4) AGING, EXERCISE AND SHORT TERM POWER

The effects of age and exercise on short term maximal performance: A model based on physiological systems.

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Maximal exercise of short duration is most often associated with sport performance, but is also a factor in many daily activities. Climbing stairs, hurrying across a busy street, or carrying a heavy grocery bag may represent short term maximal efforts, and the ability to perform these types of activities can have a major influence on the independence of older adults, and may serve as an index of an individual's ability to live independently. This type of performance has rarely been studied in older adults, primarily due to safety concerns. Therefore, we are limited to examining short term maximal performance in the elderly by first determining the physiological systems that limit such performance in general, and then examining the effect of aging on each individual system. Since it is often difficult to separate the effects of aging from those of disuse, we will assume that aging means sedentary aging. Subsequently, we will consider the effects of exercise on these systems.

Among the few studies of maximal short term performance in older adults, Makrides et al., (1985) showed that maximal power output for 30 seconds decreased with age. However, they also showed that lean thigh volume was highly correlated ($r = 0.84$) with power. Therefore, it may be that power would not have shown a decrease with age, if lean thigh volume had been controlled for statistically. In a subsequent investigation, Makrides et al., (1990) showed that 12 weeks of high-intensity training produced greater increases in total work accomplished in 30 seconds in old (60-70 year old, 12.5%) than young (20-30 year old, 8%) subjects. One interpretation of this greater increase in work after training is that the older subjects were more detrained at the beginning of the study, due to the effects of sedentary lifestyle. Also, the fact that the older subjects adapted at least as well as the younger subjects serves to refute the claim that aging alone reduces short term performance, because these older subjects retained the ability to adapt to a training stimulus.

General model of short term maximal performance

Maximal performance of short duration (i.e. 30 - 120 sec) is often thought of as "anaerobic", or not relying on aerobic energy production. However, Medbo and Tabuta (1989) have shown that aerobic processes accounted for 40% of

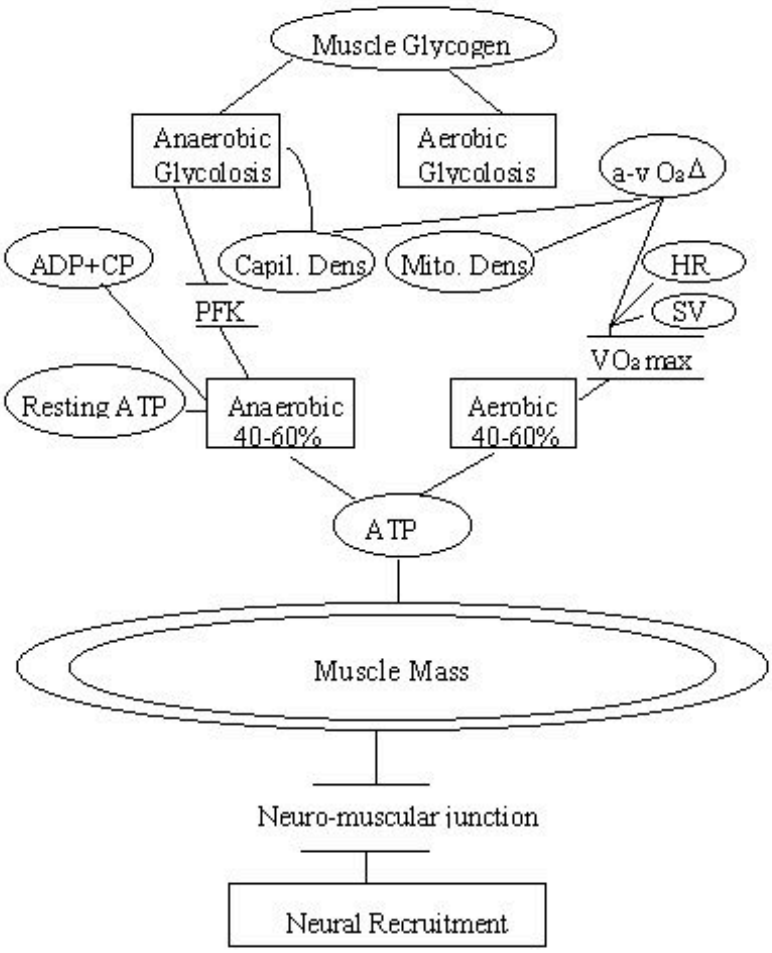
the energy produced in a 30 second maximal exercise bout, and 65% of the energy produced in a 2 minute maximal exercise bout. Therefore, the physiological mechanisms of both aerobic and anaerobic energy production must be accounted for in order to properly model short term maximal performance. Additionally, the ability to recruit muscular activity of high intensity may be limited by central neural drive and neural signal transmission.

