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Skeletal Muscle Adaptation to Endurance Exercise: Fibre Type Peculiarities

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Abstract

High-volume endurance exercise programmes improve the energetic potential of skeletal muscle and result in the effective functioning of the contractile apparatus of muscle for longer periods of time. Endurance exercise increases skeletal muscle's oxidative capacity, stimulating mitochondrial biogenesis and improves the functional parameters of muscles. The muscle fibres where myosin heavy chain I and IIA isoforms are dominant have a relatively high oxidative capacity and are recruited during endurance exercise. In the recent decade the phenomenon of muscle fibres with higher oxidative capacity remaining relatively small compared to muscle fibres with low oxidative capacity, and the issue of the role hybrid fibres play in endurance athletes' physical work capacity have attracted the attention of exercise physiologists. Analysis of structural rearrangements in the nerve-muscle synapses shows that destructive changes in intrafusal fibres are smaller than in extrafusal fibres. The comparison of changes in the ultrastructure of different types of extrafusal and intrafusal muscle fibres, and their innervation and regeneration potential during endurance exercise show that the described changes are in mutual relationship and depend on the type of exercise. The aim of this review is to treat the adaptation of skeletal muscle to endurance exercise; the main attention has been paid to the role of different types of muscle fibres (both extrafusal and intrafusal), their oxidative capacity, relation to renewal of myofibrillar proteins, rearrangements of contractile proteins and the resulting skeletal muscle remodeling.

Keywords: Skeletal muscle; Fibre types; Contractile Proteins; Structural Rearrangements; Endurance Exercise

Abbreviations

AMPK: Adenosine Monophosphate-Activated Protein Kinase; ATP: Adenosine Triphosphate; CSA: Cross-Sectional Area; FT: Fast-Twitch; IGF: Insulin-Like Growth Factor; MAFbx and MuRF: Ubicuitin Ligases; MyHC: Myosin Heavy Chain; MyLC: Myosin Light Chain; ST: Slow-Twitch; VO_2 max-maximal oxygen consumption.

Introduction

Adaptation of skeletal muscle to endurance exercise depends on oxidative capacity of muscle fibres, their capability of structural rearrangements, turnover rate of contractile proteins and fibres recovery from exercise-induced injury [1-6]. Endurance exercise caused changes occure in enzymes of the Krebs cycle, electron transport chain, capillary supply, in enzymes participated in fatty acid activation, and oxygen consumption [1,7,8]. Exercise in the aerobic zone of metabolism results a transition from type II to type I. This process occurs at the expense of type II fibre population [9]. The reason of that is hidden in to myofibrillar compartment of muscle fibres. Myosin, the main contractile protein, is the regulator in the conversion of chemical energy into mechanical one. The relationship exists between myosin isoforms and functional properties of the skeletal muscle. Maximal shortening velocity is higher in muscle fibres where myosin heavy chain (MyHC) fast isoforms dominate as the rate of actomyosin interaction is greater [10]. MyHC slow (I) isoform propelled actin filaments at a lower speed than fast (IIB) isoforms [11]. Endurance exercise does not cause hypertrophy of skeletal muscle fibres recruited in the exercise response as the level of force production is relatively small compared to their maximal force-generation [12]. The role of oxidative capacity of muscle fibres in the development of endurance capacity of athletes in relation with the turnover rate of contractile proteins is not clear and need further attention. The purpose of this review is to provide an overview of literature relating to the adaptation of skeletal muscle to the endurance exercise. Main attention has paid to the role of different types of muscle fibres, their oxidative capacity and its relation to myofibrillar proteins renewal. This article highlights the effects of endurance exercise on the rearrangements of contractile proteins and resulting skeletal muscle remodeling in dependence of oxidative capacity.

Adaptation of Myofibres to the Volume of Exercise

High volume (long duration) endurance exercise programs improve the energetic potential of skeletal muscle and result in the effective functioning of the muscle contractile apparatus for longer periods of time [13]. All these changes are mainly related with structural changes in type I and IIA muscle fibres (Figure 1) like the lesions in myosin and actin filaments, the distributed regularity of Zdisc in sarcomeres, swelling and destruction of mitochondria,

