Published in final edited form as:

Clin J Sport Med. 2008 November ; 18(6): 501–507. doi:10.1097/JSM.0b013e3181845f1c.

The aging of elite male athletes: age-related changes in performance and skeletal muscle structure and function

John A. Faulkner, PhD, FACSM, Carol S. Davis, BS, Christopher L. Mendias, PhD, ATC, and Susan V. Brooks, PhD

Department of Molecular & Integrative Physiology, University of Michigan Medical School, Biomedical Sciences Research Building, Ann Arbor, MI, 48109-2200

Abstract

Objective—The paper addresses the degree to which the attainment of the status as an elite athlete in different sports ameliorates the known age-related losses in skeletal muscle structure and function.

Design—The retrospective design, based on comparisons of published data on former elite and masters athletes and data on control subjects, assessed the degree to which the attainment of ‘elite and masters athlete status’ ameliorated the known age-related changes in skeletal muscle structure and function.

Setting—Institutional.

Participants—Elite male athletes.

Interventions—Participation in selected individual and team sports.

Main Outcome Measurements—Strength, power, VO₂ max and performance.

Results—For elite athletes in all sports, as for the general population, age-related muscle atrophy begins at about 50 years of age. Despite the loss of muscle mass, elite athletes who maintain an active life style age gracefully with few health problems. Conversely, those who lapse into inactivity regress toward general population norms for fitness, weight control, and health problems. Elite athletes in the dual and team sports have careers that rarely extend into the thirties.

Conclusions—Life long physical activity does not appear to have any impact on the loss in fiber number. The loss of fibers can be buffered to some degree by hypertrophy of fibers that remain. Surprisingly, the performance of elite athletes in all sports appears to be impaired before the onset of the fiber loss. Even with major losses in physical capacity and muscle mass, the performance of elite and masters athletes is remarkable.

INTRODUCTION

The age-related changes in the skeletal muscles of both genders, but generally men, have been described in considerable detail by us (1–4) and others (5–14). In contrast, the elite, world-class athletes have received much less recent attention (15–20). As a result of societal restrictions on participation by women in a wide variety of sports that extended until the passage of Title IX (the Educational Amendments Act of 1972 that prohibited gender-based discrimination in federal funded educational programs, including athletics), little published data exist on the performance characteristics of female ‘elite’ athletes, or on the age-related

changes that occur in their skeletal muscles (4). Consequently, this review will be restricted to elite male athletes. We do so in the hope that the conclusions will be applicable to both genders.

AGE-RELATED ATROPHY OF SKELETAL MUSCLES

The basic contractile unit of a skeletal muscle is the fiber and in humans the number of fibers per muscle varies from hundreds for the lumbrical muscles that abduct and adduct the fingers to hundreds of thousands of fibers for large thigh muscles (Figure 1). Fibers are highly adaptable in both structure and function to changes in the type and frequency of contractions as a result of habitual patterns of physical activity and to aging. For a given fiber, the mass is the product of the density of the tissue, the cross-sectional area, and the length of the fiber. The mass of a whole skeletal muscle is then obtained by multiplying the average fiber mass by the number of fibers in the muscle. Fiber lengths increase during growth and development, but stabilize after maturity. The only subsequent changes in muscle fiber lengths occur with significant hypertrophy, or atrophy (21). Branching of fibers may occur, but only under specific circumstances (22). From birth through to adulthood, the numbers of fibers within mammalian skeletal muscles remain constant (23). Consequently, through to adulthood, any change in muscle mass occurs as a result of atrophy or hypertrophy of single fibers. A loss of the mass of muscles occurs with a decrease in dietary input or in physical activity, particularly a decrease in physical activities that incur decreased loading of the muscles (24;25). Conversely, hypertrophy of select muscle groups occurs with weight training and with repeated drills and performance in events that involve the 'overloading' of muscle groups (25–27).