Aerobic energy production is limited by VO2 max, the maximum ability of an individual to take in and use oxygen. VO2 max is limited by both the amount of oxygenated blood the cardiopulmonary system can provide to the working muscles and by the amount of oxygen the working muscles can extract from the blood. The delivery of oxygenated blood to the working muscles is limited by cardiac output, which is the product of heart rate and stroke volume. The ability of the working muscles to extract oxygen is characterized by the arterial-venous oxygen difference (a-v O2 difference) and is limited by muscle mass, capillary density, and mitochondrial density.

Anaerobic work capacity is defined as the maximum amount of work that can be produced from anaerobic energy systems (Green, 1994). These systems include provision and phosphorylation of ATP from 3 sources: (1) stored ATP, (2) ATP that is resynthesized via the ADP+CP to ATP reaction, and (3) ATP produced by anaerobic glycolosis. The limiting factors for anaerobic work capacity are muscle mass, and muscle metabolic characteristics. There are several critical or rate limiting metabolic characteristics for anaerobic energy production, including catalyzing enzymes and substrates:

1. Resting levels of ATP
2. ATPase, which is rate limiting for hydrolysis of ATP,
3. Resting creatine phosphate (CP) levels, which limits the total energy available via the ADP+CP to ATP pathway,
4. Creatine kinase (CK) which catalyzes and is thought to be rate limiting for ADP+CP to ATP reaction,
5. Muscle glycogen as a substrate for glycolosis,
6. Phosphorylase (PHOS) and phosfofructokinase (PFK), which are thought to be rate limiting for anaerobic glycolosis, and
7. Anaerobic work capacity may be indirectly dependent upon capillary density which facilitates diffusion of metabolic byproducts out of the muscle.

Figure 1 below presents a general model for maximal short term performance which is based on these physiological systems. At the center of the model is the active muscle mass, which is supplied with ATP from both aerobic and anaerobic systems, and which is recruited by neuromuscular signals. Next, we will examine the effects of aging on physiological systems.



Age related changes to the determinants of short term maximal performance

Many of the determinants for aerobic and anaerobic capacity change with age. Muscle mass, which is central to both aerobic and anaerobic capacity, decreases with sedentary aging, as shown by several investigations (Lexell et al, 1988, Coggan et al, 1992a, Gollnick et al, 1972, Larsson and Karlsson 1978, 1984, Grimby et al, 1983, Grimby et al, 1984, Lexell et al, 1983, Lexell et al, 1986). These decreases appear to occur primarily due to the loss of muscle fibers (Lexell et al, 1988), but are also due to atrophy of individual

fibers, particularly fast twitch muscle fibers (Lexell et al, 1988, Coggan et al, 1992a). Some early research has suggested that there is a selective loss of fast twitch muscle fibers with aging (Gollnick et al, 1972, Larsson and Karlsson 1978). If true, this selective loss of fiber could result in a relative decrease in anaerobic work capacity, greater than the amount of muscle atrophy. However, more recent work has not confirmed this specificity of fiber loss (Sato et al, 1984, Grimby et al, 1983, Grimby et al, 1984, Lexell et al, 1983, Lexell et al, 1986), and indicates a preservation of muscle fiber type distribution with aging.

In healthy sedentary individuals, VO2 max decreases about 1% per year (Heath et al, 1981). This decrease is related to decreased capacity in all of the systems that determine VO2 max: maximum heart rate, stroke volume, and a-v O2 difference. Maximum heart rate declines about one beat per year with age (Shephard, 1987). Although results regarding stroke volume are mixed, most investigations have shown that during exercise, stroke volume decreases with sedentary aging (Hagberg et al, 1985, Ogawa et al,1992). However, other investigations show that stroke volume is maintained or even increased (Rodeheffer et al, 1984). Similarly, a-v O2 difference decreases with age in sedentary people (Hagberg et al, 1985, Ogawa et al.,1992). This decrease is related to decreased capillary density, mitochondrial density, and muscle mass.

Early studies of mitochondrial density in skeletal muscle did not indicate any age-related decrease (Oralander et al, 1978, Larsson et al, 1978a, Larsson et al., 1978b, Grimby et al, 1982). These findings may have been influenced by the effects of an active older subject population. More recently, however, investigations of subjects that were truly sedentary showed a significant (25-40%) age-related decrease in three mitochondrial enzymes (SDH, CS, and b-HAD, Coggan et al, 1992a), and in in-vitro muscle oxygen uptake (Meredith et al, 1989).

