ADAPTATION TO SUBMAXIMAL PHYSICAL TRAINING

Nickolay Boyadjiev
Department of Physiology, Medical University, Plovdiv, Bulgaria

Abstract:
Submaximal training can induce adaptational changes in the skeletal muscles, cardiovascular system, respiratory system, endocrine system, etc. Knowledge of the signs of adaptation and its magnitude, the factors causing it, as well as an awareness of the significance of the adaptation for the improvement of physical capacity to exercise is essential to develop an optimal training programme and with an adequate recovery process to achieve the maximum positive effect with the minimum negative effect on the athlete’s health. Body adaptation resulting from regular aerobic training can involve adaptation of the muscle enzyme systems, changes in the vascularization and type characteristic of muscle fibers, metabolic changes in the trained muscles and muscle groups related to the glucose utilization and the use of glycogen and fats as sources of energy, changes in the blood, the immune and coagulation status of the body, as well as changes in the parameters of aerobic working capacity of the organism. Regular submaximal exercises have been found to increase the activity of key oxidative enzymes in the metabolic pathway for the breakdown of carbohydrates - hexokinase (HK) and citrate synthase (CS) and fats – 3-hydroxyacyl-CoA dehydrogenase (3-HAD) and carnitine palmitoyltransferase (CPT) and in the respiratory chain citochrome c oxidase (CCO). Submaximal training induces vascularization in the muscles containing predominantly oxidative muscle fibers. The muscle fibers in the groups of muscles trained submaximally can even undergo a type transformation. The insulin-mediated glucose transport in the cells of the recruited muscles is selectively increased. A metabolic adaptation occurs as the body shifts from using carbohydrates for energy to using fats; the subsequent “glycogen-sparing” effect enhances the working capacity. Permanent changes occur in the humoral immunity evidenced by the increase of the serum IgA and IgG concentrations. The system of oxygen transport in the blood adjusts to more economical operation. As a final result both the external system of oxygen delivery and the mitochondrial system of oxygen utilization undergo adaptation which leads to a moderate increase of maximal oxygen consumption, but greater increase of the oxygen running economy.

Key words: submaximal exercise, adaptation, athletes, aerobic working capacity

ADAPTATION AN DAS SUBMAXIMALE KÖRPERLICHE TRAINING

Zusammenfassung:
Das submaximale Training kann Anpassungssänderungen sowohl in Skelettmuskeln, im Herz-Kreislau- und Atmungssystem und im Endokrinium als auch in anderen Systemen verursachen. Sowohl das Kenntnis der Adaptationszeichen und des Adaptationsausmaßes, sowie das Kenntnis der zugrundeliegenden Faktoren, als auch das Bewußtsein über die Wichtigkeit der Adaptation für die Verbesserung der körperlichen Leistungsfähigkeit sind unentbehrlich, um ein optimales Trainingsprogramm zu entwerfen, und mit einem angemessenen Erholungsprozess einen maximal positiven Effekt mit einer minimal negativen Auswirkung auf die Gesundheit des Sportlers zu erzielen.

Die Körperadaptation, als Folge eines regelmäßigen aeroben Trainings, umfasst die Anpassung der Muskelenzymsysteme, Änderungen der Vaskularisation und der Muskelkapsel, Stoffwechselveränderungen in trainierten Muskeln und Muskelgruppen, die mit Glukoseverbrauch verbunden sind, den Verbrauch des Glykogens und der Fette als Energielieferanten, Veränderungen im Blut, im Immunsystem und Koagulationsstatus des Körpers, sowie die Veränderungen von Parametern der aeroben Kapazität des Organismus unter Belastung.

Es wurde festgestellt, dass regelmäßige submaximale Übungen die wesentlichen oxidativen Enzymen in Stoffwechselprozessen fördern, die für die Aufspaltung von Kohlenhydraten - Hexokinase (HK) und Zitrat-Synthase (ZS), von Fetten – 3-Hydroxyacyl-CoA- Dehydrogenase (3-HAD) und Karnitin-Palmitoyl-Transferase (KPT) und Zitokrom c-Oxidase (ZCO) im Respirationsstrakt verantwortlich sind. Das submaximale Training ruft Vaskularisation in den aus überwiegend oxidativen Muskelfasern bestehenden Muskeln hervor. Die Muskelschäden in den submaximal trainierten Muskelgruppen können sogar eine
Introduction

