NATIONAL OLYMPIC COMMITTEE OF ESTONIA

ESTONIA

ACTA ACADEMIAE OLYMPIQUAE ESTONIAE

volume 14 number 1/2 2006 Tartu

NATIONAL OLYMPIC COMMITTEE OF ESTONIA

ACTA ACADEMIAE OLYMPIQUAE ESTONIAE

Indexed in International Databases of Sportdata and EBSCO Publishing SPORTDiscus with Fulltext

VOL. 14 Number 1/2 , 2006 TARTU

AS Atlex
Kivi 23
51009 Tartu
Phone +372 734 9099
Fax +372 734 8915
e-mail: atlex@atlex.ee

ISSN 1406-1287

CONTENTS

OLYMPISM PRACTIQUE. PREPARATION FOR OLYMPIC GAMES

CHRONICLES OF THE ESTONIAN OLYMPIC ACADEMY

OLYMPISM PRACTIQUE.
PREPARATION FOR OLYMPIC GAMES

Acta Academiae Olympiquae Estoniae Vol. 14 No. 1/2, pp 5–17, 2006

EFFECT OF ENDURANCE TRAINING ON THE CHARACTER OF SKELETAL MUSCLE KINETICS

Karin Alev, Teet Seene

Institute of Exercise Biology and Physiotherapy, University of Tartu

Role of oxidative capacity of muscle fibres in endurance tralning

Endurance training promotes numerous adaptations in skeletal muscle, including enhancement of fibre oxidative capacity and muscle phenotype transitions. It has been demonstrated that aerobic endurance training leads to metabolic [36] and structural changes not only in slow-twitch (ST) but also in fast-twitch (FT) muscle fibres [15, 31]. Changes in the expression of myosin heavy chain (MyHC) and myosin light chain (MyLC) isoforms are in good agreement with the above-mentioned findings [28]. Endurance training increased the oxidative capacity in the plantaris muscle by 16% and in the extensor digitorum longus muscle by 12%. It has been shown that exercise training in FT fibres causes up-regulation of MyHC IIa and IIx isoforms, whereas the MyHC IIb is down-regulated [10]. Multidirectional changes have also been revealed in the relative contents of MyHC isoforms in different FT muscles if the training volume is increased [29]. These changes might be related to the differences in oxidative capacity of muscles [31]. Until now the question to what extent gene expression of MyHC isoforms is due to genetic predisposition or to the specificity of training remains unanswered [1].

Changes in molecular motor of contractile machinery in endurance tralning

The contractile protein myosin plays an important role in dictating the functional properties of skeletal muscle fibres. Myosin is known to exist as multiple isoforms in skeletal muscle as a result of polymorphic expression of both heavy and light chain components. It is unlikely that the whole variability in contractile machinery during endurance training depends on the content of MyHC isoforms. MyHC isoforms are the main determinants of muscle contraction, but it has been shown there is also some ground to believe that MyLC has a functional significance in the process of adaptation of sarcomeric proteins to the long-lasting exercise.

As a myosin molecule is formed by MyHC and MyLC isoforms and the functional significance of both in muscle contraction has been proved, it should be more useful to study the expression of MyHC and MyLC isoforms in parallel during endurance exercise training in order to obtain information about the adaptive pecularities in the contractile machinery.

The effect of endurance exercise on the MyHC profile appears to be both muscle specific and dose dependent, increase in the duration of training resulted in a progressive fast-to-slow shift in MyHC composition in rat skeletal muscle [10]. Wahrmann *et al.* [41] reported obvious fast-to-slow transitions of MyHC and MyLC profiles in rat hindlimb muscles with controlled regimens of endurance training. Edurance training also evokes transition in MyHC isoforms, in most cases transitions are limited to the fast-type subtypes and thus consist of a decrease of the fastest MyHC IIb isoform with attendedant increase in the MyHC IIa isoform [37].

The decrease of MyHC IIb isoforms during endurance training in FT muscles does not necessarily show that in these muscles contractile properties change towards ST as the relative content of MyHC IIa and IId isoforms increases [28]. Rather, these changes show that it is more economical for the FT muscles to perform the exercise. The decrease in slow isoforms both in alkali and regulatory MyLC during endurance training and the increase in MyLC 3_f isoforms in FT muscles is at first glance not in logical agreement with changes in the MyHC isoform pattern. However, the stoichiometry of these subunits and their association with each other do not change (MyHC IIb decreased but IId increased and MyLC 3_f is associated with both, IIb and IId MyHC to form myosin molecules). This shows that there are no definite adaptational borders between MyHC and MyLC isoforms in FT muscles to the long-lasting endurance exercise [28]. The adaptation process consists of both changes in MyHC and MyLC during aerobic endurance training and MyLC may also be associated with transformation of muscle function [29].