Parizcova et al, (1971), demonstrated that the number of capillaries per unit of cross-sectional area did not change with age, but that, due to muscle fiber atrophy, there was a significantly lower capillary to fiber ratio in the older subjects. These findings were supported by those of Grimby et al, (1982). More recently, however, Coggan et al, (1992a) has shown that in truly sedentary subjects, both capillary density and capillary to fiber ratio decrease with aging (Coggan et al, 1992a).

Early studies of age-related effects on the anaerobic enzymes in skeletal muscle showed that the maximal activity of PFK, MK, and ATPase did not change with age, but that LDH decreased significantly (Oralander et al., 1978, Oralander et al, 1980, Larsson et al, 1978a, Larsson et al, 1978b). These findings were supported by Aniansson et al., (1981) except that they did not find an age related change in LDH. Grimby et al, (1982) found no age related decrease in the activity of HK and LDH. More recently, Coggan et al, (1992a) found no age related difference in phosphorylase, LDH, or PFK. Additionally, resting levels of ATP are not affected by age (Moller et al, 1980). Perhaps the most thorough investigation of glycolytic enzymes and aging was performed by Essen-Gustavsson and Borges (1986), who found no age-related change in HK, LDH, CK, or MK. Aside from early findings that LDH was reduced with aging, no age-related decrease in glycolitic enzyme activity or high energy phosphates has been demonstrated.

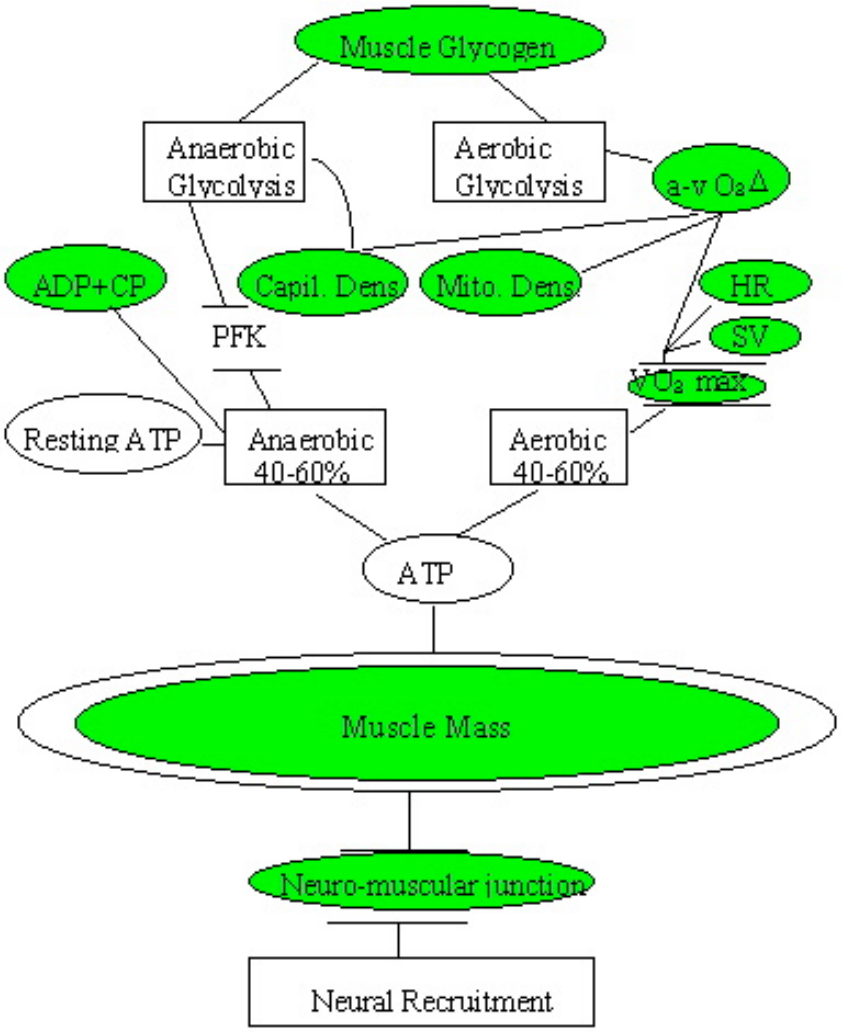
The findings regarding metabolism of glycogen might indicate that older individuals can metabolize muscle glycogen as well as younger individuals. Unfortunately, Meredith et al, (1989) have shown that older people have less muscle glycogen to metabolize. They showed that muscle glycogen in vastus lateralis was 61% higher in sedentary 24 year old men compared with sedentary 65 year old men.

Creatine phosphate concentrations have been shown to decrease slightly with respect to aging. Moller et al (1980) found a slight (5%) but significant decrease in creatine phosphate in skeletal muscle of men and women 52-79 years of age, compared with younger adults. McCully et al (1991) also found a small decrease in the creatine phosphate to inorganic phosphate ratio using magnetic resonance spectroscopy.