Data on males ranging in age from children to age 90 indicate that the average number of muscle fibers in vastus lateralis muscles of male cadavers (12) and the average number of motor units in the extensor digitorum brevis muscles (7) are remarkably stable through to ~50 years of age and then show a linear decline throughout the remainder of the life span. Despite the stability in the best fit lines, the range in the number of fibers at 20 to 50 years of age is from 400,000 to 900,000 and the number of motor units from 125 to 325. Obviously, these are very different muscles with regard to both mass and function, but the two muscles show very similar age-related changes. The large variation in the number of muscle fibers amongst individuals may result from polymorphisms in genes that determine fiber number during embryonic development such as myostatin (28) and IGF-1 (29). These polymorphisms may also explain some of the individual variability observed in the performance capabilities of elite athletes. Fifty percent of the average of 600,000 fibers present at age 50 are lost by age 80 (Figure 1). After age 80, the range in the number of fibers is from 200,000 to 350,000 and for motor units 1 to 125. The loss of fibers in skeletal muscles appears to result directly from a loss of motor units. The loss in motor unit numbers have been measured with indirect techniques in several skeletal muscles of humans (7;30) and by direct measurements in rats (31). The loss of muscle fibers and of motor units have very similar onsets and slopes in rats and humans. The loss in whole motor units appears to arise from age-related changes in the nervous system, likely beginning with the anterior horn cell (32). Some of the fibers left denervated as a motor unit is lost appear to be re-innervated by slow type 1 fiber motor nerves by axonal sprouting (33). The age-related loss of fibers (12) and motor units (7;30) appears to involve most if not all of the muscles in the mammalian organism (34;35). All of the fibers in a single motor unit are generally of the same fiber type and the loss of motor units with aging appears to involve exclusively those composed of fast powerful type 2 fibers (31). The loss of the type 2 motor units explains in large measure the greater loss in power than occurs in force during aging (1;8;9;14). With fewer fast motor units to recruit, movements become less rapid and less powerful (6;36).

There are no direct measurements of fiber numbers, or in the number of motor units in the skeletal muscles of aging elite athletes in any sport. Despite the absence of direct measurements of these phenomena on this specific subpopulation, comparisons between elite athletes and untrained normal subjects show parallel declines in strength (Figure 2), power (Figure 3) and VO_2max (ml/kg/min) (Figure 4). The similarity in the time of the onset and the rate of the declines for each of these three variables strongly support that the muscles of elite athletes undergo similar changes to those observed in control male subjects in fiber number and that the time of the onset of fiber loss is comparable. The critical difference between the elite athlete and the untrained and frequently less athletically gifted untrained subject is the substantial difference in each of these variables. As shown for VO_2max , but equally true for both strength and power, a cessation of high quality, regular training, results in a gradual loss of the 'elite status'. Interestingly, the rate of decline for the mass lifted in the 'clean & jerk' of the weight lifter and the running time for the Masters Records of the marathon run (Figure 5) are in reasonable agreement with the losses in muscle power (Figure 3). Consequently, despite the absence of direct data on elite athletes, the assumption that the achievement of elite status in specific sports does not protect the skeletal muscles of elite athletes from the gradual losses in the number of fibers and motor units measured in control male subjects appears to be reasonable and explains a substantial portion of the losses in athletic performance observed with the aging of the elite athlete. In particular, the loss in power (Figure 3) is troublesome for elite athletes, whose performance depends primarily on high power output. High power output is required not only during a single contraction as in the shot put, discus throw, or weight lifting (Figure 5); but also during the repeated contractions required for the 'dashes' in the sprint, or long distance running (Figure 5); or the explosive power for the darting, dodging, leaping, or evasive maneuvers required in almost all of the dual and team sports.

Repair and regeneration of muscle tissue is vital to the long term viability of the elite athlete at all ages, but particularly as they age. Satellite cells are stem cells within muscle fibers that provide a source of nuclei for muscle fibers. Satellite cells normally exist in a quiescent state, between the muscle fiber plasma membrane and the basal lamina. Following damage to muscle fibers, such as the damage that occurs following lengthening contractions (37), satellite cells become activated, migrate to the site of injury, proliferate, and fuse with the damaged fiber to regenerate the sarcomeric structure of the damaged region (Figure 7). The satellite cells also repopulate the nuclei lost as a result of injury. Thus, the activity of satellite cells is critical in the adaptations that occur in skeletal muscles with exercise training. There is a progressive decrease in the proliferative capacity of satellite cells with aging (38). Compared with younger individuals, the activated satellite cells of elderly individuals have a diminished ability to fuse with existing myofibers in response to exercise training (39). There is also an age-related decline in the density of satellite cells surrounding type 2 muscle fibers, and an increase in the density of satellite cells surrounding type I muscle fibers (40). In addition to the relationship between aging and satellite cell activity, the myonuclear domain, the volume of cytosol under the control of a single myonucleus, decreases with age in both type I and type 2 muscle fibers (40). Compared with young subjects, both myofibrillar and non-myofibrillar protein synthesis rates are decreased in elderly subjects (41).