The slowest and fastest MyHC isoforms have a higher sensitivity to the process of degradation. In myopathic FT muscles MyHC IIb isoform is more sensitive to the serine proteinase than other isoforms [30]. Together with the slower synthesis rate [29] this may also explain the decrease in the MyHC IIb isoform in FT muscles during six weeks of endurance exercise.

Changes in contractiie proteins turnover rate in endurance training

As all myofibrillar proteins are in the continuous process of synthesis and degradation, changes in the turnover rate of the main contractile protein, myosin molecule, characterize these renewal processes in the contractile apparatus during the adaptation to the aerobic endurance training. The turnover of MyHC and MyLC isoforms provides a mechanism by which the type and amount of protein can be changed in accordance with the needs of contractile machinery during the adaptation to the exercise training [29]. It has been demonstrated that the turnover rate of MyHC isoforms shows differences between the FT muscles. The turnover rate is faster in FT muscles with a higher oxidative potential. Myosin turnover supports qualitative remodelling of FT muscles, so that the former pattern of MyHC and MyLC isoforms is changing, and the contractile process is better suited to new conditions of long-lasting muscle activity [1].

Changes in the turnover rate of MyHC isoforms in the FT muscles during the adaptation to the endurance training characterize also changes in the myofibrillar apparatus through protein metabolism [28, 29]. The latitude of changes (increase, decrease) in the turnover rate of a certain myosin isoform shows also the significance of MyHC isoforms in the process of adaptation to the endurance training. During endurance training MyHC IIa, IIb, IId, and MyLC 2_s and 3_f isoforms in FT muscles refiected explicitly the process of adaptation through changes in the relative content of myosin isoforms.

When seeking an answer to the question how does the prolonged mechanical activity affect the contractile apparatus in FT muscles, it is expedient to begin with the backbone – the myosin molecule. Although the exact role of MyLC isoforms in FT muscles during the adaptation to the aerobic endurance training is not fully known, changes in the relative content of MyLC isoforms and their relations with the character of training show that they play an important role in the process of modulation of contractile machinery during the increase in the oxidative capacity and more intensive degradation rate of contractile proteins [29, 32]. Simultaneously with increased degradation of contractile proteins, endurance training also increased the degradation rate of MyHC isoforms. The degradation rate of MyHC isoforms increases in spite of the increase in the oxidative potential of the FT skeletal muscle. The decrease in the expression of MyHC IIb isoform in FT muscles is caused by the intensive degradation of the isoform during endurance training, which is probably the main reason for unchanged turnover rate of MyHC IIb isoform in endurance-trained rats [28]. During the adaptation to the long-lasting endurance exercise a decrease in MyHC IIb isoform in FT skeletal muscle points to the transformation of the muscle contractile apparatus in accordance with the increase in muscle oxidative capacity. This adaptational process shows coordination between changes in oxidative capacity and contractile machinery in skeletal muscle during the adaptation to the endurance training first of all in relation to muscle metabolism [29]. Adaptational processes in FT muscles during endurance training show high potential of recruiting these muscles [28].

Role of myosin in the diversity of skeletal muscie fibres

The generation of muscle fibre heterogenity is based on the gene regulation through two main mechanisms.

1. Qualitative mechanism – muscle protein (like myosin) may exist in forms, which are similar but not identical (isoforms). Replacement of isoforms represents the frist mechanism generating diversity among muscle fibres
2. Quantitative mechanism – differential expression of the same gene. The proportion of the products of these genes will therefore be modified and new functional or structural features will appear [7].

During muscle development from embryonic till adult stage several myosin isoforms are sequentially expressed. Synthesis of these isoforms is repressed at a given stage of development when they are replaced by the adult isoforms [42].