Neural activation of skeletal muscles may change with aging. Evidence to support this hypothesis stems from the finding that specific tension (i.e.; force per unit cross sectional area) is maintained with aging in muscle fibers in-vitro (McCarter 1978), but not in whole muscles in-vivo (Phillips et al, 1992). Since neural enervation is maintained (Phillips et al, 1992 and Walters et al, 1990) with respect to aging, it is possible that "age-related deficiencies of motor function are probably related to other factors, such as those associated with neuromuscular transmission or propagation of nerve impulses" (McCarter and McGee, 1987). Smith and Rosenheimer (1984) investigated neural transmission velocity and found no age related effects in rats. They concluded that "motor deficits during aging must be associated primarily with changes in central pathways or at the muscular apparatus." To test that

hypothesis, they examined architectural changes to the neuromuscular junctions (Rosenheimer and Smith 1985), and found age-related changes in nerve terminal branch number; the rate of changes in end-plate morphology of all muscles studied increased after 25 months of age. In rats, 25 months is equal to the mean lifespan, so it is unclear whether these changes occur as a linear function of aging, or as an end lifespan event. In summary, the neural signal to fire muscle contraction is maintained with age, but the ability to communicate that signal to the contractile machinery of the muscle may be compromised by changes at the motorneuron endplate.

Figure 2 below presents the effects of aging on the mechanisms of maximal short term performance. The shaded portions represent those systems in which maximal performance or function has been shown to decrease with age.



Next we will examine the effects of exercise on these systems in an attempt to determine if exercise can help maintain short term maximal performance with increasing age.

The age-related loss of muscle mass can be ameliorated with strength training (Brown et al, 1990, Charette et al, 1991, Fiatarone et al, 1990, Frontera et al, 1988). The subjects in the study of Frontera et al, (1988) increased muscle cross sectional area by 11% after 12 weeks of intense training, which is similar to the increases experienced by young adults for similar training (Hurley et al, 1991). The subjects in the study of Brown et al., (1990) trained only one arm for twelve weeks, and increased CSA in the trained arm by 17%. Charette et al, (1991) trained older women for 12 weeks. These women increased CSA by 20%. In all these studies, cross sectional area was increased due to hypertrophy of individual fibers, not by restoration of lost fibers. In an animal model study, Klitgarrd et al, (1989) found that weight training reduced the age-related decline in rat muscle.

In cross-sectional studies of VO2 max, older individuals who maintain vigorous endurance training, experience a decrease of about one-half of one percent per year, compared to younger adults (Heath et al, 1981, Ragers et al, 1990), or about half the decline experienced by sedentary adults. Some of this difference may be related to training between older and younger subjects. To control for training differences, Hagberg et al, (1985) matched older (56 year old) runners for training volume, intensity and 10k performance, with younger runners (25 years old) and still found a 9% difference, or 0.3%/year decrease in VO2 max. However, in a longitudinal investigation, runners who maintained their training over a ten year period did not exhibit any decrease in VO2 max (Pollock et al, 1987). Several of the runners in this study stopped training and they experienced a 14% decrease in VO2 max. This is interesting in that it is similar to the decrease in VO2 max experienced after only 56 days of detraining (Coyle et al, 1984). Taken together, these findings suggest that the effects attributed to aging in sedentary individuals and detraining in previously trained individuals may be difficult to separate.

With regard to the components of cardiac output, training has no effect on the age-related decrease in maximum heart rate (Rodeheffer et al, 1984, Hagberg et al, 1985, Shephard 1987). However, stroke volume is improved significantly with endurance training (Levy et al, 1993, Stratton et al, 1994). Specifically, training appears to enhance diastolic filling (Levy et al, 1993). In one of the best conceived investigations of training and hemodynamic function, Hagberg et al, (1985) studied older and younger runners who were

matched for performance, training intensity and training volume. Their findings indicate that the decreased VO2 max of the older runners was solely a function of decreased maximum heart rate, and that stroke volume and a-v O2 difference were similar to those of the younger runners.

The age related decline in mitochondrial density may be ameliorated with exercise training. Early studies found little or no change in markers of mitochondrial density (Aniansson and Gustafsson, 1981, Oralander and Aniansson, 1980, Souminen et al., 1977). However, these studies used exercise protocols that were relatively low intensity, and were maintained for only a brief time. Also, the subjects in these studies may have been moderately active rather than truly sedentary. More recently, Coggan et al., (1992b), screened carefully to obtain a truly sedentary subject population, and employed a vigorous exercise regime for 9 to 12 months. They found that this exercise significantly increased citrate synthase, the best predictor of mitochondrial density (29% in men and 17% in women). Furthermore, masters runners who were matched for training volume and intensity with young runners had similar CS activity (Coggan et al, 1993). Also, the primary cause of loss of mitochondrial function is thought to be the prolonged effects of oxidative free radical stress (Ames et al, 1995 review), therefore it is possible that chronic intake of antioxidants may preserve mitochondrial density independent of exercise.

