Adaptation of Skeletal Muscle to Prolonged Activity: Role of Myosin

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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](image_url). 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 exists 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.

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**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**


Actin and Myosin Heavy and Light Chains in Different Types of Skeletal Muscle. International Journal of Sports Medicine, 12, 204-207. 


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