PERFORMANCE OF ELITE MALE ATHLETES DURING AGING

Regardless of the age of the elite athlete, or the sport in which he participates, all volitional bodily movements arise from the activation of groups of fibers innervated by the same motor neuron, and thus termed a motor unit. When activated, all muscle fibers contract and attempt to shorten. During a specific activation, whether a muscle shortens, stays at the same length (isometric), or is lengthened, depends on the interaction between the strength of the

contraction and the load on the muscle (37). Each sport has a specific pattern of bodily movements associated with it and the success of the athlete in the sport is determined by his skill and efficiency in performing these specific movements. The movements in all sports involve various combinations of isometric, shortening, and lengthening contractions. 'Weight lifting' by its very name, involves primarily shortening contractions of the muscles involved in the 'lifting' and isometric contractions of a wide variety of other muscles that provide a 'force platform' for the 'lift'. The lengthening contraction is the only type of contraction that produces a self-induced injury to muscle fibers (37)(Figure 7). To avoid lengthening contractions during the lowering of weights, the 'lifter' frequently walks out from under the weight and drops it. Similarly, the propulsion of the body through the water in 'swimming' is achieved solely by shortening contractions with the return of the limbs following the pull, or the kick, largely unloaded lengthening. In contrast, the running, jumping, twisting and turning movements required in all team and dual sports involve significant contributions of all three types of contractions. Thus, the success of an elite athlete in a particular sport depends upon the skill, strength and power, and in some activities, the efficiency, with which various muscle groups are recruited to perform the bodily movements required of the sport.

For many elite athletes, maturation occurs 'early' and peak performance may be attained in the late teens, whereas 'later maturers' will not reach peak performance capacity until the mid- to late twenties. Regardless of the age at which peak performance is attained, most elite athletes begin to show some decline in performance by their early thirties (Figures 5 & 6). Despite the onset of irretrievable declines, some very 'late maturers' and some exceptionally gifted athletes may still out-perform younger men at an elite level of performance at up to forty years of age while already showing declines from their own peak levels (Figure 6). The Masters Performance Records in the 'clean & jerk' lift provides ample evidence of the impressive capabilities of the lifters who maintain a surprising percentage of their maximum capabilities throughout their life span (Figure 5). Similarly, a highly conditioned endurance runner at age 74 years has recorded a time of less than 3 hours for the marathon run. In many sports, the elite athlete is simply competing against the clock, such as in running or cross-country skiing, or for the distance he can put a shot put or throw a discus or javelin. Records show age-related declines in these sports, but the decline in performance is due solely to declines in coordination, strength, power and overall skill of the individual competitor. In contrast, in the competitive sports of badminton, squash, or tennis, or the team games of basketball, football, hockey, rugby, or soccer, as one ages, the older elite athlete is playing against younger and younger opponents. This age-differential has a greater and greater effect as the elite athlete ages.

Elite Masters weight lifters lose lean lower limb volume at a slower rate, but normalized isometric strength (Figure 2) and power (Figure 3) in specific muscle groups at a slightly higher rate than age-matched untrained control subjects (36). The competitive weight lifting performances for 'clean & jerk' and 'snatch' display declines with the age of the Masters Lifters (Figure 5) at a rate closely related to the loss in peak power (Figure 3). The lift accomplished by the over 80 year old for the 'clean & jerk' of 55 kg (121 lbs) is truly remarkable, when one considers the difficulties encountered by many frail 80 year olds in performing the normal activities of daily living (42;43). Along with running speed, the VO_2max has since the 1930s been widely accepted as the premier measure of the capability of elite distance runners based initially on reports that distance runner Lash had a VO_2max of 81.5 ml/kg/min, compared with values of 47.8 ml/kg/min for untrained young men (44). The VO_2max values for distance runners who maintain high intensity training programs decline at comparable rates to those of sedentary age-matched men. In contrast, former elite distance runners who become sedentary show rapid losses in VO_2max and eventually have values that are indistinguishable from those of untrained subjects (Figure 4).

In many professional team sports, declines in performance are measurable by statistics that are applicable for all players. Professional basketball is renowned for the mass of statistics that are kept on each player during each game. Consequently, for three elite basketball players, Kareem Abdul-Jabbar, Michael Jordan, and Charles Barkley, the game by game and year by year statistics on the 'percentage of free throws made' and the 'total points scored per game' were averaged and graphed against age from their first year as a professional player until their retirement (Figure 6). The 'free throw percentage' represents an uncontested, high skill, motor control task, whereas the 'total points scored per game' represents a highly contested, motor control task of shooting field goals during full court play. To permit each of the two variables to be graphed together, each variable was expressed as a percentage of the highest value achieved during each player's long and illustrious career, which extended far beyond the norm. The 'percentage of free throws made' remained high throughout their careers, at between 85% and 90% of the highest value for each of the three players with no sign of an age dependent decline. The variable that showed the greatest decline for each player was the 'points scored per game'. The points scored per game decreased to 30% of the highest value for Abdul-Jabbar, 60% for Jordan, and 55% for Barkley. When Abdul-Jabbar's percentage was corrected for his 55% decrease in playing time with increasing age, his percentage is about the same as that of the other two players. A reasonable conclusion is that the ability to shoot accurately is not lost at least prior to age 40, but under circumstances of being guarded by ever younger players, the 40-year-old player frees himself for a high quality shot about half as frequently as he did when he was younger. Similar problems are encountered, to an even greater extent in the physically more demanding collision sports of football, hockey, rugby, and soccer. In these sports, injuries are more common than in basketball (45) and the number of games played in a given season fluctuates rather dramatically as a result. Despite these intermittent injuries, the length of the career in a variety of team sports is quite similar, although hockey legend, Gordie Howe, actually played until he was 50 years of age.

