Scientific Research Publishing

Health, 2019, 11, 195-200
http://www.scirp.org/journal/health
ISSN Online: 1949-5005
ISSN Print: 1949-4998

Adaptation of Skeletal Muscle to Prolonged Activity: Role of Myosin

Teet Seene*, Karin Alev, Priit Kaasik

Institute of Sport Sciences and Physiotherapy, University of Tartu, Tartu, Estonia
Email: *teet.seene@ut.ee, karin.alev@ut.ee, priit.kaasik@ut.ee

How to cite this paper: Seene, T., Alev, K. and Kaasik, P. (2019) Adaptation of Skeletal Muscle to Prolonged Activity: Role of Myosin. *Health*, **11**, 195-200.
https://doi.org/10.4236/health.2019.112017

Received: November 8, 2018
Accepted: February 16, 2019
Published: February 19, 2019

Abstract

The aim of this short review is to describe the role of myosin isoforms during the adaptation of skeletal muscle to prolonged physical activity (for example endurance exercise) and to show the coordination between changes in muscle oxidative capacity and myofibrillar apparatus in slow-twitch and fast-twitch muscles. Adaptational changes in myosin isoforms during long lasting muscle activity (decrease of MyHC IIb isoforms relative content and increase of that MyHC IIa and decrease of MyLC 1 fast isoforms in fast-twitch muscles) are in good coordination with changes of muscle oxidative capacity. These changes show that during regular endurance exercise fast-twitch muscle fibers (type IIA) are also recruited and create the potential source of increase in endurance capacity during the process of adaptation to the prolonged physical activity.

Keywords

Prolonged Muscle Activity, Endurance Exercise, Myosin Light Chain, Myosin Heavy Chain, Endurance Capacity

1. Introduction

Prolonged physical activity like endurance exercise influences the enzymes of the metabolic cycle, electron transport chain, capillary supply, changes in key metabolic enzymes involved in fatty acid activation, and increases oxygen uptake in skeletal muscle [1] [2] [3] [4]. Endurance exercise training (ET) does not cause hypertrophy of muscle, as the level of force production is relatively small compared to their maximal force generation and long lasting aerobic exercise promotes a transition from type II muscle fibers to type I fibers [5]. Endurance type of prolonged muscle activity improves capillary blood supply, increases mitochondrial biogenesis and muscle oxidative capacity, causes faster turnover rate

of sarcoplasmic proteins and qualitative remodelling of type I and IIA muscle fibers [6] [7]. Prolonged muscle activity induces significant destructive changes, most of all in myofibrils of type I and type IIA muscle fibers, including damage of myosin and actin filaments and the disturbance of the regularity of the Z-line in sarcomeres [7]. The destruction of myofibrils and mitochondria is characteristic of both fiber types [8]. This process is related to the myofibrillar apparatus, as myosin is the regulator in the conversion of chemical energy into mechanical activity. Among multiple isoforms of muscle proteins, myosin heavy chain (MyHC) and myosin light chain (MyLC) isoforms play an essential role in regulation of muscle main function [6] [7] [8]. Differences in MyHC (**Figure 1**) and MyLC (**Figure 2**) isoforms pattern are existing between fast and slow muscles. Changes in myosin isoforms composition during adaptation to endurance exercise are in accordance with structural, metabolic and conformational changes in muscle fibers [7] [9] [10]. The aim of this short review is to describe the role of myosin isoforms in the adaptational process of skeletal muscle to prolonged muscle activity (endurance exercise) and show coordination between changes in muscle oxidative capacity and myofibrillar apparatus in slow-twitch and fast-twitch muscles.

2. Myosin Isoforms in Adaptation to Prolonged Activity

During adaptation to endurance exercise, a decrease of MyHC IIb isoform in fast-twitch (FT) skeletal muscle fibers shows the transformation of muscle contractile apparatus in accordance with the increase in muscle oxidative capacity (**Figure 3**) and does not necessarily show the decrease of muscle contraction speed. For better understanding the role of MyLC in endurance exercise, it is important to study changes in MyLC in parallel with the quantification of MyHC under the same conditions [11] [12]. During muscle atrophy MyLC 1fast and 2fast isoforms increase in parallel with the increase in the relative content of MyHC IIb isoforms [9] [13]. A decrease in the relative content of MyLC 1fast isoform is an indicator of the slowing of muscle contraction [14]. This standpoint has been supported in rat skeletal muscle by decreasing MyLC 1fast isoform in the following direction: extensor digitorum longus (EDL) → plantaris