The results of investigations of capillary density are similar to those of mitochondrial density. Exercise regimes that involve low intensity exercise for a short period, and those that trained active older people did not increase muscle capillary density (Aniansson and Gustafsson, 1981, Oralander and Aniansson, 1980, Denis et al, 1986). However, Coggan et al, (1992b) found significant increases in both capillaries per unit cross-sectional area (16% for men and 25% for women) and in capillaries per fiber (26% for men and 38% for women) when truly sedentary individuals performed more intense exercise for a longer period. Furthermore, masters runners who were matched for training volume and intensity with young runners had similar capillary density in capillaries per mm2 and a greater number of capillaries per fiber (Coggan et al., 1993). Interestingly, the strength training performed by the subjects of Frontera et al., (1988) elicited an unexpected increase in VO2 max, capillary to fiber ratio, and mitochondrial enzyme activity.

Muscle glycogen is increased in young people after exercise training, but has apparently only been investigated once, by Meredith et al, (1989). They showed that a relatively short (12 week), but fairly intense (70% of VO2 peak)

training program produced significant (27%) increase in muscle glycogen content. Even with this increase, the older subjects still had less muscle glycogen than either the trained or sedentary younger men. It remains to be determined if higher intensity or longer duration training would restore muscle glycogen content to levels equal to those of younger people.

The small age related decrease in creatine phosphate may be eliminated with training. Moller and Brandt (1982) showed that six weeks of cycle ergometer training increased creatine phosphate levels of 61-80 year old men to those of younger adults.

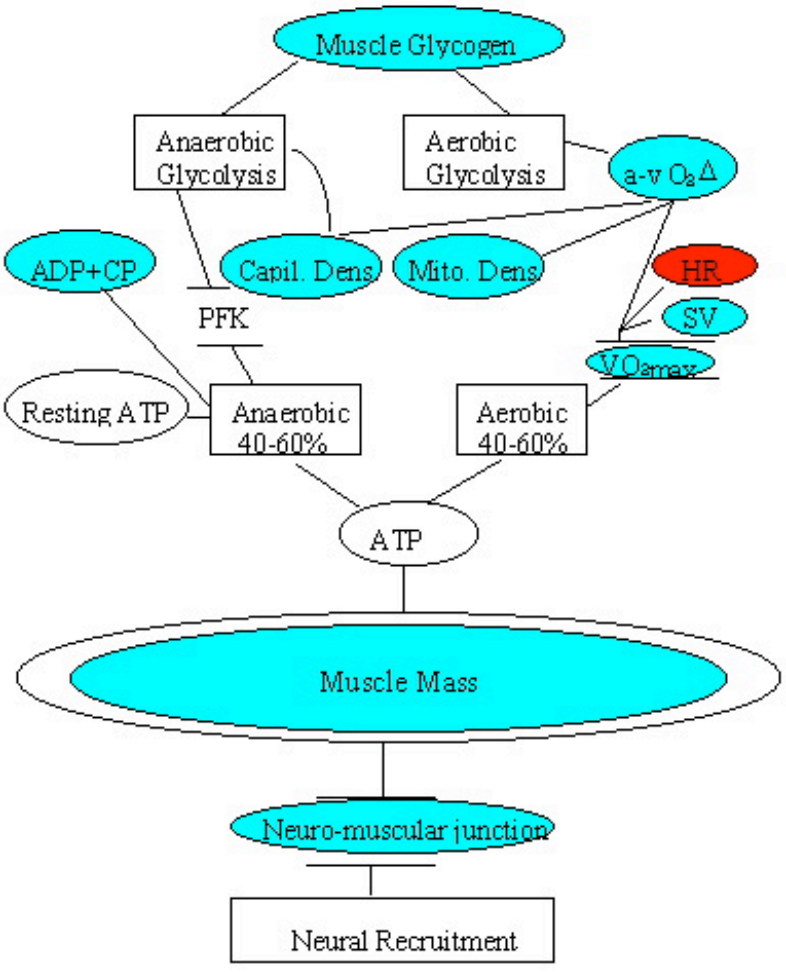
Although the joint effects of aging and exercise on motorneuron endplate structure has not been specifically investigated, we may be able to hypothesize on the effects based on other findings. Specifically, in the strength training studies cited, the increases in strength are greater than the increase in muscle cross sectional area, indicating an increase in specific tension. It is known that specific tension in muscle fibers in-vitro is maintained with aging (McCarter, 1978) but decreased in whole muscle in-vivo (Phillips, et al, 1992), probably due to changes in motorneuron endplate structure (Rosenheimer and Smith, 1985). Therefore, we can surmise that the apparent increase in specific tension with strength training is due, at least in some measure, to increased performance of the motorneuron. It should also be mentioned that improvements in expressed strength after training may be in part due to improved coordination and learning.

