads to an increased percentage of type I fibers in older adults. Metabolically, the glycolytic enzymes seem to be little affected by aging, but the aerobic enzymes appear to decline with age. Aged skeletal muscle produces less force and there is a general "slowing" of the mechanical characteristics of muscle. However, neither reduced muscle demand nor the subsequent loss of function is inevitable with aging. These losses can be minimized or even reversed with training. Endurance training can improve the aerobic capacity of muscle, and resistance training can improve central nervous system recruitment of muscle and increase muscle mass. Therefore, physical activity throughout life is encouraged to prevent much of the age-related impact on skeletal muscle.

Biologic aging results in an unavoidable decrease in physiologic capacity. A decline in all the major systems (for example, cardiovascular, metabolic, respiratory, and neuromuscular) contributes to weakness, fatigue, and slowing of movement that have been the hallmarks of aging. These

changes limit the ability of the elderly to perform activities of daily living and their ability to exercise.⁴⁶ The purpose of this paper is to characterize the structural and physiologic changes that occur to the neuromuscular system with aging and to discuss briefly the response of skeletal muscle in the elderly to exercise and specific training.

Before any discussion of aging and skeletal muscle, certain variables must be considered so that accurate conclusions can be drawn. Habitual activity is inversely related to age, that is, younger people are more active than older people.⁸ With any reported body changes it must be considered whether neuromuscular responses are a natural outcome of aging or simply a reflection of the age-related decline in physical activity. Other confounding factors include the chronologic age of the subjects, body composition, sex, body part studied, and how these all interact with the physical activity level.

BODY COMPOSITION

Aging usually leads to an increase in the amount of body fat and a decrease in fat-free lean body mass of a person. These changes are not inevitable. Muscle occupies about 40% of the body fat-free mass and changes in the fat-free mass will reflect, in large part, changes in the muscle mass.¹¹

Alterations in muscle mass reflect other changes. For example, protein in the lean muscle compartment decreases, but protein in the lean, nonmuscle compartment does not decrease.¹⁹ With the loss of muscle mass we would expect a loss of potassium. Potassium loss in men is rapid between the ages of 41 and 60, and in women potassium losses are most rapid after the age of 60.²⁷ There is a concomitant parallel loss of calcium suggesting that a relationship exists between the loss of muscle mass and the reduction in bone density. In addition, aging results in declines in total body water,⁴⁸ creatinine excretion,²⁵ and basal metabolic rate.⁵⁶

† Address correspondence and reprint requests to Donald T. Kirkendall, PhD, Box 3435, Duke University Medical Center, Durham, NC 27710.

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As muscle mass decreases with age it is related to osteoporosis,⁵⁰ frequency of falls,⁵² and hip fractures.⁷ Maintaining a balance between fat and muscle mass throughout life is crucial because the loss of muscle mass is implicated in variables of metabolic rate and physical activity, and increases in body fat are associated with type II diabetes, hypertension, certain cancers, and coronary artery disease.⁴⁸ Muscle loss leads to a reduction in muscle function; therefore, muscle mass should be maintained throughout life to maintain function.

MORPHOLOGIC CHANGES IN MUSCLE

Cadaveric studies show that overall cross-sectional area of muscle declines with age.⁴¹ The only possible explanations for this reduction in mass are 1) a reduction of the volume of individual fibers, 2) a reduction of the total number of fibers, or 3) a combination of both.

Interpretations are difficult because aging affects muscle fiber types differently. The slow-contracting type I fiber is small, has a low tension output, but is highly resistant to fatigue because type I fibers have numerous, large mitochondria that contain the enzymes of the Krebs cycle and the electron transport chain. The type I fiber is also effective at metabolizing fats. The fast-contracting type IIB fiber is larger, has a large tension output, but has poor resistance to fatigue. The type IIA fiber is intermediate in size and function.

AGE-RELATED CHANGES IN MUSCLE FIBER DISTRIBUTION

The earliest studies on aging and muscle fiber composition suggested that type I fiber percentage increases with aging.^{29,37} Subjects in their 20s had 39% type I fibers while subjects in their 60s had 66% type I fibers. Others, particularly Grimby and Saltin,³¹ were in disagreement.⁵¹ They examined muscle biopsies of persons in the age range of 66 to 100 years and found no age-related changes in type I distribution. There are two possible reasons for the discrepancy. First, there is the inherent problem with the biopsy approach. Only small samples of a portion of the muscle were removed. The second reason is the age of the subjects. Because younger subjects were excluded, it may have been possible that the age-related changes in type I distribution of these older subjects had already occurred and would show no further changes with aging.