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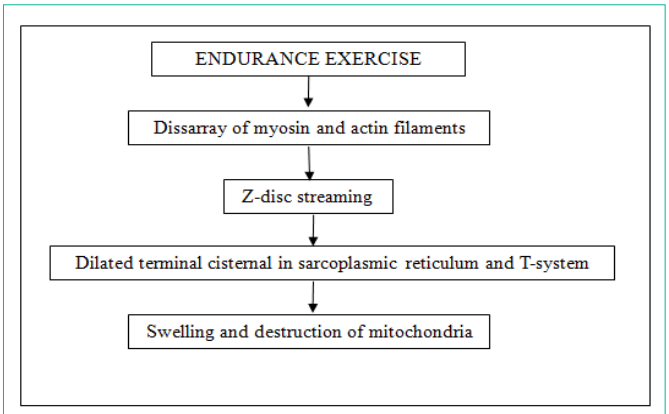

Figure 1: Destruction of muscle structures.

and dilation of terminal cisternae of sarcoplasmic reticulum and T-system tubules [3]. Endurance exercise caused structural changes are considerably less evident in fibres, which have low oxidative capacity than in fibers with relatively high oxidative capacity. Endurance exercise also induce the destruction in the myofibrillar compartment of the nuclear-bag intrafusal muscle fibers in the region of type I fibers. The increase in the number of satellite cells under the basal lamina of muscle fibers during high volume endurance exercise is a source of renewal of damaged structures [5]. Adaptation of skeletal muscle to high volume endurance exercise cause changes in muscle glycogen level [14] and protein synthesis [15] within several hours after exercise. This post exercise response is more important to the state of protein metabolism that occurs during exercise [16]. The volume of an endurance exercise determines the time period of depressed protein synthesis rate and the rate of degradation of muscle protein during the recovery period after endurance exercise [17,18]. Taking into account that protein synthesis is an energy consuming process and related to implication recovery period. Low cellular energy level induces in response to sustained contractile process activation of the 5′adenosine Monophosphate Activated Protein Kinase (AMPK). AMPK reduces translational processes and a low energy status is associated with a high rate of protein turnover, which limits the increase of fibre size [19]. Lack of recovery after high volume endurance exercise also leads to changes in the skeletal muscle myofibrillar compartment, particularly the destruction of contractile proteins and decreased exercise performance [20]. Changes in myosin isoform composition during high volume endurance exercise may be qualified as qualitative remodeling of skeletal muscle by replacing isoforms, which better suits the energetic adaptation to prolonged force-generation activity [12].

Adaptation of Neuromuscular Junctions to Exercise

The neuromuscular system controls physic motor impulses, neurotrophic influences, and plastic activity of skeletal muscle. Endurance exercise affects the structure of neuromuscular junctions depending on age, muscle twitch characteristics and volume and intensity of exercise [3]. In the beginning of adaptation to endurance exercise, a lot of neuromuscular terminals are branching [21]. Endurance exercise causes heterogeneity of the neuromuscular synapses, which is clearly expressed in type IIA muscle fibres. The synapses of type IIA muscle fibers have a large postsynaptic area and fast turnover of the muscle contractile and regulatory proteins. The axon terminals of type I muscle fibres are relatively small, round or oval shaped and closely located. The surface of the neighboring neuromuscular contacts is smooth. The sarcoplasm near the terminals of the muscle fiber contains a great number of mitochondria, in which a lot of cristae [3]. The axon terminals of type IIB fibers are elliptical and their synaptic vesicles are more generously provided with acetylcholine and other trophic factors. Postsynaptic folds of the synapses have linked with each other. In comparison with type IIA muscle fibers, the postsynaptic folds of type IIB fibers are longer and more regular and they cover a much larger area of the sarcoplasm [21]. In type IIB fibers, the contact area is the largest between the ending and the surface of the muscle fiber. In type IIB fibers, the postsynaptic folds extend near myofibrils and are separated from contractile structures by a thin sarcoplasmic layer. There is a large number of glycogen granules, few mitochondria and rarely any lysosomes in the terminals of neuromuscular synapses and in the postsynaptic area. Coated vesicles appear in the sarcoplasm of the postsynaptic area of type IIA muscle fibers [21]. The occurrence of coated vesicles is not only related to the resynthesis of acetylcholine in nerve endings, but these vesicles also carry the proteins of choline receptors onto the postsynaptic membrane. The connection with the rough sarcoplasmic reticulum influences the regulation of muscle fiber protein metabolism [22]. If sub synaptic folds open into T-tubules, they participate in the formation intermyofibril triads [23]. T-tubules in the sole plate form an extensive network, which together with the sarcoplasmic reticulum can form triads, the position of which makes them unusable for triggering muscle contraction [24].