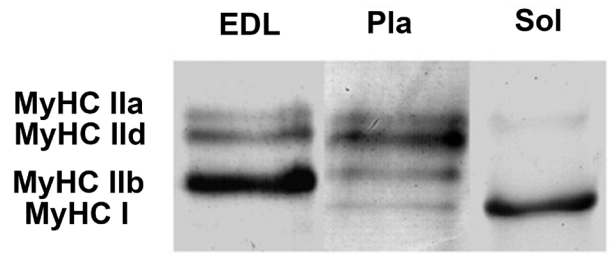

Figure 1. Differences in Myosin Heavy Chain isoforms relative content between fast- and slow-twitch muscles. MyHC—myosin heavy chain; EDL—extensor digitorum longus muscle; Pla—plantaris muscle; Sol—soleus muscle. EDL and Pla—fast-twitch muscle; Sol—slow-twitch muscle.

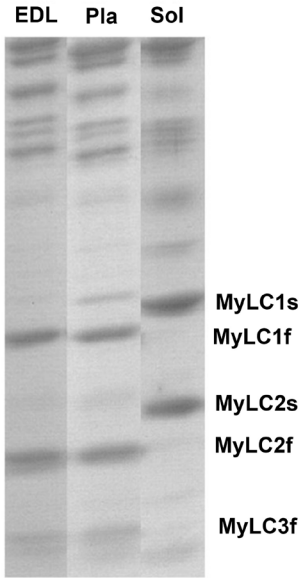

Figure 2. Differences in Myosin Light Chain isoforms relative content between fast- and slow-twitch muscles. MyLC—myosin light chain; EDL—extensor digitorum longus muscle; Pla—plantaris muscle; Sol—soleus muscle. EDL and Pla—fast-twitch muscle; Sol—slow-twitch muscle.

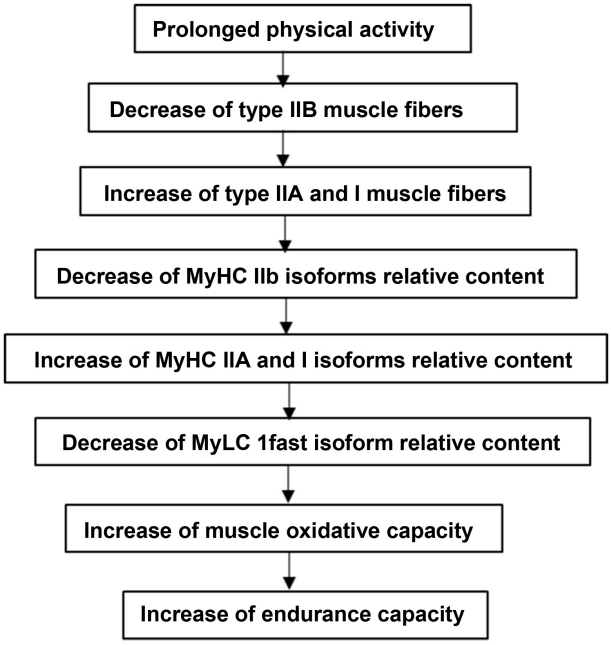

Figure 3. Changes in myosin isoforms in skeletal muscle during adaptation to prolonged physical activity. MyHC—myosin heavy chain; MyLC—myosin light chain.

(Pla) → diaphragm (Dia) → soleus (Sol) muscle [9]. It has been demonstrated that a positive correlation exists between MyHC IIb isoforms, MyLC 3fast isoform relative content and muscle contraction speed [6]. A positive correlation in