Myosin plays an important role in dictating the functional properties of skeletal muscle fibres. Myosin is known to exist as multiple isoforms in striated muscle as a result of polymorphic expression of both its heavy and light chain components [12]. At present nine distinct isoforms of the MyHC have been identified in mammalian skeletal muscle, of which four are thought to be expressed in rodent limb muscles [25]. Like the other contractile proteins the MyLC represents a family of isoforms, at least three isoforms of the alkali LC (one slow – $LC1_s$ and two fast – $LC1_f$ and $LC3_f$) and two isoforms of the regulatory LC – $LC2_s$ and $LC2_f$ have been identified in rat skeletal muscle.

Changes in the expression of MyLC can be included in FT muscle by chronic low-frequency stimulation [9]. Stimulation induces a sequential exchange of fast light-chain isoforms with their slow counterparts. *In vitro* experiments show, the alterations at the translation process change the pattern of specific mRNAs. Studies of co-existence of MyHC and MyLC isoforms in the same muscle fibre show that MyHC IIa is preferably associated with MyLC 1_f, whereas MyHC IIb is favourably associated with MyLC 3_f. Early studies of Pette *et al.* [24] considered that variations in an amount of MyLC 3_f in single type II fibres reflected subpopulations of type II fibres.

Prolonged endurance training elicits a decrease in the ratio of MyLC 3_f to MyLC 1_f concurrently with the transformation from type MyHC IIb to MyHC IIa fibres [38].

The possible lower affinity of MyLC 3_f for MyHC IIa than MyHC IIb may be related to enhanced degradation of MyLC isoform [30]. In FT muscles stimulated with low frequency, MyLC 3_f is related to an increase in the free form on MyLC 3_f, concomitant with the replacement of MyHC IIb by MyHC IIa [39]. However, it is uncertain whether or not in all mammalian skeletal muscles such a relationship between MyLC and MyHC isoforms is applicable and maintained with increased or decreased contractile activities since the stimulation-induced changes in the MyLC pattern of rabbit FT muscles vary greatly from those of the rat [9].

Force development and shortening in muscle result from interaction of myosin and actin. In vertebrate muscle fibres, the extent of interaction between actin and myosin is regulated by the concentration of sarcoplasmic Ca^{2+}. Ca^{2+} regulation of contraction in vertebrate striated muscle is mediated by troponin and tropomyosin whereas striated muscles of various invertebrate species are regulated by Ca^{2+} binding directly to myosin [22].

Information from the crystal structure of the subfragment 1 (S1) of skeletal muscle myosin suggests that MyLC may stabilize the α-helical neck region of the myosin head [23] so that the force resulting from conformational changes near the active site is transmitted to the rod region of the molecule [26]. It has been shown that the removal of up to 50 per cent of the endogenous regulatory MyLC has little effect on either maximum Ca^{2+}-activated force or stiffness but significantly increases force and stiffness at submaximum levels of Ca^{2+} of skinned skeletal muscle fibres [14]. In vitro force measurements [35] confirmed the results of Hofmann *et al.* [14] that the removal of regulatory MyLC has little effect on maximum force. Partial extraction of regulatory MyLC from skinned skeletal fibres indicated that it may be involved in conferring Ca^{2+} sensitivity on cross-bridge transitions that limit the rate of force development in steadily Ca^{2+}-activated fibres [22]. In fibres containing a mutant myosin regulatory LC having a defective divalent cation binding site, both maximum tension and stiffness were significantly reduced compared to control values [11], suggesting that myosin heads containing regulatory LC that is unable to bind Ca^{2+} or Mg^{2+} have a reduced ability to form strongly bound cross-bridges. These findings suggest that rather than playing a strictly structural role such as stabilizing the structure of the myosin head, regulatory MyLC may also serve a regulatory role, such as modulating the availability of cross-bridges to bind to actin.

Two myosin LCs, one regulatory LC and one alkali LC, stabilize an extended α-helical segment in the hinge region of each MyHC. The MyLCs are necessary for full force development [19]. Removal of either alkali or regulatory LC markedly reduces myosin velocity *in vitro* motility assay [20]. Like the other contractile proteins, the alkali MyLCs represents a family of isoforms. Five alkali MyLC isoforms have been found in human skeletal muscle: embryonic alkali LC that is mainly expressed in embryonic muscle tissue, a major and minor slow isoform, and two fast isoforms alkali $LC1_f$ and $LC3_f$, which are both encoded on the same gene. Alkali MyLC isoforms can bring forward different contractile properties on a given MyHC [27]. The alkali MyLCs are expressed in a fibre-type specific manner. In adult human skeletal muscle, FT fibres contain fast MyLC, whcreas ST fibres contain alkali MyLC 1_s and variable amounts of the two fast alkali MyLCs [16].

