Changes in Muscle Morphology and Activity in Trained and Sedentary Children

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Abstract: Children's muscles differ from adults' muscles both morphologically and in terms of their functional characteristics. Children's muscles consist of thinner fibers and rely more on oxidative metabolism rather than glycolytic metabolism. Due to the smaller diameter of the fibers, muscles in children have smaller conduction velocity. The morphological changes observed in trained children include type II fiber hypertrophy and increased fiber density. Nerve adaptation appears to contribute more to increased muscle strength than fiber hypertrophy. Neural muscle activation in children is rather limited because of the delayed maturation and myelination of efferent and afferent pathways. Children are more resistant to fatigue because of the smaller ratio of fast-twitch fibers, marked oxidative metabolism of muscles and lesser nerve activation of fast motor units. Moreover, they recover quicker than adults after physical exercise. It has been observed that the more efficient energy metabolism, the competent acid-base balance regulation, and the quick activation of the respiratory and cardiovascular system contribute to fast recovery. Decreased neural activation and increased fatigue resistance are also observed in individuals over 60 years of age.

1 INTRODUCTION

When it comes to children practicing sports, there are a lot of age-related changes in the muscles to be considered. These changes affect the composition and thickness of muscle fibers, muscle activation, fatigue resistance, etc. In older adults, muscle fibers become thinner, and certain changes in their metabolic profile and neural regulation are also observed. Physical exercise causes changes in children's muscles, so that their morphological structure resembles that of adults' muscles. Another effect of physical exercise is a delay in the age-related alterations in the muscles in old age. The dynamic muscle development at different ages and physical fitness can be evaluated by electromyographic techniques.

2 AGE-ASSOCIATED CHANGES IN MUSCLES

Children's muscles develop until adulthood (Ramaekers et al, 1993). The muscle cross-section is almost doubled between 5 to 20 years of age due to an increase in muscle fibre diameter and density which is accompanied by the functional division of fibre populations. Muscle development depends on genetic factors, age, muscle function, and physical activity (Gajdosik & Gajdosik, 2000). Thick muscle fibres (type II) are approximately 35% of all fibres at the age of 5 and about 50% at 20 years of age (Lexell et al, 1992; Noakes, 1992). That means that the oxidative metabolism in the muscles of children prevails over the glycolytic metabolism. Conduction velocity in adolescents' muscles is low because of the thinner muscle fibres. In the case of sedentary children, conduction velocity with minor and great effort is the same, which means efforts are executed with the help of identical in thickness thin oxidative fibres (Angelova, 2007).

Children demonstrate significantly lower achievements than adults during anaerobic activities. Anaerobic power increases during puberty and is associated with neuromuscular factors, hormonal factors, and improved motor coordination (Van Praagh & Dore, 2002).

In trained children, there are differences in conduction velocity during minor and intense efforts, similarly to adults. That is due to specific muscular

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changes in trained people such as fiber hypertrophy and changes in their metabolic profile.

Morphological changes in muscles of trained children such as hypertrophy of type II fibers, increased angle of spread and fiber density (Folland & Williams, 2007) are similar to those observed in maturation when fast-twitch muscle fibers become much thicker than slow-twitch muscle fibers.

In the mature individual, both slow and fast fibres are involved in the formation of muscle strength with great effort. Older adults (> 60 years) experience a loss of fast muscle fibres and an increased percentage of oxidative fibres (Boisseau & Delamarche, 2000).

3 CHANGES IN MOTOR CENTRES AND CENTRAL COMMAND WITH AGEING

During adolescence, the neural control of muscle activity changes and the muscle power increases. Peripheral efferent and afferent neural pathways mature and myelinate before 5 years of age. Central pathways take longer to maturate – up to 7 years for the afferent and 10 years for corticospinal pathways (Müller et al, 1994).

The motor unit synchronization level is limited in children up to 11 years, while in 14-year-old boys, the capacity for force generation is lower than that of young, trained men due to differences in muscle mass (Kanehisa et al, 1995). Children have less muscle mass and achieve less absolute power during high-intensity exercise. They develop less muscle force relative to the cross-sectional area of the muscle in comparison with adults (Kanehisa et al, 2001).