some studies was also found between MyLC 3fast/MyLC 1fast isoforms ratio and contraction speed [6] [15]. High affinity exsists between the MyLC 3fast isoform and MyHC IIb and IId isoforms [12] [16]. The distribution of MyLC isoforms in different muscles is following: in ST muscle the slow isoforms are dominating over fast isoforms [17], and this shows the physiological role and adaptational capacity of MyLC isoforms in skeletal muscle during adaptation to regular muscular activity [9]. MyLC 3fast isoform increases in rat Pla muscle as well as in EDL muscle during endurance exercise. The MyLC alkali isoforms content as well as MyLC regulatory isoforms content does not change in FT muscles during endurance training [18]. The decrease of slow isoforms, both in alkali and in regulatory MyLC, during endurance training and the increase of MyLC 3fast isoform in FT muscle are not in agreement with changes in MyHC isoforms pattern. Therefore, the stoichometry of these subunits and their association with each other do not change [9] [18] and this shows that there are no differences between MyHC and MyLC isoforms in FT muscles during adaptation to endurance exercise [9] [11]. Mitochondria in type IIB fibers are located in small groups near nuclei and between myofibrils on the level of Z-line but not in each sarcomere [7]. The effectiveness of metabolic signalling strongly depends on structural-functional relationships of the interaction between mitochondria and sarcomeres [18] [19] Hypoxia disturbs connections between mitochondria and sarcomeres as sarcomeric components disintegrate the muscle cell structure, cause cell injury and death [19]. The apoptosis is responsible for the initiation of protein degradation and loss of muscle nuclei associated with local atrophy, disruption of desmin impairs the linking of mitochondria to Z-disc and skeletal muscle exhibits impaired oxidative phosphorylation [19].

The adaptational process shows the existence of coordination between changes in oxidative capacity and contractile machinery in skeletal muscle (**Figure 3**), mainly in relation to muscle metabolism [19] [20]. Adaptation to endurance training shows that there are high potential to increase endurance capacity by recruiting of FT muscles [18]. Changes of myosin isoforms pattern during endurance exercise confirm that effective recruitment of these FT muscle fibers which are resistant to fatigue (type IIA), is the potential source of increase of endurance capacity.

3. Conclusion

Low intensity prolonged physical activity does not cause hypertrophy of skeletal muscle, but promotes a transition from type II muscle fibers to type I fibers. This process is related to the myofibrillar apparatus where myosin isoforms (MyHC and MyLC) play an essential role in regulation of muscle main function. Changes in myosin isoforms composition are in accordance with structural, metabolic and conformational changes in muscle fibers. Adaptation to prolonged physical activity shows that there is high potential to increase endurance capacity by recruiting of fast-twitch muscle fibers. Changes of myosin isoforms pattern during prolonged activity confirm that effective recruitment of fast-twitch

muscle fibers which are resistant to fatigue (type IIA), is the potential source of increase in endurance capacity.

Acknowledgements

This study was supported by the Estonian Research Council, research project number IUT20-58.

Author Contributions

Seene, Kaasik and Alev designed, participated in interpretation of data and final approval of the manuscript.

Conflicts of Interest

The authors declare no conflict of interest.

References

[1] Holloszy, J.O. and Booth, F.W. (1976) Biochemical Adaptations to Endurance Exercise in Muscle. *Annual Review of Physiology*, **38**, 273-291. https://doi.org/10.1146/annurev.ph.38.030176.001421

[2] Coffey, V.G. and Hawley, J.A. (2007) The Molecular Bases of Training Adaptation. *Sports Medicine*, **37**, 737-763. https://doi.org/10.2165/00007256-200737090-00001

[3] Hood, D.A. (2009) Mechanisms of Exercise-Induced Mitochondrial Biogenesis in Skeletal Muscle. *Applied Physiology, Nutrition, and Metabolism*, **34**, 465-472. https://doi.org/10.1139/H09-045

[4] Thomson, D.M. and Winder, W.W. (2009) AMPK Control of Fat Metabolism in Skeletal Muscle. *Acta Physiologica*, **196**, 147-154. https://doi.org/10.1111/j.1748-1716.2009.01973.x

[5] Thayer, R., Collins, J., Noble, E.G. and Taylor, A.W. (2000) A Decade of Aerobic Endurance Training: Histological Evidence for Fibre Type Transformation. *The Journal of Sports Medicine and Physical Fitness*, **40**, 284-289.

[6] Bottinelli, R. (2001) Functional Heterogeneity of Mammalian Single Muscle Fibres: Do Myosin Isoforms Tell the Whole Story? *Pflügers Archiv*, **443**, 6-17. https://doi.org/10.1007/s004240100700

[7] Seene, T., Kaasik, P. and Umnova, M. (2009) Structural Rearrangements in Contractile Apparatus and Resulting Skeletal Muscle Remodelling: Effect of Exercise Training. *Journal of Sports Medicine and Physical Fitness*, **49**, 410-423.