Role of myosin in muscle fibres shortening velocity

The MyHC isoform composition of individual muscle fibre is the primary determinant its maximal shortening velocity and power output, while the MyLC isoform complement has a modulatory influence in regulating these properties [7]. It is generally accepted that the maximum velocity of shortening correlates with the MyHC isoform, several observations suggest an additional impact of the alkali light chain complement. Several studies suggest that alkali MyLCs also have a role in determining maximum velocity of shortening [4, 13, 18, 34].

Difficulties in establishing a relation between maximum shortening velocity and myosin isoforms are caused by the preferential association between MyLC 3_f and MyHC IIb and between MyLC 1_f and MyHC IIa [38]. The question whether the functional significance of variations in the relative concentration of $LC3_f$ has been adressed by several authors. The maximum contraction velocity of a single type I fibre is approximately one tenth that of type IIX fibre. The velocity of type IIA fibres is somewhere between those of type I and type IIX. Although the maximum velocity of shortening correlates with both MyHC and alkali MyLC isoforms, several authors suggest an additional impact of regulatory MyLC [17, 21]. *In vitro* motility assay indicates that the removal of a regulatory LC evokes a pronounced decline in the velocity of actin filaments on myosin [20, 35]. The role of regulatory LC in shortening velocity is supported by a single fibre study on human muscle [17]. A role of MyLC 3 in maximum shortening velocity was suggested by the findings that maximum shortening velocity is higher fibres containing larger amounts of MyLC 3_f [4, 7]. The variability of maximum shortening velocity observed in type II fibres is interpreted to be attributable primarily to differences in the alkali MyLC complements.

IIB fibres, in fact, could be faster than IIA fibres, not because they contained MyHC IIb, but because they contained larger amounts of MyLC 3_f and *vice versa* [7]. To address this problem, it is necessary to relate maximum shortening velocity to the alkali MyLC ratio in single fibre containing only one known MyHC isoform. The only paper that followed this approach [17] had to deal not only with the inability to separate all three fast MyHC isoforms but also with the problem that the human fast fibres showed co-existence of two of regulatory MyLC isoforms (MyLC 2_f and MyLC 2_s). Under these circumstances, no relationship between maximum shortening velocity and alkali MyLC ratio was found either in IIA or in IIB fibres. Undetected MyHC co existence and variations in the alkali MyLC isoform ratio might form the basis for the large variability of maximum shortening velocity among fast fibres presumed to contain the same fast MyHC isoform [5, 8, 17].

It seems established that the high variability of maximum shortening velocity in fibres with the same MyHC content can fully account for alkali MyLC composition, that is the higher the MyLC 3_f content the greater the maximum shortening velocity [6, 7]. However, in human fibres, the same considerable variability in shortening velocity cannot be satisfactorily explained on the basis of MyLC isoform content. Alkali MyLC and regulatory MyLCs have no or hardly any role in explaining the variability in maximum shortening velocity independently of MyHC isoforms in nonnal physiological conditions. So far the findings show that it is unlikely that the whole variability in shortening velocity observed in human muscle fibres depends on MyLC content. The wide scattering of the relative concentration of $LC3_f$ within fibre types IIB, IID, and IIA indicated the existance of fibres identical by their myosin heavy chain complement, but differing their fast LC-based isomyosins. Differences in the fractional expression or the relative concentration of $LC3_f$ are of special interest with regard to the fact that $LC3_f$ and $LC1_f$ are both encoded by the same gene, although their trancription is under control of two specific promoters. The variable concentration of $LC3_f$ within each of the three fast fibre populations points to an independent regulation or to the existence of different thresholds of the two promoters toward a common regulatory signal. In this respect, it is interesting to note that the MyHC isoform, which seems more sensitive to alkali MyLC modulation in the rat, that is MyHC IIb, is not present in human skeletal muscle. This might partly explain why alkali LC has not been shown to significantly affect shortening velocity in human fibres [3].

In some studies, maximal shortening velocity was also affected by the ratio of MyLC 3/MyLC 1_f [5, 34]. However, Larsson and Moss [17] found no relationship between MyLC 3/MyLC 1_f and in human. In rodent muscle, MyLC 3/MyLC 2_f content varied significantly among a population of fibres that expressed only the IIB MyHC isoform and greater values of MyLC 3/ MyLC 2_f were associated with increased maximum shortening velocity [4].