A training exercise, methodologically properly performed and conducted regularly at submaximal levels (with an intensity which requires oxygen consumption of about 65-75% of VO_2max), is a major way to create structural and functional bases to achieve a high aerobic working capacity (Wilmore & Costill, 1999). This type of training causes adaptational changes in the skeletal muscles (increased activity of key oxidative enzymes), in the cardiovascular, respiratory, and endocrine systems which lead to a greater aerobic working capacity (increased maximal oxygen uptake and submaximal endurance) and hence to an improvement in sports results (Boyadjiev, 1996a). To know these changes, their scope, and the direct factors causing them gives you a powerful means to gain, through an optimal training plan and adequate recovery under the guidance of a trainer and medical staff, maximum results with a minimum adverse effect on the health of athletes.

Enzyme adaptation in muscles

Endurance training induces a distinct adaptation in the recruited muscles. They can be evaluated both morphologically and biochemically by using biopsy techniques.

It is well known that the major pathways for energy supply to the working muscles are:
- glycolysis / glycogenolysis,
- fatty acid oxidation (beta-oxidation),
- Krebs cycle,
- oxidation in the respiratory chain.

The capacity of these pathways is restricted mainly by the quantity (activity) of key enzyme active there. An increase of the quantity (activity) of some of these key enzymes can theoretically increase the capacity of the entire metabolic pathway with the activity of other enzymes (inactive in this pathway) remaining unchanged (Newsholme & Leech, 1983).

It is worth noticing here that the changes in the metabolic pathway capacity (increasing as a result of training or decreasing as a result of detraining) occur simultaneously with the changes in quantity (activity) not only of the key enzyme but also of the other enzymes of the pathway. For example, as a result of chronic (10-week) stimulation of rabbit's muscles the activity of succinate dehydrogenase (SDH) and citrate synthase (CS) in m. soleus increases linearly for three weeks to respectively 600% and 300% of baseline levels, and then starts decreasing until the 10th week to 250% and 150% of the baseline values (Figure 1).

![Figure 1. Enzyme activity changes (% of initial level) as a result of muscle electrostimulation in experimental rabbits.](image-url)
in the analysis of beta-oxidation of fatty acids,
- citrate synthase (CS) in the analysis of Krebs cycle,
- cytochrome c oxidase (CCO) for the assessment of the respiratory chain.

The enzymes in the Krebs cycle and fatty acid beta-oxidation are usually defined as oxidative. The maximal change due to regular submaximal training (7- to 10-fold increase) occurs in the period between weeks 3 and 5 (Boyadjiev, 1996b). After cessation of training their activity drops to baseline levels after 5 to 6 weeks.

While studies on muscle tissue enzymes of experimental animals had been performed even by 1970, human studies of biopsic material were not performed until 1970 and 1971 (Morgan, Cobb, Short, Ross, & Gunn, 1971; Verinauskas, Bjortorp, Fahlen, Prerovsky, & Sternberg, 1970). From 1970 to 1980 the techniques for obtaining biopsic material as well as the methods for its processing (buffering, organelle disintegration, etc) were perfected. The changes are differently expressed in experimental animals and people but in all cases they are similar (Figure 2 and Figure 3).

The explanation that enzyme adaptation occurs as a result of submaximal training can be explained by the fact that the enzymes have a specific “life cycle” and, accordingly, each has a specific half-life, which varies from 1 week (for the mitochondrial enzymes) to 1 day (for the glycolytic enzymes). The cellular content (the metabolic activity) of the enzyme is always a result of the balance between its synthesis and its disintegration. The submaximal workload affects the intensity of enzyme synthesis (Williams, Salmons, Newsholme, Kaufman, & Mellor, 1986). The following is believed to be at the basis of the enzyme adaptation:
- reduced content of ATP and/or of other high-energy phosphates in the cell,
- reduced O₂ pressure in the muscle tissue,
- increased sympathoadrenal stimulation of the muscle cell,
- Ca-induced release of diacylglycerol with ensuring activation of protein kinase C.

**Changes in the vascularization of the trained muscles**

Regular aerobic exercise leads to an almost two-fold increase of the number of capillaries per unit of muscle cross-sectional area which results in the increase of the blood flow rate through the working muscles. This can take as long a time as it takes for the enzyme adaptation to occur - for two months vascularization can increase by about 50%, sometimes even two- or threefold.