GENE THERAPY & DOPING

Gene therapy, while offering much hope for the treatment of genetic diseases of skeletal muscle (46), has potential as an effective, yet hard to detect, form of doping for athletes. Using a vector, such as a virus, a modified sequence of DNA can be inserted into the genome of muscle fibers. This modified DNA sequence could be used to control genes that affect athletic performance. Two potential targets for athletes wishing to increase muscle mass are IGF-1 and myostatin. IGF-1 promotes muscle hypertrophy by increasing satellite cell proliferation and muscle protein synthesis (47;48). Infecting rats with an adeno-associated virus (AAV) that increased the production of IGF-1 in muscle resulted in a 15% increase in muscle mass and a 17% increase in maximum isometric force (49). In contrast, myostatin is a hormone that causes muscle atrophy by decreasing satellite cell proliferation and muscle protein synthesis (50;51). Injecting mice with AAV that contained genes to induce the expression of a myostatin inhibitor, follistatin, resulted in an increase of greater than 50% mass of hind limb muscles and of grip strength (52). For athletes that compete in aerobic sports, two other genes are strong candidates for illegal doping, erythropoietin and the cytosolic form of phosphoenolpyruvate carboxykinase (PEPCK). Injecting muscles of rats with a lentivirus designed to synthesize erythropoietin increased hematocrit values from 46% to 69% and enhanced oxidative capacity (53), while overexpression of PEPCK in the muscles of transgenic mice produced animals with amazing endurance (54). During treadmill endurance tests, PEPCK over-expressing mice ran nearly 30X farther than controls before reaching exhaustion, and in strenuous running tests PEPCK mice ran 56% faster and 66% longer than controls with no change in blood lactate values, whereas in control mice, blood lactate went from 4.9 mM before the test to 8.1 mM after the test. Although the PEPCK over-expressing mice were transgenic and not produced using a gene therapy

approach, virus containing the same modified DNA sequence could be made for use as a doping agent. Traditional anti-doping strategies look for the presence of exogenous drugs or hormones in blood or urine samples, but gene doping modifies the expression of endogenous hormones and proteins, some of which are not present in blood or urine. Thus, virtually no methods are currently available for detecting the presence of gene doping.

CLINICAL IMPLICATIONS OF THE AGE-RELATED CHANGES IN MUSCLE

Structural changes in height, weight, lean body mass and VO_2max of both trained and untrained men as they age are shown in Table 1. The oft observed lack of any change in height, but significant increase for untrained men in body mass of 18% by age 50 that was sustained through age 75, is quite evident. By age 84 these untrained subjects display a 6% loss of height and a 16% loss of body mass. In such cross-sectional studies, the losses of height and body mass with aging have frequently been attributed to the higher mortality rates for the heavier males (55). For the elite trained males, neither height nor body mass changed appreciably from 22 to 75 years of age. The percentage of body mass that was fat varied markedly from a value of 9–10% for young and adult endurance-trained athletes to 28% for 65 year old untrained men, while lean body masses of all groups were remarkably consistent at 56 to 59 kg for all groups. The exception was that of the untrained 50-year olds with a lean body mass of 68 kg. The VO_2max was highest for the young endurance-trained athletes with a value of 69 ml/kg/min, although this group mean is considerably below the value of 81.5 ml/kg/min reported by Robinson (44) for a world record holder in the two mile run. Both of these values for VO_2max are considerably higher than the 40 ml/kg/min reported for untrained young men and 27 ml/kg/min for untrained 65 year old men (Table 1). A life long commitment to either long distance or sprint running clearly has a substantial impact on body composition and aerobic capacity that if continued carries over into the status of the individual in old age.

For the sports medicine physician, the recognition of the losses that will occur, even in elite athletes as they age offers the opportunity to anticipate these changes and aid the elite athlete in adjusting to the changes. The immutable loss of the fast type 2 fibers (56), results inevitably in a gradual decrease in strength and power beginning at about age 40 (36). For aging elite athletes in dual or team contact sports, this translates immediately into a greater incidence of lengthening contractions as stronger, younger opponents overpower them in one on one confrontations. Furthermore, fibers in the muscles of older men, even elite older athletes, are more susceptible to contraction-induced injury than those of younger men (37). Training with protocols that include lengthening contractions, termed plyometric training (57;58) increase strength and power and prevent subsequent injury, but such plyometric training must be undertaken with care and usually trained supervision, to prevent injury.