The maximal shortening velocity of fast-twitch fibres increase with decreasing $LC1_f/LC3_f$ ratios. In IIA fibres the LC1/LC3 ratio is higher than in IIB fibres, but it is not entirely clear which subunits determine the contractile characteristics. Thus, although preliminary data showed differences in the unloaded shortening velocity of rabbit tibialis muscle fibres depending on whether they contain IIa or IIb MyHC isoform, these fibres also differed in their LC1/LC3 ratio [34]. It has been shown that the unloaded shortening velocity of rabbit *soleus* muscle fibres containing both type I and type IIa MyHC isoforms was related to their ratio. Until now it has been impossible to ascribe a role only to the MyLC of mammalian skeletal muscle. Phosphorylation of the regulatory LC alters the force-calcium relationship but has no clear effect on shortening velocity [33, 34]. Most studies have examined the possible role of the two alkali LC heterogeneity of myosin by measuring ATPase activity *in vitro*. In view of these findings, one may assume that $LC3_f$ is characterized by a lower affinity for MyHC I isoform than for MyHC IIa, IId(x), and IIb isoforms. It is likely that in ST fibres are composed solely of MyHC I isoform but the majority of the translated MyLC 3_f exists in free form [40].

Although the MyLC does not seem to affect the actin-activated myosin ATPase activity, it has a significant impact on the shortening velocity [21]. This influence is of interest in view of the existence of various isomyosins. The existence of two fast alkali MyLCs ($LC1_f$, $LC3_f$) generates three combinatorial patterns – a MyLC 1 homodimer, a MyLC 1/ MyLC 3 heterodimer, and a MyLC 3 homodimer. Their combination with a pair of regulatory MyLC and a MyHC homodimer results in three electrophoretically distinct isomyosins. Obviously, the number of isomyosins increases in hybrid fibres especially by coexistence of fast and slow MyLC isoforms in combination with MyHC isoforms.

References

1. **Baldwin KM, Haddad F.** Plasticity in skeletal cardiac, and smooth muscle. Invited review: Effect of different activity and inactivity paradigms on myosin heavy chain gene expression in studied muscle. J Appl Physiol 2001, 90: 345–357.
2. **Bicer S, Reiser PJ.** Myosin light chain isoform expression among single mammalian skeletal muscle fibers: species variations. J of Muscle Res and Cell Motolity 2004, 25: 623–633.
3. **Bottinelli R.** Functional heterogeneity of mammalian single muscle fibres: do myosin isoform tell the whole story? Eur J Physiol 2001, 443: 6–17.
4. **Bottinelli R, Betto R, Sciaffino S, Reggiani C.** Maximum shortening velocity and coexistance of myosin heavy chain isoforms in single skinned fast fibres of rat skeletal muscle. J Mus Res Cell Motil 1994a, 15: 413–419.
5. **Bottinelli R, Betto R, Sciaffino S, Regglani C.** Unloaded shortening velocity and myosin heavy chain and alkali light chain isoform composition in rat skeletal muscle fibres. J Physiol (Lond) 1994b, 478: 431–449.
6. **Bottinelli R, Reggiani C.** Force-velocity properties and myosin light chain isoform composition of an identified type of skinned fibres from rat skeletal muscle. Pflügers Arch. 1995, 429: 592–594.
7. **Bottinelli R, Reggiani C.** Human skeletal muscle fibers: molecular and functional diversity. Progress in Biopsyhs And Mol Biol 2000, 73: 195–262.
8. **Bottinelli R, Schiaffino S, Reggiani C.** Force-velocity relations and myosin heavy chain isoform compositions of skinned fibres from rat skeletal muscle. J Physiol 1991, 437: 655–672.
9. **Bär A, Simoneau JA, Pette D.** Altered expression of myosin light-chain isoforms in chronically stimulated fast-twitch muscle of the rat. Eur J Biochem 1989, 178: 591–594.
10. **Demirel HA, Powers SK, Naito H, Hyghes M, Coombes JS.** Exercise-induced alterations in skeletal muscle myosin heavy chain phenotype: dose-response relationship. J Appl Physiol 1999, 86: 1002–1008.
11. **Diffee GM, Greaser ML, Reinach F, Moss RL.** Effects of non-divalent cation binding mutant of myosin regulatory light chain on tension generation in skinned skeletal muscle fibres. Biophys J 1995, 68: 1443–1452.
12. **Diffee GM, McCue S, LaRosa A, Herrick RE, Baldwin KM.** Interaction of various mechanical activity models in regulation of myosin heavy chains isoform expression. J Appl Physiol 1993, 75 (5): 2517–2522.

