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Deceleration of Skeletal Muscle Atrophy and Disability in Aging Population: Effect of Exercise

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Abstract

Exercise is a effective measure for the prevention and management of different muscle injuries, diseases and atrophy in elderly. Endurance exercise increase skeletal muscle oxidative capacity, resistanc exercise cause hypertrophy of fast-twitch muscle fibers. Exercise improved muscle plasticity in the elderly, and makes skeletal muscle possible to modify age-associated decline in physical performance and enhancing life quality in the elderly. Resistance exercise increase the turnover rate of contractile proteins, and following adaptational changes first appeared in newly formed or regenerating fibers and these changes lead to the remodeling of contractile apparatus and increase in strength generating capacity of skeletal muscle. Dependance of turnover rate of contractile proteins from oxidative capacity of muscle show that turnover of contractile proteins provides a mechanism by which the effect of exercise causes changes in muscle metabolism in accordance with the needs of the myofibrillar apparatus. Both endurance and resistance exercise have a preventive role in the development of muscle atrophy, but a combination of both with different frequency, intensity and duration has been shown to be more effective among aging population. Aim of this paper is discuss about possibilities of using exercise in prevention of muscle atrophy in elderly and describe how exercise improve muscle plasticity, make skeletal muscle possible to modify age-associated decline in physical performance and enhancing life quality in aging population.

Keywords: Aging; Skeletal muscle; Atrophy; Preventive role ef exercise

Introduction

Aging is a multifactorial process associated with changes in muscle mass, strength, endurance and the inability to maintain balance [1,2]. Risk factors for falling lead to severe injury [3], changes in skeletal muscle quantity and quality lead to disability in the aging population [4]. The rate of muscle loss is 1% to 2% per year past the age of 50, as a result 25% underage of 70% and 40% over of 80% are sarcopenic [5,6]. In both young and aged skeletal muscle unloading and hormonal myopathies [7], including glucocorticoid myopathy have an important role in development of muscle atrophy when atrophic muscle becomes active [4,8,9], muscle mass increases, but the recovery of muscle strength takes much longer time [4,10,11]. For prevention or management of many different injures and diseases, a specific exercise tailored for rehabilitation needs, including assisted exercise are widely used in recent years. In this short review we discuss about possibilities of using exercise in prevention of muscle atrophy in elderly and describe how apropriate exercise improve muscle plasticity, make skeletal muscle possible to modify age-associated decline in physical performance and enhancing life quality in aging population.

Aging Muscle

The decline of muscle mass in elderly is caused by type II fiber atrophy and loss in the number of Fast-Twitch (FT) muscle fibers. Increased variability in fiber size, accumulation of non grouping, scattered and angulated fibers, atrophy [12,13]. Loss of fiber number, decreased production of anabolic hormones testosterone, growth hormone, Insulin-like Growth Factor-1 (IGF-1), and an increase in the release of catabolic agents are principal causes of sarcopenia. Interleukin-6 also amplify the rate of muscle wasting [14,15]. Aging skeletal muscle becomes less powerful, fat is redistributed from the depot to muscle [16], intensive collagen synthesis and post-translational changes in its structure reduce the elasticity of ligaments [17,18].

Effect of Exercise

Exercise is a effective measure for the prevention and management of different muscle injuries

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and diseases. For example, in case of glucocorticoid caused myopathy, both endurance and resistance exercise have a preventive role in the development of muscle atrophy, but a combination of both with different frequency, intensity and duration has been shown to be more effective among aging population [4,8,19]. Intensive short-lasting resistance exercise have an anticatabolic effect on the contractile apparatus and the ECM of skeletal muscle [4,20]. Glucocorticoids increased myofibrillar protein degradation in FT muscles, while fibril-and network-forming collagen specific mRNA levels decreased in FT and Slow-Twitch (ST) muscles [21]. Both the myofibrillar apparatus and the Extracellular Matrix (ECM) play a crucial role in changes of muscle strength during glucocorticoid administration and following muscle loading [22]. Decrease in endurance capacity in elderly is related with decrease in oxidative capacity of muscle. Endurance exercise are effective in increasing of skeletal muscle oxidative capacity, metabolim and plasticity [23,24].