In a study on rats Dudley, Abraham and Terjung (1982) found that the effect exerted by aerobic exercises on muscles differs for the different types of muscle fibers. In fast oxidative glycolytic fibers (type IIa or FOG) the effect enhances with the increase of training intensity up to intensities equaling 80% of VO₂max. Fast glycolytic fibers (type IIb or FG) can be affected by regular exercises at intensity over 80% of VO₂max. The effect for the slow fibers of type I is better pronounced in higher intensity of training up to 80% of VO₂max. In intensities higher than this the effect is less pronounced.
Changes in the type characteristic of muscle fibers

There are basically two types of muscle fibers - type I (slow-twitch or ST) and type II (fast-twitch or FT). Type I muscle fibers have high oxidative capacity, that is, high activity of the oxidative enzymes. Type II fibers are characterized by relatively higher glycolytic capacity and lower resistance to fatigue than type I fibers. In submaximal exercises, the lactate produced in the muscles is oxidized by type I fibres but utilized by type II fibers for glycogen resynthesis. This difference in the metabolic “behavior” of the two types of muscle fibers is due to the relative difference in the activity of the glycolytic system in the cytosol and the oxidative enzyme system in the mitochondria as well as to the differences in the activity of the enzymes responsible for glycogen resynthesis. For example, in systematic endurance training in type I fibers the activity of the H-LDH (heart specific iso-enzyme) increases.

Fast-twitch fibers (type II) have a high degree of myosin-ATPase activity; they can rapidly release Ca++ and realize fast glycolysis. The slow-twitch fibers (type I) have a low myosin-ATPase activity, lower glycolytic capacity, greater amount of mitochondria and myoglobin and higher oxidative capacity. Type IIa fibers actually represent a transition type of fibers because although fast, they have a higher activity of the enzyme SDH (that is, they have aerobic capacity). Type IIb are typical fast-twitch fibers. There are also type IIc fibers which are few and undifferentiated.

While enzyme activity of the recruited muscles can change relatively rapidly as a result of regular training, the distribution of fibers within the active muscle probably remains constant. However, this is at present a controversial matter. Noakes (1992) reports in a study that the gastrocnemius muscle in long distance runners is composed of about 79% of type I fibers, in middle distance runners - of about 62%, and in untrained subjects - 58%.

Similar data are also reported by other authors, the differences in the fiber distribution being genetically determined according to Komi, Viitasalo, Havu, Thorstensson, Sjödin and Karlsson (1977) and Komi and Karlsson (1979) (Figure 4).

We should keep in mind that it is type I fibers (the slow-twitch fibers) that are initially activated in continuous submaximal exercises.

When, however, the energy stores in them are close to depletion, a larger number of fast-twitch fibers (type II) begins to be recruited, first type IIa, then type IIb (Saltin, 1981).

Some authors indicate that conversion from one type of muscles to another as a result of training is impossible to occur in humans (Gollnick, Armstrong, Saubert, Sembrowich, Shepherd, & Saltin, 1973; Saltin & Gollnick, 1983). Other researchers have opposite findings suggesting that with the metabolic and physiological potential of muscle fibers it is quite possible to underage transformation (Aitken, Bennet, & Thompson, 1989; Bell, MacDougall, Billter, & Howald, 1980; Pete & Vrbova, 1985; Tesch & Karlsson, 1985). There are also authors that regard as possible only the conversion of type IIb fibers to type IIa (Andersen & Henriksson, 1977; Jansson & Kaijser, 1977).

Changes associated with glucose utilization from muscle cells. The role of insulin.

It is well known that the transmembrane glucose transport is realized by facilitated diffusion in the muscle cells, the adipocytes and the fibroblasts. This is a passive process (glucose is taken up from a place with high concentration and transported to a place with low concentration) mediated by a carrier molecule which facilitates the glucose transport through the membrane. Therefore, in the course of transportation, the carrier will have a certain degree of saturation. In skeletal muscles the Michaelis constant (Km) for the
glucose transport from the external to the internal side of the cellular membrane is 5-10 mmol. Research has shown that this transport is stimulated by the increased contractility (leading to an elevated release of intracellular Ca++) and insulin. The direct Ca++ effect persists several hours after exercise while the effect from insulin lasts one or two days. It is interesting to note that the insulin effect applies to those group of muscle that are recruited in the training (Essen, Hagenfeldt & Kaijser, 1977). It has been found that the glucose transport in submaximal exercises is selectively increased in the slow-twitch muscle fibers (Ploug, Stallknecht, Pedersen, Kahn, Ohkuwa, Vinten, & Galbo, 1990). Rodnick, Reaven, Azhar, Goodman and Mondon (1990) have found that aerobic training leads to the increase of glucose transporters in the membrane of the slow-twitch muscle fibers. In already trained people the response to insulin is more markedly pronounced in this respect. Therefore, regular submaximal exercises cause the sensitivity of the muscle cells to insulin to increase.