Summary

Many, but not all of the effects of sedentary aging can be altered with exercise training. Figure 3 presents the combined effects of aging and exercise on the mechanisms of maximal short term performance. The blue shaded portions represent those systems in which age related decreases in maximal performance or function has been shown to be minimized or eliminated with exercise. The red shaded portions represent those systems in which age related decreases in maximal function is not minimized by exercise.

Muscle mass can be at least partially maintained with strength or endurance training. Mitochondrial density and capillary density can be maintained with vigorous exercise training. Stroke volume can be increased with exercise training, but heart rate declines irrespective of activity level, resulting in an age related decrease in cardiac output and VO2 max. Muscle glycogen

content can be increased in older individuals, but the studies that have been conducted to date have used low intensity or short term training programs and have not increased muscle glycogen levels up to those of younger people. Exercise probably enhances the function of the neuromuscular junction, and allows better recruitment of skeletal muscle. Based on the findings presented, our model predicts that sedentary older adults should have dramatically lower short term maximal performance than younger adults. However, an older adult who is trained at the same volume and intensity as a younger adult should be capable of performances similar to his/her younger counterpart. The unalterable decrease in heart rate and consequential reduction in VO2 max may prevent the older individual from training at the same volume and intensity as a younger athlete, and consequently, performance of the older athlete will be limited by the reduction in absolute training volume and intensity. However, the trained older adult should almost certainly have performances superior to those of younger and older sedentary adults, and therefore may have a better ability to cope with the physiological demands of free living.



Sports Science Research References

1. Smith, R, and Spinks, W.L. Discriminant analysis of biomechanical differences between novice, good, and elite rowers. Journal of Sport Sciences. 13:377-385. 1995.

2. Steinacker, J.M, Both, M., and Whipp, B.J. Pulmonary Mechanics and Entrainment of Respiration and Stroke rate during rowing. International Journal of Sports Medicine. 14(suppl 1):S15-S19, 1993.

3. Smith, R, and Rutherford, O.M. Spine and total body bone mineral density and serum testoterone levels in male athletes. European J. of Applied Physiology. 67:330-334. 1993.

4. Pelliccia, A. Et al. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. New. England J. of Medicine. 324:295-301. 1991

5. Jensen, K. et al. High altitude training does not increase maximal oxygen uptake or work capacity at sea level in rowers. Scandinavian J. of Medicine and Science in Sports. 3: 256-262, 1993.

6. Roth, W. et al. Force-time charactersitics of the rowing stroke and corresponding physiological muscle adaptations. International J. Sports Med. 14 (suppl 1):S32-S34, 1993.

7. McNaughton, L. and Cedaro, R. The effect of sodium bicarbonate on rowing ergometer performance in elite rowers. The Australian J. of Science and Medicine in Sport. 23(3) p66-69. 1991

Bibliography for Short Term Power Article- Jim Martin
(Also a good general resource for research articles on aging and exercise)

Allen WK. Seals DR. Hurley BF. Ehsani AA. Hagberg JM. Lactate threshold and distance-running performance in young and older endurance athletes. Journal of Applied Physiology. 58(4):1281-4, 1985 Apr.

Ames BN. Shigenaga MK. Hagen TM. Mitochondrial decay in aging. [Review] Biochimica et Biophysica Acta. 1271(1):165-70, 1995.

Aniansson A and Gustafsson E. Physical training in elderly men with specific reference to quadriceps muscle strength and morphology. Clin. Physiol. 1: 87-98, 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 - Supplement. 9:92-102, 1983.

Brown AB. McCartney N. Sale DG. Department of Physical Education, McMaster University, Hamilton, Ontario, Canada. Positive adaptations to weight-lifting training in the elderly. Journal of Applied Physiology. 69(5):1725-33, 1990.

Bastien C. and Sanchez J. Phosphagens and glycogen content in skeletal muscle after treadmill training in young and older rats. Eur. J. of Appl. Physiol. 52(3): 291-5, 1984.

Coyle EF. Martin WH 3d. Sinacore DR. Joyner MJ. Hagberg JM. Holloszy JO Time course of loss of adaptations after stopping prolonged intense endurance training. Journal of Applied Physiology: Respiratory, Environmental & Exercise Physiology 57(6):1857-64, 1984.