SUMMARY

Although the decrease in the performance of the elite athlete after age 40 can be explained by the loss in the powerful type 2 fiber motor units with subsequent muscle atrophy and a loss in muscle power, the decline in performance, particularly before age 40 is more difficult to explain. Clearly when playing against younger opponents, declines for most elite athletes are evident in the early to late thirties. One possibility is that prior to the actual loss of motor units, there are subtle changes in fine motor control that are not picked up by the gross motor control tests used to assess age-related changes. Despite the inevitable changes that occur in muscle structure and function with aging, the elderly highly trained and highly skilled elite athlete is still able to compete into his eighties in a wide variety of sports activities at a level unattainable by less gifted and less well trained young people (Figures 5–6). Finally, an almost entirely unexplored area is the patterns of diet, health habits, weight

control, or of the regularity, amount and intensity of physical activity of the elite athletes following retirement.

Acknowledgments

The authors acknowledge the support of the NIH-NIA grant P01 AG20591 (JAF), Nathan Shock Center Contractility Core NIA AG13283 (JAF) and the Regenerative Sciences Training Program NIDDK DK070071 (post-doctoral fellowship CLM).

Reference List

1. Brooks SV, Faulkner JA. Skeletal muscle weakness in old age: underlying mechanisms. *Med Sci Sports Exerc.* 1994; 26:432–439. [PubMed: 8201898]
2. Brooks, SV.; Faulkner, JA. Effects of aging on the structure and function of skeletal muscle. In: Roussos, C., editor. *The Thorax*. New York: Marcel Dekker, Inc; 1995. p. 295-312.
3. Faulkner JA, Brooks SV, Zerba E. Muscle atrophy and weakness with aging: contraction-induced injury as an underlying mechanism. *J Gerontol A Biol Sci Med Sci.* 1995; 50 Spec No:124–129.
4. Faulkner JA, Larkin LM, Claflin DR, et al. Age-related changes in the structure and function of skeletal muscles. *Clin Exp Pharmacol Physiol.* 2007; 34:1091–1096. [PubMed: 17880359]
5. Akima H, Kano Y, Enomoto Y, et al. Muscle function in 164 men and women aged 20–84 yr. *Med Sci Sports Exerc.* 2001; 33:220–226. [PubMed: 11224809]
6. Bassey EJ, Fiatarone MA, O'Neill EF, et al. Leg extensor power and functional performance in very old men and women. *Clin Sci (Lond).* 1992; 82:321–327. [PubMed: 1312417]
7. Campbell MJ, McComas AJ, Petito F. Physiological changes in ageing muscles. *J Neurol Neurosurg Psychiatry.* 1973; 36:174–182. [PubMed: 4708452]
8. Frontera WR, Hughes VA, Lutz KJ, et al. A cross-sectional study of muscle strength and mass in 45- to 78-yr-old men and women. *J Appl Physiol.* 1991; 71:644–650. [PubMed: 1938738]
9. Goodpaster BH, Park SW, Harris TB, et al. The loss of skeletal muscle strength, mass, and quality in older adults: the health, aging and body composition study. *J Gerontol A Biol Sci Med Sci.* 2006; 61:1059–1064. [PubMed: 17077199]
10. Grimby G, Saltin B. The ageing muscle. *Clin Physiol.* 1983; 3:209–218. [PubMed: 6347501]
11. Holloszy JO. Workshop on Sarcopenia: Muscle Atrophy in Old Age. *J Gerontol.* 1995; 50A:1–161.
12. 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. *J Neurol Sci.* 1988; 84:275–294. [PubMed: 3379447]
13. Trappe S, Gallagher P, Harber M, et al. Single muscle fibre contractile properties in young and old men and women. *J Physiol.* 2003; 552:47–58. [PubMed: 12837929]
14. Young A, Stokes M, Crowe M. The size and strength of the quadriceps muscles of old and young men. *Clin Physiol.* 1985; 5:145–154. [PubMed: 3888498]
15. Marti B, Howald H. Long-term effects of physical training on aerobic capacity: controlled study of former elite athletes. *J Appl Physiol.* 1990; 69:1451–1459. [PubMed: 2262469]
16. Marti B. Health benefits and risks in sports: the other side of the coin. *Schweiz Rundsch Med Prax.* 1989; 78:290–294. [PubMed: 2711074]
17. Pollock ML, Miller HS Jr, Wilmore J. Physiological characteristics of champion American track athletes 40 to 75 years of age. *J Gerontol.* 1974; 29:645–649. [PubMed: 4421428]
18. Pollock ML, Foster C, Knapp D, et al. Effect of age and training on aerobic capacity and body composition of master athletes. *J Appl Physiol.* 1987; 62:725–731. [PubMed: 3558232]
19. Pollock ML, Mengelkoch LJ, Graves JE, et al. Twenty-year follow-up of aerobic power and body composition of older track athletes. *J Appl Physiol.* 1997; 82:1508–1516. [PubMed: 9134900]
20. Robinson S, Dill DB, Robinson RD, et al. Physiological aging of champion runners. *J Appl Physiol.* 1976; 41:46–51. [PubMed: 972131]
21. Maxwell LC, Faulkner JA, Hyatt GJ. Estimation of number of fibers in guinea pig skeletal muscles. *J Appl Physiol.* 1974; 37:259–264. [PubMed: 4851910]