The problems with the biopsy approach were addressed by Lexall et al.,^{39,40} who sampled whole muscle tissue from autopsy material. They found that the type I distribution was 49% for men in their 20s; men in their 50s and late 70s had 52% and 51% type I fibers, respectively. These are small changes, probably reflective of the likely changes in fiber distribution.

Connective tissue in skeletal muscle of animals has been shown to increase between 20% and 40% from adult to old age.¹ Since connective tissue occupies only 2% of the cross-sectional area of muscle, any changes in connective tissues are unlikely to have an effect on force production or overall mass of skeletal muscle.²³

Age-Related Changes in the Size of Muscle Fibers

Type I fibers are little affected by aging. Numerous studies have failed to show any significant change in the cross-sectional area of type I fibers.^{5,30,37,41} Type II fibers seem to bear the brunt of age-related changes in cross-sectional area. Biopsy studies show 15% to 25% reductions in the type IIA and IIB cross-sectional area.¹⁸ The work of Lexall et al.⁴¹ indicated that there was a 26% reduction in the size of type II fibers from age 20 to 80. A large proportion of the age-related loss of muscle mass is the result of the reduction in type II muscle fiber size.³⁸

Age-Related Changes in the Number of Muscle Fibers

Lexall and associates³⁹⁻⁴¹ offer the most definitive work regarding muscle fiber number and aging. They counted the number of fibers from the vastus lateralis muscle of six men between the ages of 30 and 74 and found that the oldest man had about 25% fewer muscle fibers than the youngest man did. They demonstrated that the relationship between age and fiber number was not linear. Using their (quadratic) relationship, Lexall and associates suggested that the loss of muscle fibers begins at about age 25 and that total fiber number would decrease about 39% by age 80. A similar study looking at the pectoralis muscle of women showed that fiber numbers began to decrease at 60 years of age (similar to the onset of protein losses), with a 25% reduction in fiber number by the 7th decade.⁵¹

Age-Related Denervation of Muscle Fibers

Why should muscle fiber number decrease with age? There are two explanations. First, there might be damage to the fibers with no subsequent regeneration. There is little evidence that this is true.^{4,6,30} Second, the neural input may be disrupted.⁵⁸ There is ample evidence that this occurs.

Electromyographic data show an age-related decrease in the number of active motor units^{14,15} and that the low-threshold motor units become larger.⁵⁴ Stalberg and Fawcett⁵⁵ used EMG amplitude and muscle fiber density to show that the increase in the fiber density of a motor unit was indicative of an age-related organization of the motor unit. They estimated that about 25% of the motor neurons were nonfunctional. The increase in motor neuron density seemed to be due to new collaterals innervating denervated fibers. Cross-sections of whole muscle from older subjects showed a high density of motor units that was not seen in their younger counterparts.

To summarize, the loss of muscle mass is secondary to age-related denervation of muscle fibers, particularly the denervation of type II fibers. Denervation removes the trophic influence on the fibers and leads to atrophy. In an attempt to minimize fiber loss, collaterals from the type I motor neurons expand to some of the denervated type II fibers. This expands the type I motor neuron unit at the expense of the type II fibers. As a result, muscle mass of the elderly is smaller, has a higher proportion of type I fibers, and is weaker because of the loss of type II fibers.

METABOLIC CHANGES WITH AGE

Performance of endurance activities is the direct result of oxygen delivered to the central organs by the cardiovascular system and peripheral use of oxygen by the working muscles. Endurance capacity of humans as measured by maximal oxygen consumption declines about 10% per decade.^{33,49} Some of the decline is related to central delivery of oxygen, but the peripheral muscle tissue carries some of the responsibility.