Effect of Resistance Exercise

Muscle atrophy contributes to but does not completely explain the decrease in strength in the elderly. The age-related decrease in muscle mass and strength is a consequence of the complete loss of fibers associated with the decrease in the number of motor units and fiber atrophy [25]. Resistance exercise is strong stimulus for growth of adult skeletal muscle due to fiber hypertrophy and improving athletic performance, enhancing general health and fitness. Resistance exercise used in rehabilitation after surgery or an injury, or just for the pleasure of exercise [26]. Resistance exercise is an effective measure in the elderly, improving glucose intolerance, including improvements in insulin signaling defects, reduction in tumor necrosis factor-α, increases in adiponectin and IGF-1 concentrations, and reductions in total and abdominal visceral fat [27]. Resistance exercise improves skeletal muscle metabolism and through it muscle function in the elderly and their life quality [4]. Resistance exercise enhances the synthesis rate of myofibrillar proteins but not that of sarcoplasmic proteins and this is related to mammalian target of rapamycin by activating proteins within the nitrogen-activated protein kinase signaling [28,29]. A significant difference was observed between previously trained young and old participants in recovery from resistance exercise [30]. These results suggest a more rapid recovery in the young group. Recovery from damaging exercise is slower as a result of age, whereas there are no age related differences in recovery from less damaging metabolic fatigue [31]. Resistance exercise, during which the power of exercise increased less than 5% per session, caused hypertrophy of both FT and ST muscle fibers, an increase of myonuclear number *via* fusion of satellite cells with damaged fibers or formation of new muscle fibers as a result of myoblast fusion in order to maintain myonuclear domain size [10,32]. Resistance exercise increase the turnover rate of contractile proteins, and following adaptational changes first appeared in newly formed or regenerating fibers and these changes lead to the remodeling of contractile apparatus and increase in strength generating capacity of muscle [10].

Effect of Endurance Exercise

Endurance exercise increase skeletal muscle oxidative capacity, plasticity and enhancing life quality in the elderly. Improved muscle plasticity in the elderly makes skeletal muscle possible to modify the age-associated decline in physical performance [33]. Increased aerobic capacity in elderly people is related to an increase in the abilities of the cardiovascular system, and to the lesser extent to an increase in muscle mitochondrial concentration [34]. So, regular aerobic exercise provides a graund for an increase in muscle oxidative capacity in the elderly. Dependance of turnover rate of contractile proteins from oxidative capacity of muscle show that turnover of contractile proteins provides a mechanism by which the effect of exercise causes changes in muscle metabolism in accordance with the needs of the myofibrillar apparatus [35]. As the contractile proteins turnover decreases in the elderly, and endurance exercise stimulates an increase in the oxidative capacity of skeletal muscle by an increase in mitochondrial biogeneses and supports *via* faster protein turnover improvement of muscle function. Aging-associated reduction in AMP-Activated Protein Kinase (AMPK) activity is the main factor in reduced mitochondrial function [36]. Endurance exercise caused AMPK activation in aging FT muscles [37] and AMPK α1 isoform is related to the metabolic adaptation of skeletal muscle [38]. Myosin Heavy Chain (MyHC) and Myosin Light Chain (MyLC) isoforms play an important role in the process of modulation of contractile apparatus during endurance exercise [39], as well as C-protein binds myosin and actin and affects mechanical properties myosin cross-bridges by linking the S2 segment of myosin to the backbone of thick filament is sensitive to the volume of endurance exercise [40].