22. Blaivas M, Carlson BM. Muscle fiber branching--difference between grafts in old and young rats. *Mech Ageing Dev.* 1991; 60:43–53. [PubMed: 1745064]
23. Gollnick PD, Timson BF, Moore RL, et al. Muscular enlargement and number of fibers in skeletal muscles of rats. *J Appl Physiol.* 1981; 50:936–943. [PubMed: 7228766]
24. Haddad F, Roy RR, Zhong H, et al. Atrophy responses to muscle inactivity. I. Cellular markers of protein deficits. *J Appl Physiol.* 2003; 95:781–790. [PubMed: 12716870]
25. MacDougall JD, Elder GC, Sale DG, et al. Effects of strength training and immobilization on human muscle fibres. *Eur J Appl Physiol Occup Physiol.* 1980; 43:25–34. [PubMed: 7371625]
26. Frontera WR, Meredith CN, O'Reilly KP, et al. Strength conditioning in older men: skeletal muscle hypertrophy and improved function. *J Appl Physiol.* 1988; 64:1038–1044. [PubMed: 3366726]
27. Jones DA, Rutherford OM. Human muscle strength training: the effects of three different regimens and the nature of the resultant changes. *J Physiol (Lond).* 1987; 391:1–11. [PubMed: 3443943]
28. Mendias CL, Marcin JE, Calderon DR, et al. Contractile properties of EDL and soleus muscles of myostatin-deficient mice. *J Appl Physiol.* 2006; 101:898–905. [PubMed: 16709649]
29. Mitchell PJ, Johnson SE, Hannon K. Insulin-like growth factor I stimulates myoblast expansion and myofiber development in the limb. *Dev Dyn.* 2002; 223:12–23. [PubMed: 11803566]
30. Doherty TJ, Brown WF. The estimated numbers and relative sizes of thenar motor units as selected by multiple point stimulation in young and older adults. *Muscle Nerve.* 1993; 16:355–366. [PubMed: 8455648]
31. Kadhiresan VA, Hassett CA, Faulkner JA. Properties of single motor units in medial gastrocnemius muscles of adult and old rats. *J Physiol (Lond).* 1996; 493(Pt 2):543–552. [PubMed: 8782115]
32. Kanda K, Hashizume K, Nomoto E, et al. The effects of aging on physiological properties of fast and slow twitch motor units in the rat gastrocnemius. *Neurosci Res.* 1986; 3:242–246. [PubMed: 3010198]
33. Brown MC, Holland RL, Hopkins WG. Motor nerve sprouting. *Annu Rev Neurosci.* 1981; 4:17–42. [PubMed: 7013635]
34. Alnaqeeb MA, Goldspink G. Changes in fibre type, number and diameter in developing and ageing skeletal muscle. *J Anat.* 1987; 153:31–45. [PubMed: 3429325]
35. Hooper AC. Length, diameter and number of ageing skeletal muscle fibres. *Gerontology.* 1981; 27:121–126. [PubMed: 7239169]
36. Pearson SJ, Young A, Macaluso A, et al. Muscle function in elite master weightlifters. *Med Sci Sports Exerc.* 2002; 34:1199–1206. [PubMed: 12131263]
37. Faulkner JA. Terminology for contractions of muscles during shortening, while isometric, and during lengthening. *J Appl Physiol.* 2003; 95:455–459. [PubMed: 12851415]
38. Conboy IM, Rando TA. Aging, stem cells and tissue regeneration: lessons from muscle. *Cell Cycle.* 2005; 4:407–410. [PubMed: 15725724]
39. Petrella JK, Kim JS, Cross JM, et al. Efficacy of myonuclear addition may explain differential myofiber growth among resistance-trained young and older men and women. *Am J Physiol Endocrinol Metab.* 2006; 291:E937–E946. [PubMed: 16772322]
40. Verdijk LB, Koopman R, Schaart G, et al. Satellite cell content is specifically reduced in type II skeletal muscle fibers in the elderly. *Am J Physiol Endocrinol Metab.* 2007; 292:E151–E157. [PubMed: 16926381]
41. Toth MJ, Matthews DE, Tracy RP, et al. Age-related differences in skeletal muscle protein synthesis: relation to markers of immune activation. *Am J Physiol Endocrinol Metab.* 2005; 288:E883–E891. [PubMed: 15613683]
42. Fried LP. Conference on the physiologic basis of frailty. *Aging (Milano).* 1992; 4:251–265. [PubMed: 1420409]
43. Verdery RB. Failure to thrive in the elderly. *Clin Geriatr Med.* 1995; 11:653–659. [PubMed: 8556693]
44. Robinson S. Experimental studies of physical fitness in relation to age. *Arbeitsphysiologie.* 1938; 10:251–323.

45. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train.* 2007; 42:311–319. [PubMed: 17710181]
46. Gregorevic P, Blankinship MJ, Chamberlain JS. Viral vectors for gene transfer to striated muscle. *Curr Opin Mol Ther.* 2004; 6:491–498. [PubMed: 15537050]
47. Allen RE, Boxhorn LK. Regulation of skeletal muscle satellite cell proliferation and differentiation by transforming growth factor-beta, insulin-like growth factor I, and fibroblast growth factor. *J Cell Physiol.* 1989; 138:311–315. [PubMed: 2918032]
48. Bark TH, McNurlan MA, Lang CH, et al. Increased protein synthesis after acute IGF-I or insulin infusion is localized to muscle in mice. *Am J Physiol.* 1998; 275:E118–E123. [PubMed: 9688882]
49. Lee S, Barton ER, Sweeney HL, et al. Viral expression of insulin-like growth factorI enhances muscle hypertrophy in resistance-trained rats. *J Appl Physiol.* 2004; 96:1097–1104. [PubMed: 14766764]
50. McCroskery S, Thomas M, Maxwell L, et al. Myostatin negatively regulates satellite cell activation and self-renewal. *J Cell Biol.* 2003; 162:1135–1147. [PubMed: 12963705]
51. Welle S, Bhatt K, Pinkert CA. Myofibrillar protein synthesis in myostatin-deficient mice. *Am J Physiol Endocrinol Metab.* 2006; 290:E409–E415. [PubMed: 16219664]
52. Haidet AM, Rizo L, Handy C, et al. Long-term enhancement of skeletal muscle mass and strength by single gene administration of myostatin inhibitors. *Proc Natl Acad Sci U S A.* 2008; 105:4318–4322. [PubMed: 18334646]
53. Seppen J, Barry SC, Harder B, et al. Lentivirus administration to rat muscle provides efficient sustained expression of erythropoietin. *Blood.* 2001; 98:594–596. [PubMed: 11468155]
54. Hakimi P, Yang J, Casadesus G, et al. Overexpression of the cytosolic form of phosphoenolpyruvate carboxykinase (GTP) in skeletal muscle repatterns energy metabolism in the mouse. *J Biol Chem.* 2007; 282:32844–32855. [PubMed: 17716967]
55. Paffenbarger RS Jr, Hyde RT, Wing AL, et al. Physical activity, all-cause mortality, and longevity of college alumni. *N Engl J Med.* 1986; 314:605–613. [PubMed: 3945246]
56. Lexell J. Human aging, muscle mass, and fiber type composition. *J Gerontol.* 1995; 50A:11–16.
57. Wilson GJ, Newton RU, Murphy AJ, et al. The optimal training load for the development of dynamic athletic performance. *Med Sci Sports Exerc.* 1993; 25:1279–1286. [PubMed: 8289617]
58. Svantesson U, Grimby G, Thomee R. Potentiation of concentric plantar flexion torque following eccentric and isometric muscle actions. *Acta Physiol Scand.* 1994; 152:287–293. [PubMed: 7872006]
59. Heath GW, Hagberg JM, Ehsani AA, et al. A physiological comparison of young and older endurance athletes. *J Appl Physiol.* 1981; 51:634–640. [PubMed: 7327965]
60. Rader EP, Song W, Van Remmen H, et al. Raising the antioxidant levels within mouse muscle fibres does not affect contraction-induced injury. *Exp Physiol.* 2006; 91:781–789. [PubMed: 16675501]
61. Kasch FW, Boyer JL, Van CS, et al. Cardiovascular changes with age and exercise. A 28-year longitudinal study. *Scand J Med Sci Sports.* 1995; 5:147–151. [PubMed: 7552757]
62. Maud PJ, Pollock ML, Foster C, et al. Fifty years of training and competition in the marathon: Wally Hayward, age 70--a physiological profile. *S Afr Med J.* 1981; 59:153–157. [PubMed: 7006111]
63. Maughan RJ, Watson JS, Weir J. Strength and cross-sectional area of human skeletal muscle. *J Physiol (Lond).* 1983; 338:37–49. [PubMed: 6875963]
64. Coggan AR, Spina RJ, King DS, et al. Skeletal muscle adaptations to endurance training in 60- to 70-yr-old men and women. *J Appl Physiol.* 1992; 72:1780–1786. [PubMed: 1601786]
65. Korhonen MT, Cristea A, Alen M, et al. Aging, muscle fiber type, and contractile function in sprint-trained athletes. *J Appl Physiol.* 2006; 101:906–917. [PubMed: 16690791]

Figure 1.