Oxidative Capacity of Myofibres

The maximal oxygen consumption (VO_2 max) per Cross-Sectional Area (CSA) of muscle fibres in different vertebrates vary across a 100-fold range [19]. Muscle fibres with higher oxidative capacity have small CSA compared to fibres with low oxidative capacity. There exists relationship between VO_2 max, Succinate Dehydrogenase (SDH) activity [25] or oxoglutarate dehydrogenase activity [26] and consequently to the number of mitochondria [27]. Muscle fibres with a relatively large cross-sectional area had low SDH activities and vice versa [28,29]. So, type I and IIA muscle fibres have a relatively high oxidative capacity and small fibre CSA compared to type IIB/IIX fibres. Muscle fibre CSA, not necessarily the fibre type, is related to its oxidative capacity. There is an open question, why muscle fibres with higher oxidative capacity remain relatively small compared to muscle fibres with low oxidative capacity. Physiological function of muscle fibre type is an outcome of MyHC isoform expressed within fibre. Some fibres, the so-called hybrid fibres, express a combination of two or more MyHC isoforms [30,31]. Laboratory animal experiments have shown that the relative proportions of hybrid fibres vary significantly from muscle to muscle [30]. In human skeletal muscle, hybrid fibre types represent a significant population of fibres, but the stability of this fibre phenotype is currently unclear.

So, electrical stimulation increases the proportion of hybrid fibres [32] and mechanical load and thyroid hormone changes the proportion of hybrid fibres in skeletal muscle [33]. It has been shown that running exercise declines the hybrid fibres in human skeletal

muscle, whereas muscle hybrid fibres are relatively refractory to the effect of exercise in mice [34]. It is not clear what role hybrid fibres play in endurance athletes physical work capacity as well as the role of hybrid fibres in skeletal muscle oxidative capacity.

Protein Metabolism in Myofibres

It seems paradoxical that muscle fibres with high oxidative capacity have small CSA, but have a high intensity of protein synthesis rate compared to fibres with low oxidative capacity [19]. Muscle fibres with higher oxidative capacity contain higher quantities of satellite cells, myonuclei, mitochondria, mRNA, and total ribosomal RNA content. Expression of IGF-1, the stimulator of myofi brillar protein synthesis, is also higher in type I fibres [35]. Myostatin, inhibitor of muscle hypertrophy, expression is higher in type II fibres [36]. The components of the degradation system of muscle proteins, such as ubiquitin ligases MAFbx and MuRF, are about two-fold higher in fibres with higher oxidative capacity [19]. The higher rate of protein degradation in muscle fibres with higher oxidative capacity is balanced by a high rate of synthesis. This may be an important size limiting factor of muscle fibres with high oxidative capacity. As a result of that steady state protein turnover rate is faster in muscle fibres with higher oxidative capacity. In these fibres, the half-life of mitochondrial protein is the shortest although the turnover of cytochrome C is higher in the low oxidative fibres [37]. The term protein turnover was first described about three –quarter of century ago [38]. According to that all proteins are in a continuous renewal process and regulated by the synthesis and degradation rate. . Amino acids that are released during intracellular degradation of proteins are extensively reutilized for protein synthesis within the cell or transported to other organs where they enter intercellular recycling. The continuous turnover of muscle proteins determines how the myofi brillar or mitochondrial fractions balance change, and accordingly change muscle functional capacity [39]. Muscle fibres with higher oxidative capacity contain a higher level of cathepsin suggesting that there is a higher potential for protein degradation in muscles with high protein turnover [40]. The protein turnover rate in skeletal muscle fibres is very slow. After an acute lesion or in chronic pathological conditions, satellite cells are induced to proliferate and may change the protein turnover [41]. Vertebrate skeletal muscle fibres are multinucleate cells [42] with hundreds of thousands of myonuclei [43]. Each myonucleus regulates gene products within a finite volume called the myonuclear domain [44] or DNA unit [45] and has been defined as the theoretical volume of cytoplasm associated with a single myonucleus [46]. Cytoplasmic volume per myonucleus is smaller in fibres expressing slow MyHC isoforms and has higher oxidative capacity as compared to fast MyHC isoforms expression in fibers with low oxidative capacity [28]. The greater concentration of myonuclei in Slow-Twitch (ST) fibres has been shown to be related to a higher rate of protein turnover as compared with Fast-Twitch (FT) fibres [47]. RNA and protein ratio called the RNA unit and the protein synthesis per RNA unit has been defined as the "activity" of RNA [48] and shown as a stable indicator of protein synthesis or effectiveness of the translation process in cell in physiological conditions [49]. The protein turnover rate depends on the type of muscle fibres [50] and even functionally related proteins such as myofi brillar proteins have different speed of renewal in fibre types [51]. Changes in the turnover rate of myofi brillar proteins characterize the renewal processes in the contractile