Effect of Concurrent Resistance and Endurance Exercise

Concurrent resistance and endurance exercise decrease the gain in muscle mass in comparison with resistance exercise for alone in top athletes [41]. Described effect explained by AMPK blocking the activation of mammalian Target of Rapamycin Complex-1 (TORC 1) by phosphorylating and activating the Tuberous Sclerosis Complex-2 (TSC 2) [42]. Concurrent resistance and endurance exercise in elderly men has shown that strength gain was similar to that observed with resistance exercise alone, although resistance exercise volume was half of that resistance exercise alone [43]. Using lower exercise volumes in concurrent exercise in older men in comparison with endurance and resistance exercise alone leads to similar strength enhancement with no presence of interference in this population [44,45]. In the elderly population, improvement in both strength and cardiorespiratory fitness is important and concurrent exercise is the best strategy to enhance cardiorespiratory fitness as it hasbeen shown in the literature [46]. It has shown that concurrent exercise improved performance in all occupational tasks and did not interfere with improvements in strength, power and endurance measures compared to endurance and resistance exercise alone in recreational athletes [47]. Theoretical complications in the full understanding of concurrent exercise effect on the skeletal muscle are :

1. It is not clear whether muscle fibers are capable to undergo hypertrophy and maintain endurance capacity at the same time.

2. Why the effect ofconcurrent exercise is different in top and recreational athletes.

Conclusions

Aging is associated with changes in muscle mass, strength, endurance and the inability to maintain balance. Risk factors for falling lead to severe injury, changes in skeletal muscle quantity and quality lead to disability in the aging population. Decrease in endurance capacity in elderly is related with decrease in oxidative capacity of muscle. Endurance exercise are effective in increasing of skeletal muscle oxidative capacity and metabolim. Resistance exercise is strong stimulus for growth of adult skeletal muscle due to fiber hypertrophy and improving athletic performance, enhancing general health and fitness. Resistance exercise is an effective measure in the elderly, improving glucose intolerance, including improvements in insulin signaling defects, reduction in tumor necrosis factor-α, increases in adiponectin and IGF-1 concentrations, and effective in reduction of total and visceral fat. Concurrent resistance and endurance exercise improve performance in all occupational tasks and did not interfere with improvements in strength, power and endurance measures compared to endurance and resistance exercise alone in aging population.