13. **Greaser ML, Moss RL, Reiser PJ.** Variations in contractile properties of rabbit single muscle fibres in relation to troponin-T isoforms and myosin light chain. J Physiol 1988, 406: 85–89.
14. **Hofmann PA, Metzger JM, Greaser ML, Moss RL.** Effects of partial extraction of light chain 2 on the Ca^{2+} sensitivities of isometric tension, stiffness, and velocity of shortening in skinned skeletal muscle fibres. J Gen Physiol 1990, 96: 477–498.
15. **Hoppler H, Howald H, Conley K, Lindstedt S, Classen H, Vock P, Weibel E.** Endurance training in humans: aerobic capacity and structure of skeletal muscle. J Appl Physiol 1985, 59: 320–327.
16. **Jostarndt K, Puntschart A, Hoppeler H, Bllleter R.** Fibre type specific expression of essential (alkali) myosin light chains in human skeletal muscles. J Histochem Cytochem 1996, 44: 1141–1152.
17. **Larsson L, Moss RL.** Maximum velocity shortening in relation to myosin isoform composition in single fibres from human skeletal muscle. J Physiol Lond 1993, 472: 595–614.
18. **Li X, Larsson L.** Maximum shortening velocity and myosin isoforms in single muscle fibers from young and old rats. Am J Physiol 1996, 270: C352–360.
19. **Lowey S, Trybus KM.** Role of skeletal and smooth muscle myosin light chains. Biophys J 1995, 68: 120s–127s.
20. **Lowey S, Waller GS, Trybus KM.** Function of skeletal muscle myosin heavy and light chain isoforms by an in vitro mobility assay. J Biol Chem 1993a, 268 (27): 20414–20418.
21. **Lowey S, Waller GS, Trybus KM.** Skeletal muscle myosin light chains are essential for physiological speeds of shortening. Nature 1993b, 365: 454–456.
22. **Metzger JM, Moss RL.** Myosin light chain 2 modulates calcium-sensitive cross-bridge transitions in vertebrate skeletal muscle. Biophys J 1992, 63: 460–468.
23. **Patel JR, Dlffee GM, Moss RL.** Myosin regulatory light chain modulates the Ca^{2+} dependence of kinetics of tension development in skeletal muscle fibres. Biophys J 1996, 70: 2333–2340.
24. **Pette D, Henrlksson J, Emmericb M.** Myofibrillar protein patterns of single fibres from adult human muscle. FEBBS Lett 1979, 103: 152–155.
25. **Pette D, Staron RS.** Cellular and molecular diversities of mammalian skeletal muscle fibres. Rev Physiol Biochem Pharmacol 1990, 116: 1–76.
26. **Rayment JW, Rypniewskl WR, Scbmidt-Bäse K, Smlth R, Tomchick DR, Benning MM, Winkelmann DA, Wesenberg G, Holden HM.** Three-dimensional structure of myosin subfragment-1: a molecular motor. Science 1993, 261: 50–68.