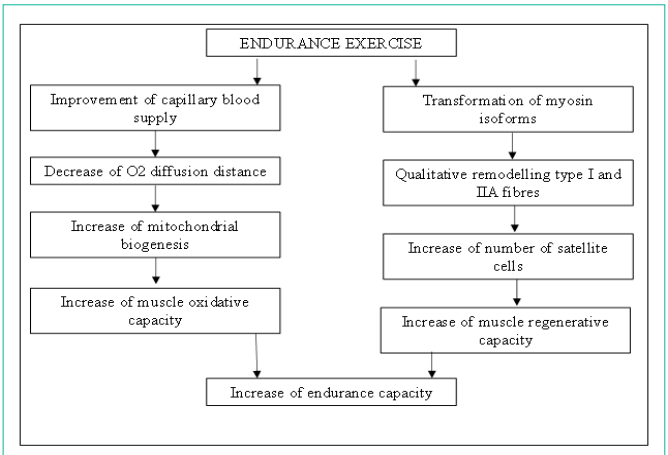

Figure 2: Effect of exercise on muscle endurance capacity.

apparatus during adaptation to endurance exercise [52]. As muscle fibres have limited capacity for hypertrophy and increase in oxidative capacity at the same time, it shows that a competition exists between the turnover rate of myofi brillar and mitochondrial proteins [19]. The turnover rate of MyHC isoforms does not only show differences between the muscle fibres of different twitch characteristics, but also between FT muscle fibres. The turnover rate is faster in FT muscle fibres with a higher oxidative capacity [20]. Faster myosin turnover rate supports qualitative remodeling of FT muscle fibres with higher oxidative capacity so that the former pattern of MyHC and Myosin Light Chain (MyLC) isoforms changes. This process shows that muscles with higher oxidative capacity adapt faster to the new condition of functional activity [53]. In the light of recent understanding, the expression of specific contractile, regulatory and minor protein isoforms is a relevant, though not the only mechanism of regulation of heterogeneity and plasticity of skeletal muscle. The pattern of myosin heavy and light chain isoforms in skeletal muscle might change for several reasons but during endurance exercise these changes reflect adaptation of contractile apparatus to the endurance exercise (Figure 2).

Endurance Exercise and Muscle Energetics

Endurance exercise increase skeletal muscle oxidative capacity as stimulate mitochondrial biogenesis and improves their functional parameters [7,54]. Exhaustive endurance exercise results in a marked decrease in cytochrome c/aa 3 ratio in striated muscles with high oxidative capacity (heart muscle), and the reason for the deficit of cytochrome C is the disruption of the mitochondrial outer membrane [55]. In striated muscles with high oxidative potential intracellular phosphotransfer systems constitute a major mechanism linking mitochondria and ATPase within specific structures - intracellular energetic units [56]. Mitochondria are positioned between the myofi laments throughout the whole muscle [57]. The effectiveness of metabolic signalling depends on structural-functional relationships of the interaction between mitochondria and sarcomeres [58]. Under conditions of hypoxia, the connections between mitochondria and sarcomeres are disturbed, sarcomeres disintegrate the muscle cell structure and cause cell injury and death [58]. The activation of apoptosis may be partly responsible for the initiation of protein degradation and loss of muscle nuclei associated with local

atrophy [59]. So, the disruption of desmin impairs the linking of mitochondria to Z-disc and skeletal muscle exhibits impaired oxidative phosphorylation [60] AMPK becomes activated in skeletal muscle during acute bouts of exercise [61]. AMPK's main function is to monitor the energy status of muscle fibres and maintain muscle energy homeostasis [62]. Exhaustive high volume endurance exercise lead to the depletion of the energy system, neuromuscular fatigue and muscle damage [63]. Children have less muscle mass than adults and generate lower absolute power during high-intensity exercise. Children's muscles are better equipped for oxidative than glycolytic pathways during exercise and have a lower ability to activate their type II muscle fibres [64]. Skeletal muscle oxidative capacity increases with endurance exercise and an age-associated decline in oxidative capacity is related to the reduction in fitness [65]. Endurance exercise in aerobic zone of metabolism is most effective to increase oxidative capacity of muscle fibres (Figure 2).