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References

1. Haus JM, Carrithers JA, Trappe SW, Trappe TA. Collagen, cross-linking, and advanced glycation end products in aging human skeletal muscle. J Appl Physiol. 2007;103(6):2068-76.
2. Trappe T. Influence of aging and long-term unloading on the structure and function of human skeletal muscle. Appl Physiol Nutr Metabol. 2009;34(3):459-64.
3. Santos CA, Dantas EE, Moreira MH. Correlation of physical aptitude; functional capacity, corporal balance and quality of life (QoL) among elderly women submitted to a post-menopausal physical activities program. Arch Gerontol Geriatr. 2011;53(3):344-9.
4. Seene T, Kaasik P, Riso EM. Review on aging, unloading and reloading: Changes in skeletal muscle quantity and quality. Arch Gerontol Geriatr. 2012;54(2):374-80.
5. Marzetti E, Leeuwenburgh C. Skeletal muscle apoptosis, sarcopenia and frailty at old age. Exper Gerontol. 2006;41(12):1234-8.
6. Hiona A, Leeuwenburgh C. The role of mitochondrial DNA mutations in aging and sarcopenia: Implications for the mitochondrial vicious cycle theory of aging. Exper Gerontol. 2008;43(1):24-33.
7. Siu PM, Pistilli EE, Alway SE. Age-dependent increase in oxidative stress in gastrocnemius muscle with unloading. J Appl Physiol. 1985;105(6):1695-705.
8. Seene T, Kaasik. Role of myofibrillar protein catabolism in development of glucocorticoid myopathy: Aging and functional activity aspects. Metabolites. 2016;6(2).
9. Powers SK, Kavazis AN, McClung JM. Oxidative stress and disuse muscle atrophy. J Appl Physiol. 2007;102(6):2389-97.
10. Seene T, Pehme A, Alev K, Kaasik P, Umnova M, Aru M. Effects of resistance training on fast- and slow-twitch muscles in rats. Biol Sport. 2010;27(3):221-9.
11. Pottle D, Gosselin LE. Impact of mechanical load on functional recovery after muscle reloading. Med Sci Sports Exerc. 2000;32(12):2012-7.
12. Kim JH, Kwak HB, Leeuwenburgh C, Lawler JM. Lifelong exercise and mild (8%) caloric restriction attenuate age-induced alterations in plantaris muscle morphology, oxidative stress and IGF-1 in the fischer-344 rat. Exper Gerontol. 2008;43(4):317-29.
13. Buford TW, Anton SD, Judge AR, Marzetti E, Wohlgemuth SE, Carter CS, et al. Models of accelerated sarcopenia: Critical pieces for solving the puzzle of age-related muscle atrophy. Ageing Res Rev. 2010;9(4):369-83.
14. Roubenoff R. Catabolism of aging: Is it an inflammatory process? Curr Opin Clin Nutr Metab Care. 2003;6(3):295-9.
15. Goldspink G, Harridge SD. Growth factors and muscle ageing. Exper Gerontol. 2004; 39(10):1433-8.
16. Toth MJ, Tchernof A. Lipid metabolism in the elderly. Europ J Clin Nutr. 2000;54:121-5.
17. Kjær M, Magnusson MP, Krogsgaard M, Boysen Møller J, Olesen J, Heinemeier K, et al. Extracellular matrix adaptation of tendon and skeletal muscle to exercise. J Anat. 2006;208(4):445-50.
18. Heinemeier KM, Olesen JL, Haddad F, Schjerling P, Baldwin KM, Kjaer M. Effect of unloading followed by reloading on expression of collagen and related growth factors in rat tendon and muscle. J Appl Physiol. 2009;106:178-86.
19. Seene T, Kaasik P. Age-associated changes in skeltal muscle regeneration: Effect of exercise. Adv Aging Res. 2015;4:230-241.
20. Riso EM, Ahtikoski AM, Umnova M. Partial prevention of muscle atrophy in excessive level of glucocorticoids by exercise: Effect on contractile proteins and extracellular matrix. Baltic J Laborat Animal Sci. 2003;13:5-12.
21. Riso EM, Ahtikoski A, Alev K, Kaasik P, Pehme A, Seene T. Relationship between extracellular matrix, contractile apparatus, muscle mass and strength in case of glucocorticoidmyopathy. J Steroid Biochem Mol Biol. 2008;108(1-2):117-20.
22. Riso EM, Ahtikoski AM, Takala TES, Seene T. The effect of unloading and reloading on the extracellular matrix in skeletal muscle: Changes in muscle strength and motor activity. Biol Sport. 2010;27(2):89-94.
23. Seene T, Kaasik P. Role of exercise therapy in prevention of decline in aging muscle function: Glucocorticoid myopathy and unloading. J Aging Res. 2012.
24. Seene T, Kaasik P, Seppet E. Changes in myofibrillar and mitochondrial compartments during increased activity: Dependance from oxidative capacity of muscle. Health. 2017;9:779-98.
25. Rader EP, Faulkner JA. Recovery from contractioninduced injury is impaired in weight-bearing muscles of old male mice. J Appl Physiol. 2006;100(2):656-61.
26. Fry AC. The role of resistance exercise intensity on muscle fibre adaptations. Sports Med. 2004;34:(10):663-79.
27. Flack KD, Davy KP, Hulver MW, Winett RA, Frisard MI, Davy BM. Aging, resistance training, and diabetes prevention. J Aging Res. 2011.
28. Moore DR, Tang JE, Burd NA, Rerecich T, Tarnopolsky MA, Phillips SM. Differential stimulation of myofibrillar and sarcoplasmic protein synthesis with protein ingestion at rest and after resistance exercise. J Physiol. 2009;587:897-904.
29. Moore DR, Atherton PJ, Rennie MJ, Tarnopolsky MA, Phillips SM. Resistance exercise enhances mTOR and MAPK signalling in human muscle over that seen at rest after bolus protein ingestion. Acta Physiol. 2011;201(3):365-72.
30. McLester JR, Bishop PA, Smith J, Wyers L, Dale B, Kozusko J, et al. A series of studies-a practical protocol for testing muscular endurance recovery. J Strength Cond Res. 2003;17(2):259-73.
31. Fell J, Williams AD. The effect of aging on skeletalmuscle recovery from exercise: Possible implications for aging ahletes. J Aging Physical Act. 2008;16(1):97-115.
32. Itai Y, Kariya Y,Hoshino Y. Morphological changes in rat hindlimb muscle fibres during recovery from disuse atrophy. Acta Physiol Scand. 2004;181(2):217-24.
33. Suominen H. Ageing and maximal physical performance. Europ Rev Aging Physical Act. 2011;8(1):37-42.
34. Sagiv M, Goldhammer E, Ben-Sira D, Amir R. Factors defining oxygen uptake at peak exercise in aged people. Europ Rev Aging and Physical Act. 2010;7(1):1-2.
35. Seene T, Kaasuk P, Seppet E. Crosstalk between mitochondria and