Coggan AR. Spina RJ. King DS. Rogers MA. Brown M. Nemeth PM. Holloszy JO. Histochemical and enzymatic comparison of the gastrocnemius muscle of young and elderly men and women. Journal of Gerontology. 47(3):B71-6, 1992a.

Coggan AR. Spina RJ. King DS. Rogers MA. Brown M. Nemeth PM. Holloszy JO. Skeletal muscle adaptations to endurance training in 60- to 70-yr-old men and women. Journal of Applied Physiology. 72(5):1780-6, 1992b.

Coggan AR. Abduljalil AM. Swanson SC. Earle MS. Farris JW. Mendenhall LA. Robitaille PM. Muscle metabolism during exercise in young and older untrained and endurance-trained men. Journal of Applied Physiology. 75(5):2125-33, 1993.

Denis C. J. C. Chatard, D. Dormois, M.T. Linossier, A. Geyssant, and J. Lacour. Efeects of endurance training on capillary supply of human skeletal muscle of two age groups (20 and 60 years) J. Phisiol. Paris 81: 379-383, 1986.

Essen-Gustavsson, B. and O. Borges. Histochemical and metabolic characteristics of human skeletal muscle in relation to age. Acta Physiol. Scan. 95: 153-165, 1986.

Frontera WR. Meredith CN. O'Reilly KP. Knuttgen HG. Evans WJ. United States Strength conditioning in older men: skeletal muscle hypertrophy and improved function. Journal of Applied Physiology. 64(3):1038-44, 1988.

Gollnick, P. D. R. B. Armstrong, C.W. Saubert IV, K. Piehl, and B. Saltin. Enzyme activity and fiber composition in skeletal muscle of trained and untrained men. Journal of Applied Physiology. 34: 107-111, 1972.

Grimby, G., B. Danneskiold-Samsoe, K. Hvid, and B. Saltin. Morpology and enzymatic capacity in arm and leg muscle of 78-81 year old men and women. Acta Physiol. Scan. 115: 125-134, 1982.

Grimby, G. and B. Saltin. The aging muscle. Clin. Phsiol. 3: 209-218, 1983.

Grimby G. Aniansson A. Zetterberg C. Saltin B. Is there a change in relative muscle fibre composition with age? Clinical Physiology. 4(2):189-94, 1984.

Green, S. A definition and systems view of anaerobic capacity European Journal of Applied Physiolgy and Occupational Physiology 69:168-173, 1994.

Hagberg, J.M., W.K. Allen, D.R. Seals, B.H. Hurley, A.A. Eshani, and J.O. Holloszy. A hemodynamic comparison of young and older endurance athletes during exercise. J. Appl. Physiol. 58:2041-2046, 1985.

Jorfeldt, L., and J. Wahren. Leg blood flow during exercise in man. Clin. Sci. 41(5):459-73, 1971

Klitgaard H. Brunet A. Maton B. Lamaziere C. Lesty C. Monod H. Morphological and biochemical changes in old rat muscles: effect of increased use. Journal of Applied Physiology. 67(4):1409-17, 1989

Lakatta EG. Laboratory of Cardiovascular Science, Gerontology Research Center, National Institute on Aging, Baltimore, MD 21224. Cardiovascular reserve capacity in healthy older humans. [Review] Aging. 6(4):213-23, 1994.

Larsson L. Morphological and functional characteristics of the aging skeletal muscle in man. Acta. Physiol. Scand. Suppl. 457: 1-36, 1978.

Larsson L. Sjodin B., and Karlsson J. Histochemical and biochemical changes in human skeletal muscle with age in sedentary males, age 22--65 years. Acta Physiol Scand. 103(1):31-9, 1978

Larsson L. and J. Karlsson Isometric and dynamic endurance as a function of age and skeletal muscle characteristics. Acta Physiol Scand. 104(2):129-36, 1978.

Levy, W. C., M. D. Cerqueira, I. B. Abrass, R. S. Schwartz, and J. R. Stratton. Endurance exercise training augments diastolic filling at rest and during exercise in healthy young and older men. Circulation. 88: 116-26, 1993.

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 Nerve. 6(8):588-95, 1983.

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. J Neurol Sci. 72(2-3):211-22, 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. J Neurol Sci. 84(2-3):275-94, 1988

Makrides L. Heigenhauser GJ. Jones NL. High-intensity endurance training in 20- to 30- and 60- to 70-yr-old healthy men. Journal of Applied Physiology. 69(5):1792-8, 1990.

Makrides L. Heigenhauser GJ. McCartney N. Jones NL. Maximal short term exercise capacity in healthy subjects aged 15-70 years. Clinical Science. 69(2):197-205, 1985.