The relationships between the number of motor units in the extensor digitorum brevis muscles and the age of men between five and 88 years of age (solid line) and between the total number of fibers in the vastus lateralis muscles and the age of the cadavers between 18 to 82 years of age (dashed line). The number of motor units remained constant from 5 years to 50 years of age, but then decreased linearly with a zero intercept at ninety-five years of age (modified from Campbell et al., 1973 with permission). Similarly, the average number of fibers in the vastus lateralis muscle did not change between 18 and 50 years of age, but by age 80, the mean number of fibers decreased to 50% of the number for younger men (modified from Lexell et al., 1988 with permission).

Figure 2.

The isometric knee strengths for untrained control subjects (solid line) and for elite Masters weight-lifters (dashed line) from 40 to 88 years of age were obtained from Pearson et al., 2002 with permission. Note: Surprisingly, the isometric knee strength of the untrained control subjects did not decrease with age, whereas the weight-lifters showed a decline.

Figure 3.

The peak power for untrained control subjects (solid line) and for elite Masters weight-lifters (dashed line) from 40 to 88 years of age were obtained from Pearson et al., 2002 with permission. Note that for peak power both untrained control subjects and the elite Masters weight-lifters showed declines in peak power with age, with the slope for the weight-lifters greater than that of the control subjects.

Figure 4.

VO_{2max} of sedentary non-athletes and endurance-trained elite runners of various ages were obtained from Heath (59) and Pollock (19). Note the gradual conversion of the VO_{2max} of an endurance-trained elite runner to that of a sedentary non-athlete over time as a result of the cessation of run-training (Pollock et al., 1997, with permission; Robinson et al., 1976, with permission).

Figure 5.

The performances of Masters athletes for the Marathon run and for weight-lifters for the 'clean & jerk'. The data for the marathon run were taken from the Alan L. Jones website [<http://home.stny.rr.com/alanjones/AgeGrade.html>] "Age graded running races" 2006. The Masters weight-lifting data was obtained from IWF Masters Records [<http://www.iwfmasters.net/records/iwf-men.pdf>] December 2006 (men's weight class 85 kg., clean & jerk).

Figure 6.

Average statistics for three basketball players; Kareem Abdul-Jabbar, Charles Barkley and Michael Jordan for the number of points per game (PPG) and for free throw percentage each expressed as a percentage of the highest value achieved during the players' careers. Note the consistency of the free throw percentage for each of the three players compared with the declining values for points per game (see text for explanation). Data were obtained from the nba.com website [<http://www.nba.com>].

Figure 7.

Schematic diagram of the sequence of events for a typical muscle fiber within a skeletal muscle exposed to a severe lengthening contraction protocol. The damaged fibers would be scattered throughout the injured muscle both singly and in small clusters. Each damaged fiber goes through this sequence of events, with full recovery of fibers in young animals complete within a few weeks and those in old animals requiring up to a month with the possibility of permanent damage (reproduced from Rader et al., 2006).

Table 1
Structural differences in skeletal muscles of untrained, sprint trained and endurance trained men

Status	Untrained				Sprint Trained		Endurance Trained	
	Young (n=25)	Adult (n=9)	Old (n=12)	Oldest Old (n=4)	Young (n=16)	Old (n=20)	Young (n=16)	Adult (n=16)
Age								
Reference #	63	59	64	36	65	65	59	59
Years	28 ± 1	50 ± 2	65 ± 1	84 ± 1	24 ± 1	75 ± 1	22 ± 1	59 ± 2
Height (cm)	174 ± 1	175 ± 3	178 ± 2	168 ± 4	178 ± 1	171 ± 1	176 ± 2	173 ± 1
Mass (kg)	72 ± 2	85 ± 4	81 ± 2	69 ± 8	77 ± 1	70 ± 2	65 ± 2 [‡]	65 ± 2 [‡]
Fat (%)	16 ± 1	20 ± 1	28 ± 1	--	17 ± 1	15 ± 1	9 ± 1 [‡]	10 ± 0 [‡]
LBM (kg)	59 ± 1	68 ± 2	59 ± 1	--	65 ± 3 [*]	56 ± 3 [*]	59 ± 1 [‡]	57 ± 2 [‡]
VO ₂ max (ml/kg/min)	40 [‡]	30 ± 1	27 ± 1	--	--	--	69 ± 1 [‡]	59 ± 1 [‡]

[‡] Data on endurance trained young and adult men different from untrained adult men.

^{*} LBM calculated from data on body mass and percent fat.

[‡] Data obtained from Heath et al., 1981.