In trained individuals, the EMG amplitude is increased (Vikne et al, 2006) because of changes in neural activation of the various motor units (Gabriel et al, 2006), increased central nervous command to muscles and increased activation of single motor units (Griffin & Cafarelli, 2005).

Nerve adaptation appears to be more significant than muscle hypertrophy in trained children (Sale, 1989). The mechanisms, responsible for increasing muscle strength in children subjected to physical training, include increased activation of motor units (Gabriel et al, 2006) and improved motor habit, as well as increased muscle strength (Ozmun et al, 1994). The increase in muscle strength through training in adolescents, and especially in pre-puberty children, is mainly due to neural mechanisms. Additional reasons for this improvement are alterations in muscle function - the strength of a single muscle contraction and to some extent muscle hypertrophy (Falk & Eliakim, 2003).

High-frequency movement training leads to improved coordination and increased neural activation of muscles meaning an increased number of activated motor units and increased impulse frequency (Sale, 1989). That, in turn, leads to the development of muscle velocity, especially in childhood due to greater nervous system plasticity.

Muscle velocity of trained individuals is increased due to central factors – high-frequency impulses and increased muscle activation (Knight & Kamen, 2008).

4 INFLUENCE OF AGE AND FITNESS ON MUSCULAR FATIGUE

Central and peripheral fatigue occurs as a result of sustained maximal and submaximal (Löscher et al, 1996) volitional muscle contraction. Some possible mechanisms explaining central fatigue are suboptimal conduction facilitation from the motor cortex (Taylor et al, 2000), reduced facilitation of muscle spindles (Macefield et al, 1991), increased inhibition of afferent fibre type III and IV (Garland & Kaufman, 1995) and motoneuron desensitization (Kernell, 1969). The ability of the central nervous system to activate all muscle units during a maximal contraction varies with muscle groups, the type of muscle fibres and the individual (Löscher & Norlund, 2002).

Peripheral fatigue is a consequence of changes in the neuromuscular synapse and the contractile apparatus. Individual muscle fibers, motor units and whole muscles have been analyzed to study these changes. The accumulation of metabolic products and electrolytes as well as the depletion of energy substrates affect excitation-contraction coupling and lead to loss of strength (Green, 1997). Peripheral muscle fatigue is associated with disturbed calcium release and uptake by the sarcoplasmic reticulum. The change in electrolyte concentrations around the cell membrane, in turn, affects excitability and conductibility of action potentials (Westerblad & Allen, 1996).

Factors influencing peripheral fatigue are the supply of energy substrates (Sahlin et al, 1998), muscle fibre ratio (Thorstensson & Karlsson, 1976b), muscle strength before fatigue (Hunter &

Enoka, 2003), muscle length (Fitch & McComas, 1985) and the degree of fitness of the individual.

A smaller percentage of fast-twitch fibres, a greater oxidative muscle capacity, as well as a lesser ability for quick activation of fast motor units are observed in prepubescents, which explains their better resistance to fatigue. With such metabolism, muscles accumulate fewer metabolites during muscle contractions that contribute to the subjective feeling of fatigue. Faster recovery is observed after physical exercise and is the result of intensive resynthesis of macroergic phosphates, higher oxidative capacity, better regulation of the acid-base status, more efficient clearance of metabolites, and faster adaptation of the cardiovascular and respiratory systems to the physical exercise (Ratel et al, 2006). Children are more resistant to fatigue than adults during a maximal isometric effort and a series of high-intensity cyclic contractions.

Gradual age-related increase of glycolytic muscle activity

It is observed that in young athletes (20- 30 years of age) anaerobic metabolism and fatigue processes are more pronounced than in older athletes. Older individuals (over 60) have increased fatigue resistance with a lesser reduction in impulse frequency and conduction velocity of sustained isometric contraction in comparison with middle-aged people (Merletti et al, 2002). This difference occurs in dynamic and static contractions (Lanza et al, 2004). The reasons for this are mainly in the structures after the sarcolemma (Kent-Braun et al, 2000). In elderly individuals, the decrease in muscle strength is more gradual due to the loss of mostly fast-twitch muscle fibres with ageing and an increased percentage of oxidative fibres (D'Antona et al, 2003).