Meredith CN. Frontera WR. Fisher EC. Hughes VA. Herland JC. Edwards J. Evans WJ. Peripheral effects of endurance training in young and old subjects. Journal of Applied Physiology. 66(6):2844-9, 1989.

McCarter R. Effects of age on contraction of mamillian skeletal muscle. Aging. 6, 1-21, 1978.

McCarter R. and McGee J Influence of nutrition and aging on the composition and function of rat muscle. Journal of Gerontology. 42(4):432-41, 1987.

McCully, K.K., M.A. Forciea, L.M. Hack, D. Donlon, R.W. Wheatley, C.A. Oatis, T. Goldberg, and B. Chance. Muscle metabolism in older subjects using 31P magnetic resonance spectroscopy. Can. J. Physiol. Pharmacol. 69:576-580, 1991.

Medbo JI. Tabata I. Relative importance of aerobic and anaerobic energy release during, Journal of Applied Physiology. 67(5):1881-6, 1989.

Moller P. Bergstrom J. Furst P. Hellstrom K. Effect of aging on energy-rich phosphagens in human skeletal muscles. Clinical Science. 58(6):553-5, 1980

Moller P. Brandt R. The effect of physical training in elderly subjects with special reference to energy-rich phosphagens and myoglobin in leg skeletal muscle. Clinical Physiology. 2(4):307-14, 1982 Aug

Orlander J. Kiessling KH. Larsson L. Karlsson J. Aniansson A. Skeletal muscle metabolism and ultrastructure in relation to age in sedentary men. Acta Physiologica Scandinavica. 104(3):249-61, 1978.

Orlander J. Aniansson A. Effect of physical training on skeletal muscle metabolism and ultrastructure in 70 to 75-year-old men. Acta Physiologica Scandinavica. 109(2):149-54, 1980.

Ogawa, T., R.J. Spina, W.H. Martin, W.M. Kohrt, K.B. Schechtman, and J.O. Holloszy. Effects of aging, sex, and physical training on cardiovascular responses to exercise. Circulation 86:494-503, 1992.

Phillips SK. Bruce SA. Newton D. Woledge RC. Department of Physiology, University College London. The weakness of old age is not due to failure of muscle activation. Journal of Gerontology. 47(2):M45-9, 1992.

Pollock ML. Foster C. Knapp D. Rod JL. Schmidt DH. Effect of age and training on aerobic capacity and body composition of master athletes. J Appl Physiol. 62(2):725-31, 1987.

Parizkova J. Eiselt E. Sprynarova S. Wachtlova M. Body composition, aerobic capacity, and density of muscle capillaries in young and old men. Journal of Applied Physiology. 31(3):323-5, 1971.

Rodeheffer RJ. Gerstenblith G. Becker LC. Fleg JL. Weisfeldt ML. Lakatta EG. Exercise cardiac output is maintained with advancing age in healthy human subjects: cardiac dilatation and increased stroke volume compensate for a diminished heart rate. Circulation. 69(2):203-13, 1984.

Rosenheimer JL. Smith DO. Differential changes in the end-plate architecture of functionally diverse muscles during aging. Journal of Neurophysiology. 53(6):1567-81, 1985.

Sale DG. MacDougall JD. Upton AR. McComas AJ. Effect of strength training upon motor neuron excitability in man. Medicine & Science in Sports & Exercise. 15(1):57-62, 1983.

Shephard RJ, Physical activity and aging. Rockville MD., Aspen Publishers. 1987.

Smith DO. Rosenheimer JL. Factors governing speed of action potential conduction and neuromuscular transmission in aged rats. Experimental Neurology. 83(2):358-66, 1984.

Stolarek I. Scott PJ. Caird FI. Department of Geriatric Medicine, Stobhill General Hospital, Glasgow, Scotland. Physiological changes due to age. Implications for cardiovascular drug therapy. [Review] Drugs & Aging. 1(6):467-76, 1991.

Stratton JR. Levy WC. Cerqueira MD. Schwartz RS. Cardiovascular responses to exercise. Effects of aging and exercise training in healthy men. Circulation. 89(4):1648-55, 1994.

Wahren, J., B. Saltin, L. Jorfeldt, and B. Pernow. Influence of age on the local circulatory adaptation to leg exercise. Scand. J. Clin. Lab. Invest. 33:79-86, 1974.

Walters TJ. Sweeney HL. Farrar RP. Aging does not affect contractile properties of type IIb FDL muscle in Fischer 344 rats. American Journal of Physiology. 258(6 Pt 1):C1031-5, 1990.

Wollein W. Bachl N. Prokop L. Endurance capacity of trained older aged athletes. European Heart Journal. 5 Suppl E:21-5, 1984 Nov.