Effect of Endurance Exercise on Contractile Proteins Metabolism

Changes in muscle proteins renewal during endurance exercise show the process of adaptation of muscle structures to the endurance type of activity. From functional point of view this is the redistribution of amino acids .Amino acids are derived from protein breakdown and incorporated into the newly synthesized protein. Exercise in the aerobic metabolic zone stimulates protein renewal by increasing muscle protein degradation and synthesis rate in the recovery period after exercise [66]. The renewal of MyHC and MyLC isoforms provides a mechanism by which the type and amount of protein changed in accordance with the needs of the myofibrillar compartment in the process of adaptation to endurance exercise [20,67]. In muscle fibres where MyHC I and IIa isoforms are dominant isoforms have relatively high oxidative capacity and are recruited during endurance exercise [68]. In rat FT muscles, the difference in oxidative capacity is about 10 % and endurance exercise increased the oxidative capacity about 15-20% [20]. Therefore the role of gene expression and exercise separately in MyHC isoforms formation is unresolved [69]. In different FT muscles MyHC isoforms turnover rate between FT muscles is faster in muscles where oxidative capacity is higher [20]. MyLC isoforms' relative content plays an important role in the process of modulation of the contractile apparatus during the increase of oxidative capacity and degradation rate of contractile proteins [67]. C-protein is sensitive to endurance exercise volume and affects muscle mechanical properties [70]. Particularly sensitive are C-protein and MyHC fast isoforms to excessive increase of exercise volume as functional properties of myofibrils are changing [17].

Changes in MyHC and MyLC Isoforms

Myosin isoforms play essential regulatory role in muscle contraction. Myosin isoforms have different function as well as different protein metabolism. MyLC isoforms renewal is faster than MyHC and their recovery period after endurance exercise is shorter [71]. Endurance exercise intensified degradation rate of MyHC isoforms [17], cause decrease in expression of MyHC IIB isoform and this is the main reason of unchanged turnover rate of MyHC IIB isoform [4]. Decrease of MyHC IIB isoform relative content during endurance exercise in FT skeletal muscle shows the transformation of muscle contractile apparatus in accordance with the changes of muscle oxidative capacity. Decrease of MyHC IIB isoform relative content in this case does not show decrease of muscle contraction velocity. A positive correlation between MyHC IIB isoforms, MyLC 3 fast isoform relative content and muscle contraction velocity shows the regulatory function of MyLC isoforms [10]. MyLC 3 fast isoform relative content is increasing in rat FT muscle during endurance exercise. MyLC alkali as well as MyLC regulatory isoforms relative content does not change in FT muscles during endurance exercise [20]. Decrease of alkali and in regulatory MyLC slow isoform relative content during endurance exercise and increase of MyLC 3 fast isoform relative content in FT muscle are not in agreement with changes in MyHC isoforms pattern. Therefore, stoichiometry of Myosin subunits and their association with each other do not change [67]. This support the standpoint that no restrictions exists in adaptation to endurance exercise between MyHC and MyLC isoforms in FT muscles and shows that there are coordination between changes in skeletal muscle oxidative capacity and contractile apparatus through muscle metabolism. All these changes in FT muscles during endurance exercise show recruitment of these muscles in endurance exercise [20,67].

Adaptation of Neuromuscular Spindles to Endurance Exercise

Muscle spindles (intrafusal muscle fibres) are responsible for muscle stretch and are composed of nuclear Bag1 (dynamic Bag1), nuclear Bag2 (static Bag2), and chain fibers (static chain). During endurance exercise, intrafusal muscle fibres show metabolic changes but not signs of hypertrophy [72]. Neuromuscular spindles have ability to contract and shorten simultaneously with skeletal muscle (extrafusal muscle) shortening. So, it is like channel of transmission of information about muscle length of muscle fibres and the speed of contraction to higher centers of motor control at any time during exercise. The motor zone of neuromuscular spindles is located near the poles, where the space under the capsules significantly diminishes. Each intrafusal fiber type has specific MyHC composition [73]. Intrafusal fibers have great variability in phenotypic expression and this is related with plasticity of muscle precursor cells [74]. During muscle spindle regeneration, intrafusal satellite cells develop into extrafusal-like muscle fibers due to their motor innervation [75]. Endurance exercise does not change the myosin isoform composition in Bag1 and Bag2 fibers but does so in chain fibers [76]. At the intensity of exercise 60-75% of VO_2 max, a correlation was found between the recruitment of Bag1 and extrafusal slow-twitch type I fibers, between Bag2 and extrafusal type IIA fibers and between nuclear chain and extrafusal IIB fibers [77]. Endurance exercise causes essential destruction in the myofibrillar apparatus of nuclear bag spindles (Figure 3). Focal destruction of myofibrils occurs both in the extrafusal and intrafusal fibers [78]. Mainly peripheral myofibrils lyse [3]. There are sarcomeres in intrafusal fibers, where only single thick filaments are missing on the border of the H-zone and where the majority of thick filaments in the A disc have completely lysed [3]. Actin filaments are more resistant to proteolytic enzymes activity also in intrafusal muscle fibers. During short lasting intensive exercise, Bag2 fibers play the important role in the early phase of adaptation to exercise [79]. Destruction of structures in the fibers of muscle spindles is intensive after exercise. During the recovery period the regeneration of these structures intensifies as endurance exercise increase the regenerating potential. This is confirmed by a large