myofibrils in adult and aging striated muscle tissue: Effect of increased functional activity. Asian J Res Med Pharm Sci. 2017;1(3):1-13.

36. Reznick RM, Zong H, Li J, Morino K, Moore IK, Yu HJ, et al. Aging-associated reductions in AMP-activated protein kinase activity and mitochondrial biogenesis. Cell Metab. 2007;5(2):151-6.

37. Thomson DM, Brown JD, Fillmore N, Ellsworth SK, Jacobs DL, Winder WW, et al. AMP activated protein kinase response to contractions and treatment with the AMPK activator AICAR in young adult and old skeletal muscle. J Physiol. 2009;587:2077-86.

38. Mcgee SL, Mustard KJ, Hardie DG, Baar K. Normal hypertrophy accompanied by phosphoryation and activation of AMP-activated protein kinase alpha1 following overload in LKB1 knockout mice. J Physiol. 2008;586(6):1731-41.

39. Alev K, Kaasik P, Pehme A, Aru M. Physiological role of myosin light and heavy chain isofornsin fast- twitch and slow-twitch muscles: Effect of exercise. Biol Sport. 26(3):215-34.

40. Seene T, Alev K, Kaasik P, Pehme A, Parring AM. Endurance training: Volume dependent adaptational changes in myosin . Int J Sports Med. 2005;26(10):815-21.

41. Hickson RC. Interference of strength development by simultaneously training for strength and endurance. Europ J Appl Physiol Occup Physiol. 1980;45(2-3):255-63.

42. Inoki K, Li Y, Zhu T, Wu J, Guan KL. TSC2 is phosphorylated and inhibited by Akt and suppresses mTOR signalling. Nat Cell Biol. 2002;4(9):648-57.

43. WoodRH, Reyes R, Welsch MA, Favaloro-Sabatier J, Sabatier M, Matthew Lee C, et al. Concurrent cardiovascular and resistance training in healthy older adults. Med Sci Sports Exerc. 2001;33(10):1751-8.

44. Izquierdo M, Ibanez J, Hakkinen K, Kraemer WJ, Larrión JL, Gorostiaga EM. Once weekly combined resistance and cardiovascular training in healthy older men. Med Sci Sports Exerc. 2004;36(3):435-43.

45. Karavirta L, Tulppo MP, Laaksonen DE, Nyman K, Laukkanen RT, Kinnunen H, et al. Heart ratedynamics after combined endurance and strength training in older men. Med Sci Sports Exerc. 2009;41(7):1436-43.

46. Cadore EL, Pinto RS, Lhullier FL, Correa CS, Alberton CL, Pinto SS, et al. Physiological effects of concurrent training in elderly men. Int J Sports Med. 2010;31(10):689-97.

47. Hendrickson N, Sharp M, Alemany J, Walker L, Harman E, Spiering B, et al. Combined resistance and endurance training improves physical capacity and performance on tactical occupational tasks. Europ J Appl Physiol. 2010;109(6):1197-208.