27. **Schiaffino S, Reggiani C.** Myosin isoforms in mammalian skeletal muscle. J Appl Physiol 1994, 77: 493–501.
28. **Seene T, Alev K, Kaasik P, Pehme A, Parring AM.** Endurance training: volume dependent adaptational changes in myosin. Int J Sports Med 2005, 26: 815–821.
29. **Seene T, Kaasik P, Alev K, Pehme A, Riso EM.** Composition and turnover of contractile proteins in volume – overtrained skeletal muscle. Int J of Sports Medicine 2004, 25: 438–445.
30. **Seene T, Kaasik P, Pehme A, Alev K, Riso EM.** The effect of glucocorticoids on the myosin heavy chain isoforms' turnover in skeletal muscle. Journal of Steroid Biochemistry & Molecular Biology 2003, 86 (2): 201–206.
31. **Seene T, Umnova M.** Relations between the changes in the turnover rate of contractile proteins, activation of satellite cells and ultra-structural response of neuromuscular junctions in the fast-oxidative-glucolytic muscle fibres in endurance trained rats. Journal of Basic and Applied Myology 1992, 2 (1): 34–46.
32. **Seene T, Umnova M, Kaasik P.** The Exercise myopathy. In: *Overload, Performance Incompetence, and Regeneration in Sport.* Edited by M. Lehmann *et al.* Kluwer Academic Plenum Publishers, NY, 1999, 119–130.
33. **Sweeny HL, Bowman BF, Stull J.** Myosin light chain phosphorylation in vertebrate striated muscle: regulation and function. Am J Physiol 1993, 264 (Cell Physiol 33): C 1085–C 1095.
34. **Sweeny HL, Kushmerick MJ, Mabuchi K, Sréter AF, Gergely J.** Myosin alkali light chain and heavy chain variations correlate with altered shortening velocity of isolated skeletal muscle fibres. J Biol Chem 1988, 263 (18): 9034–9039.
35. **van Buren PG, Waller GS, Harris DH, Tryhus KM, Warshaw DM, Lowey S.** The essential light chain is required for full force production by skeletal muscle myosin. Proc Natl Acad Sci USA 1994, 91: 12403–12407.
36. **Viru A, Viru M.** Nature of training effect. In: Rarrett WE, Kirkendale DT (eds), Exercise and Sport Science 2000, 67–97.
37. **Wada M, Inashima S, Yamada T, Matsunaga S.** Endurance training-induced changes in alkali light chain patterns in type IIB fibers of the rat. J Appl Physiol 2003, 94: 923–929.
38. **Wada M, Pette D.** Relationships between alkali light-chain complement and myosin heavy chain isoforms in single fast-twitch fibres of rat and rabbit. Eur J Biochem 1993, 214: 157–161.
39. **Wada M, Katsuta S, Doi T, Kuno S.** Favourable associations between the myosin heavy-chain and light-chain isoforms in human skeletal muscle. Pflügers Arch 1990, 416: 689–693.

40. **Wada M, Okumoto T, Toro K, Masuda K, Fukuhayashi T, Kikushi K, Neihata S, Katsuta S.** Expression of hybrid isomyosins in human skeletal muscle. Am J Appl Physiol 1996, 271 (Cell Physiol 40): C1250–C1255.
41. **Wahrmann JP, Winand R, Riu M.** Plasticity of skeletal muscle in endurance-trained rats (I). A quantitative study. Eur J Appl Physiol 2001, 84: 367–372.
42. **Whalen R, Sell S, Butler-Browne G, Schwartz K, Bouveret P, Pinset-Hastrom I.** Three myosin heavy chain isozymes appear sequentially in rat muscle development. Nature 1981, 292: 805–809.

Acta Academiae Olympiquae Estoniae Vol. 14 No. 1/2, pp 18–24, 2006

IS STRENGTH TRAINING IN SPORT CENTRE AN AEROBIC ACTIVITY?

Toomas Karu[1], Ants Nurmekivi[2], Jaan Loko[2], Tõnis Saag[1]
[1]*Vomax OÜ*
[2]*University of Tartu, Institute of Sport Pedagogy and Coaching*

Abstract

The examination of athletes with stepwise increasing loads on an ergometer or a treadmill mostly show heart rates on an anaerobic threshold in the range of 160–180 beats per minute, measured with the methods of gas analysis and blood lactate concentration analysis. Persons training strength in sport centres claim that this is a pure aerobic activity, since the heart rate measured with sport tester is usually 120–140 beats per minute. This study aims at providing an analysis of the physiological effects produced in the organism by usually practiced strength-type training programme in a sport centre. The subjects investigated were two individuals who had been training in a fitness centre for a long period of time. Each exercise was performed 10 times; an exercise series comprised a four-fold repetition of this 10-fold exercise. During the whole training session the heart rate of the sportsmen was measured with sport tester, after each series of exercises a fingertip blood sample was taken to measure lactate concentration. After each exercise series the sportsmen were asked to evaluate on a 10-point scale, how difficult it had been for them to perform the series.

Conclusion. Strength exercises performed by fitness sportsmen in a sport centre produce a strong acidosis effect, as they are essentially of an anaerobic character. Simultaneously, relatively small changes occur in the heart rate even in the case of a subjectively very strenuous strength training.

Key words: strength training, heart rate, lactate, intensity.