Halin et al. (Halin et al, 2002) measured a greater peak force relative to muscle cross-section, maximal average power spectrum and maximal conduction velocity in muscle fibres of middle-aged individuals as compared to prepubescent boys. In adults, these indicators show a more marked decrease in contrast with children. The fast-twitch muscle fibres in adults are activated to a higher degree at a maximal force which leads to the accumulation of lactate and ions. That leads to a reflex reduction in impulse frequency which can be explained by the "muscle wisdom" hypothesis for neural command adaptation during a sustained contraction. Adult individuals are more resistant to fatigue in both isometric and dynamic contractions (Lanza et al, 2004). The explanation for that is the reduced number and size of the fast-twitch muscle fibres. Sustained physical activity may counter the loss of fast-twitch muscle fibres with ageing (Casale et al, 2003).

Influence of fitness activity

Fatigue processes are more pronounced in trained children than in sedentary ones due to a higher percentage of activated motor units at maximal effort. (Gabriel et al, 2006). Some differences in fatigue development are known to result from individual variations in muscle activation. Muscle activation during maximal voluntary contraction varies between 80 and 100 %, (Kawakami et al, 2000). In elite athletes trained for strength or speed, motor unit activation approaches 100%. The more pronounced processes of muscle fatigue in trained children compared to non-trained children are manifested by a reduction in conduction velocity during considerable muscular effort (Angelova et al, 2007). That is due to the increased capacity for activation of fast motor units that are less resistant to fatigue (Klaver-Król et al, 2007).

In a study of gymnasts and sedentary 10-year-old boys, Halin et al. (Halin et al, 2002) found that the gymnasts had a greater maximal muscle force correlated with muscle cross-section as well as a higher median of the power spectrum at a maximal contraction and a steeper decrease in the power spectrum median during fatigue. The higher frequency of the power spectrum results from the activation of more motor units during intense efforts. Mendez-Villanueva et al (Mendez-Villanueva et al) and Nordlund et al. (Nordlund et al, 2004) found that athletes who had a higher maximal muscle speed and anaerobic reserve power (the difference between the maximal anaerobic and aerobic power) are prone to faster muscle fatigue development, resulting in reduced conduction power and EMG amplitude. More lactate accumulates, and a more significant reduction of conduction velocity and power spectrum during fatigue is reported in people with predominantly type II muscle fibres (Taylor, 1997).

Prolonged physical exercise leads to an increase in the potassium concentration in the plasma (Overgaard et al, 2002) and the interstitial space (Nielsen & Clausen, 2000). This alters the electrochemical gradient of the sarcolemma for potassium and reduces its excitability (Fitts & Balog, 1996b). Small changes in the M-wave after prolonged aerobic exercises (Millet & Lepers, 2004) show that changes in excitability are not significant, but during intensive exercise, they become considerable (Green, 1997). Studies on endurance-trained athletes have shown that increased potassium concentration has a less significant role than the changes in the potassium concentration in isolated

muscles (Nielsen & De Paoli, 2001). The sodium-potassium pump density in muscle membrane increases after a 100-metre sprint (Overgaard et al, 2002) which is also valid for individuals who train regularly (Nielsen & Clausen, 2000). This adaptation mechanism reduces the ionic imbalance in the interstitial space of the muscle. This causes a decreased likelihood of changes in the membrane excitability which leads to increased muscle resistance to fatigue (Green, 2004).

Conduction velocity of muscle fibres correlates with the percentage of myoglobin heavy chains type I, characteristic of slow muscle twitch fibres, as well as with the power corresponding to the lactate threshold and the maximal oxygen consumption (Farina et al, 2007). Thus, conduction velocity, as an indicator of muscle fatigue, is directly linked to the percentage of oxidative metabolism in muscles.

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