The cellular aspects of energy metabolism are best studied by determining the activity of marker enzymes of anaerobic and aerobic production of adenosine triphosphate (ATP). Many studies have shown little or no age-related changes in enzyme activity of anaerobic energy production. Enzymes such as myosin adenosine triphosphatase (ATPase), myokinase, phosphofructokinase, hexokinase, and phosphorylase show minimal, if any, difference in activity from younger subjects.^{4,18,30} Resting high-energy phosphate concentration is also similar between the young and old.⁴⁴ Total phosphagen content is due less to the previously mentioned muscle mass decreases; any changes in the elderly are due to a change in their activity pattern rather than any inherent age-related modifications.^{6,12,44,48}

The lack of changes in enzymes of anaerobic energy production in the elderly is in contrast with the enzymes of aerobic energy production. Early studies indicated that aging apparently had little effect on the aerobic enzyme activities of the Krebs cycle, oxidative phosphorylation, and β -oxidation of fats.^{30,37,45} More recent work shows that markers of aerobic energy production are about 25% lower in the elderly.^{16,21} The impact of physical activity makes for a difficult research design problem in cross-sectional studies. The inclusion criteria for subjects in the early studies were quite broad and included subjects who were still very active. The inclusion criteria for the later projects were very strict in the classification of the "sedentary" category.⁴⁸ It seems safe to conclude that while aging has minimal effect on the activities of enzymes of anaerobic energy production, the activity of enzymes of aerobic energy production are reduced in comparison with younger subjects.

MECHANICAL CHANGES WITH AGE

The maximum force a muscle can generate increases in parallel with muscle mass: the larger the muscle, the greater the force capacity. As muscle mass declines with age, there should be a loss of total force production. Less clear are changes in specific force (newtons of force per fiber cross-sectional area). In some animal studies no change in specific force has been seen,^{13,43} while another report shows up to a 20% decrease in specific force with aging.⁵⁹ In humans, the reduction in force has been reported to be caused by changes in muscle architecture^{42,59}; in animal preparations, the reduction in specific force has been attributed to a decrease in the contractile protein.²³

The speeds of muscle contraction time and muscle re-

laxation time lengthen with age.^{26,57} The mechanism for these changes has yet to be described, but any changes to these phases of muscle contraction influences the efficiency of the older adult to perform activities.

Total power output of muscle (measured in watts) is a function of the average force developed and the shortening velocity. Slow-contracting type I muscle fibers exhibit low force output and a slow maximum velocity of shortening, while fast-contracting type II muscle fibers show the opposite. The velocity of contraction is correlated with myosin ATPase activity.⁹ Enzymatically, aging skeletal muscle shows little change in the activity of the contractile enzymes. It is difficult to measure maximum velocity of contraction for intact muscles in humans, but contraction velocity may be estimated isokinetically. Older adults show a loss in strength with aging, but estimated maximum velocity of shortening changes little, which leads to some overall reduction of power output.³⁶

The resistance to fatigue is determined by the response of the muscle to repetitive stimulation. There seems to be a reduction in the enzymatic activities for aerobic energy production. Any reduction in the major aerobic pathways will decrease the ability of the muscle to adequately produce ATP. As a result, the muscle's ability to sustain power (resist fatigue) is reduced with aging.

Another interesting phenomenon of aging has to do with injury to muscle fibers after activity. Muscles develop tension as they shorten, but tension may also be developed as muscles lengthen. During these lengthening eccentric contractions, more force may be developed with the use of fewer muscle fibers. Thus, the force per unit of active fiber area is greater, leading to the possibility of injury. Older mice⁶⁰ and humans²² show greater amounts of ultrastructural injury with eccentric contraction than their paired younger counterparts. Recovery from contraction is delayed in both older mice²³ and humans.³⁴

ADAPTATION OF AGING MUSCLE TO ENDURANCE TRAINING

Although an early study on physical training in the elderly failed to show significant improvements in maximal oxygen consumption,³⁵ later studies dispute this finding. Training programs must systematically manipulate training frequency, intensity, and duration to be effective and to improve endurance at any age. When following accepted training prescriptions, maximal oxygen consumption can increase by 20% to 30% in adults 60 to 80 years of age.^{16,32,53} Any peripheral adaptations are related to the muscle's ability to generate energy aerobically.

These systemic adaptations also reflect an improvement of the respiratory capacity of the mitochondria of the recruited muscle cells,^{16,29} increased capillary density,^{3,20} and a shift in the metabolic profile of the type IIB fibers toward that of type IIA fibers.² Markers of the Krebs cycle, electron transport, and β -oxidation increased between 25% and 50% in elderly men and women who trained for 10 months.¹⁶ Improvements for men were about 70% (Krebs cycle and electron transport markers) and 15% (β -oxidation) greater than for women.