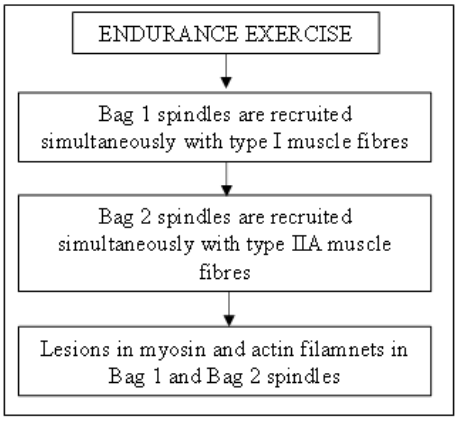

Figure 3: Destruction of muscle spindls structures.

number of satellite cells and polyribosome [3].

Adaptation of Nerve Endings of Neuromuscular Spindles to Endurance Exercise

Motor neurons controlling extrafusal muscle fibers are larger (alpha motor neurons), whereas the motor neurons that innervate the muscle spindles (gamma motor neurons) are smaller. The interaction of the alpha and gamma motor neurons during muscle contraction in the endurance exercise is important because the central part of the intrafusal fibers must not become slack at any time of during muscular activity [3]. There are two types of motor nerve endings of the intrafusal nuclear bag static muscle fiber. Motor nerve endings located near the centre of the spindle have postsynaptic membranes. This membrane form postsynaptic folds around the gamma-axon terminal [3,78]. The synaptic cleft filled with the basal membrane is between pre- and postsynaptic membranes of synapses. The axon terminal contains a lot of mitochondria, which are full of cristae. There are small vesicles containing acetylcholin located between mitochondria, and very few mitochondria in the postsynaptic area [78]. As intrafusal muscle fibers are destined to become slack when the extrafusal fibers shorten, unless they also shorten to the same degree due to the gamma motoneurons, these ultrastructural changes during exercise support the idea of an increase of alpha-gamma co activation during regular exercise [3]. Intrafusal muscle fibers located in the region of type I extrafusal muscle fibers adapt to endurance exercise by using a response reaction similar to that of extrafusal fibers. Comparison structural rearrangements in the nerve-muscle synapses of the extra- and intrafusal muscle fibers show that destructive changes in the intrafusal fibers are smaller than changes in the synapses of extrafusal fibers [3]. This is result of the effect of tension, which in case of endurance exercise in the synapses of intrafusal muscle fibers is lower than in the synapses of extrafusal fibers [78]. The comparison of changes in the ultrastructure of different types of extrafusal and intrafusal muscle fibers, and their innervation and regeneration potential during endurance exercise show that all described changes are in mutual relationship and depending on the character of exercise

Conclusion

This review has investigated the adaptation of skeletal muscle to endurance exercise including different types of extra- and intrafusal muscle fibres. Endurance exercise improves the energetic of skeletal muscle, supports the functioning of the myofibrillar apparatus for a longer time and increases the oxidative capacity of skeletal muscle, stimulating mitochondrial biogenesis. The muscle fibres where myosin heavy chain I and IIa isoforms are dominant have a relatively high oxidative capacity and are recruited during endurance exercise. Analysis of structural rearrangements in the nerve-muscle synapses shows that destructive changes in intrafusal fibres are smaller than in extrafusal fibers. The comparison of changes in the ultrastructure of different types of extrafusal and intrafusal muscle fibres, and their innervation and regeneration potential during endurance exercise show that the described changes are in mutual relationship and depend on the type of exercise.

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