[8] Seene, T., Kaasik, P. and Seppet, E. (2017) Changes in Myofibrillar and Mitochondrial Compartments during Increased Activity: Dependance from Oxidative Capacity of Muscle. *Health*, **9**, 779-798. https://doi.org/10.4236/health.2017.95056

[9] Alev, K., Kaasik, P., Pehme, A., Aru, M., Parring, A.-M., Elart, A. and Seene, T. (2009) Physiological Role of Myosin Light and Heavy Chain Isoforms in Fast- and Slow-Twitch Muscles: Effect of Exercise. *Biology of Sport*, **26**, 215-234. https://doi.org/10.5604/20831862.894654

[10] Seene, T. and Kaasik, P. (2017) Skeletal Muscle Adaptation to Endurance Exercise: Fibre Type Peculiarities. *Austin Sports Medicine*, **2**, 1-7.

[11] Seene, T. and Alev, K. (1991) Effect of Muscular Activity on the Turnover Rate of

Actin and Myosin Heavy and Light Chains in Different Types of Skeletal Muscle. *International Journal of Sports Medicine*, **12**, 204-207. https://doi.org/10.1055/s-2007-1024668

[12] Wada, M. and Pette, D. (1993) Relationships between Alkali Light-Chain Complement and Myosin Heavy-Chain Isoforms in Single Fast-Twitch Fibres of Rat and Rabbit. *European Journal of Biochemistry*, **214**, 157-161. https://doi.org/10.1111/j.1432-1033.1993.tb17908.x

[13] Seene, T., Alev, K., Kaasik, P., Pehme, A. and Parring, A.M. (2005) Endurance Training: Volume Dependent Adaptational Changes in Myosin. *International Journal of Sports Medicine*, **26**, 815-821. https://doi.org/10.1055/s-2005-837449

[14] Hayashibara, T. and Miyanishi, T. (1994) Binding of the Amino-Terminal Region of Myosin Alkali Light Chain to Actin and Its Effect on Actin-Myosin Interaction. *Biochemistry*, **33**, 12821-12827. https://doi.org/10.1021/bi00209a013

[15] Sweeney, H.L., Kushmeric, M.J., Mahuchi, K., Sreter, F.A. and Gerery, J. (1988) Myosin Alkali Light Chain and Heavy Chain Variations Correlate with Altered Shortening Velocity of Isolated Skeletal Muscle Fibres. *Journal of Biological Chemistry*, **263**, 9034-9039.

[16] Stevens, L., Firinga, C., Gohlsch, B., Bastide, B., Mounier, Y. and Pette, D. (2000) Effects of Unweighting and Clenbuterol on Myosin Light and Heavy Chains in Ast and Slow Muscles of Rat. *American Journal of Physiology-Cell Physiology*, **279**, C1558-C1563. https://doi.org/10.1152/ajpcell.2000.279.5.C1558

[17] Bottinelli, R., Betto, R., Schiaffino, S. and Reggiani, C. (1994) Unloaded Shortening Velocity and Myosin Heavy Chain and Alkali Light Chain Isoform Composition in Rat Skeletal Muscle Fibres. *The Journal of Physiology*, **478**, 341-349. https://doi.org/10.1113/jphysiol.1994.sp020254

[18] Seene, T., Alev, K., Kaasik, P. and Pehme, A. (2007) Changes in Fast-Twitch Muscle Oxidative Capacity and Myosin Isoforms Modulation during Endurance Training. *Journal of Sports Medicine and Physical Fitness*, **47**, 124-132.

[19] Seene, T., Kaasik, P. and Seppet, E. (2017) Crosstalk between Mitochondria and Myofibrils in Adult and Aging Striated Muscle Tissue: Effect of Increased Functional Activity. *Asian Journal of Research in Medical and Pharmaceutical Sciences*, **1**, 1-11. https://doi.org/10.9734/AJRIMPS/2017/33584

[20] Seene, T. and Kaasik, P. (2018) Physical Activity in Prevention of Glucocorticoid Myopathy. In: Ahmad, S.I., Ed., *Aging Exploring a Complex Phenomenon*, CRC Press, Taylor & Frances Group, Boca Raton, 543-557.