Training results in little change in the activity of glycolytic enzymes. As in younger people, endurance training has little effect on enzymes of anaerobic energy production in older people.

An important adaptation to endurance training is the increase in capillary density in the active muscle. An increase in the capillary-to-fiber ratio decreases the diffusion distance for oxygen and may be one of the most important adaptations to physical training.

The percentage of type I muscle fibers does not change with training in either younger or older subjects.^{17,47} With endurance training, type II fibers become more aerobic. Thus, an increase in the type IIA fibers with a similar decrease in the type IIB fibers can occur.¹⁷

Controlled studies on the effect of years of training by humans have not been reported. The best descriptions involve follow-up assessment of competitive athletes who have continued to train after their competitive years have passed.^{47,49} With training, the expected age-related decline in $\dot{V}O_2\text{max}$ was reduced or even eliminated. At the cellular level there did not appear to be an age-related reduction in the ability to use the oxygen delivered. If young and old runners are matched on performance, the older athletes do have a lower $\dot{V}O_2\text{max}$, but do have elevated aerobic enzymes and capillary density.¹⁸

ADAPTATIONS OF AGING MUSCLE TO RESISTANCE TRAINING

Endurance training improves the ability of muscle to meet metabolic demands. Thus, metabolic proteins (enzymes) are the main beneficiaries of this training. Resistance training improves the ability of muscle to develop tension. Contractile proteins that affect tension production are the main beneficiaries of resistance training. Table 1 gives a summary of the effects of training of muscle adaptation in the aging.

For resistance training to be effective, the program cannot be a short-term one. Studies of 12- to 24-week training periods at lower intensities tend to show some improvements in muscle mass, but no increase in strength. However, if the training program is longer and of sufficient intensity and duration, the elderly may also demonstrate adaptations typically seen in younger participants.

In a study of 66-year-old men who trained at 80% of

1 RM (repetition maximum) for 12 weeks, strength improved by roughly 5.0% per day, similar to data on younger subjects.²⁸ Cross-sectional area of muscles of both the thigh and of type I and II fibers increased significantly. In addition, capillary density and some aerobic enzymes also were increased. When a protein-calorie supplement was given to half the men, that group had larger increases in the cross-sectional area of muscle.

There is a concern as to the ability of the oldest old to improve their functional capacity. Muscle strength relates to walking speed, the ability to move from sitting to standing positions, and stair climbing.¹⁰ The loss of type II muscle fibers from normal aging, disuse, and disease that causes weakness will increase the risk of fall and fracture. If skeletal muscle in the frail elderly could be improved, more functional aspects of their lives would benefit. Fiat-arone and colleagues²⁴ directed a progressive resistance program for institutionalized men and women in their 10th decades. After 8 weeks, the absolute amount of weight lifted improved by nearly 175% and the cross-sectional area of the thigh muscle increased by 15%. If done with sufficient intensity, even the frail elderly responded to a resistance training program much like their younger counterparts.

SUMMARY

Much of the decline in skeletal muscle function with aging seems to be related to the progressive reduction in the demands on muscle and thus does not appear to be inevitable. The adaptations that are evident with aging can be minimized with training (Table 1). Aging muscle responds to training in a similar manner to the muscle of younger subjects. Endurance training leads to an increase in $\dot{V}O_2\text{max}$, capillarization, and aerobic enzyme activity. Resistance training can improve central nervous system recruitment of muscle, hypertrophy, and force output. In either case, skeletal muscle responds according to the demands placed on it. Reduce the demand on skeletal muscle and it will adapt to the new lower requirement; increase the demands and the declines due to aging can be minimized, if not eliminated. A lifetime of physical activity appears to be the critical factor in maintaining the structure and function of skeletal muscle.

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TABLE 1
Summary of Muscle Adaptations to Aging and Training by the Elderly

Variable	Aging	Training
Muscle mass	Increase	Increase or no change
Type I%	Increase	No change
Type II%	Decrease	No change
Type I area	No change	Increase
Type II area	Decrease	Increase
Oxidative capacity	Decrease	Increase
Glycolytic capacity	No change	No change
Capillary density	Decrease	Increase
Contraction time	Increase	Decrease or no change
Relaxation time	Increase	Decrease or no change
Shortening velocity	No change	Increase

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