Changes in muscle glycogen

In trained people at rest, the glycogen concentration in their muscles is higher than that of untrained subjects (140 - 230 mmol/g ww vs. 70 -110 mmol/g ww). Identical relationship is found in comparing a trained with an untrained extremity of one and the same individual. In the process in detraining the glycogen content becomes equal in both extremities. Mikines, Sonne, Tronier and Galbo (1989) have found that in trained subjects the activity of the enzyme glycogen synthase in the muscle cells is high, while Bogardus, Ravussin, Robbins, Wolfe, Horton and Sims (1984) and Devlin and Horton (1985) have demonstrated that this enzyme enhances its activity under the effect of insulin. Therefore, the repletion of glycogen stores after a submaximal exercise is associated with an increased sensitivity of the muscle cell to insulin. According to Gulve, Cartee, Zierath, Corpus and Holloszy (1990) this intensifies the process of glucose uptake into the cell.

When the exercise performed at 60 to 80% of the maximal oxygen consumption continues for more than 60 minutes, the initial glycogen concentration in muscles is undoubtedly crucial for the submaximal endurance. It is directly proportional to the degree of muscle glycogen depletion (ΔG) in the process of the performed work. When an alternative substrate for aerobic oxidation (fatty acids) is available we have a “glycogen sparing effect” which occurs during exercise which leads to a delay of fatigue and eventually to an increase of the submaximal endurance. The glycogen sparing effect usually occurs after 6 weeks of regular aerobic training and is mainly caused by enzyme adaptation in the beta oxidation pathway (CPT). The capillarization additionally facilitates this process. Thus it turns out that trained individuals can utilize fatty acids in submaximal exercises as a source of energy better than untrained subjects can which leads to a preservation of the supplies of glycogen and hence to greater endurance in an identical intensity of performed work. The respiratory exchange ratio (RER) in trained people is lower than that in untrained people; they have also a higher degree of utilization of muscle triglycerides (Figure 5).

In a previous study on 233 rowers training for more than 3 years we found that the total serum cholesterol (means ± SD) was lower than that of age-matched non-trained controls (3.90±0.76 versus 4.31±0.76 mmol/l; p < 0.001); no differences between the serum triglycerides of these groups were found (Tarakov, Boyadjiev, & Georgieva, 2000).

In summary, the mechanisms leading to an enhanced fatty oxidation due to training are:

- increased lipolysis of available triglycerides in the muscles,
- enhanced transport of fatty acids in the mitochondria,
- increased number of mitochondria in the muscle cells,
- increased activity of the mitochondrial enzymes in beta-oxidation (3-HAD and CPT),
- increased sympathoadrenal activity.

**Immunity**

It is well known that physical activity affects the body’s immune system. Submaximal exercises are considered to stimulate both the nonspecific and the specific immunity which reduces the risk of inflammatory diseases. There have been studies, however, which demonstrate that immunodepression can be caused by intensive exercise training and accompanied by an increased morbidity rate of infectious diseases, especially acute respiratory infections (Pedersen, Rohde, & Zacho, 1996; Pyne & Gleeson, 1998).

It is widely thought that a single-bout exercise can reduce immune reactivity, while a regular submaximal exercise training going on for many years can have a heterogeneous effect on the parameters of the immunity and the systemic inflammatory response. In a study including 143 rowers from sports schools in Bulgaria (age 14.01±0.06 yrs; 56.35±0.49 kg; 3.44±0.06 yrs of training, training 5 days per week, two times a day) and 61 untrained controls (age 14.12±0.09 yrs; 57.01±0.23 kg) we found that the mean serum concentration of Ig A in the athletes was by 47.5% higher (р < 0.001), of IgM - by 22.0% lower (р<0.001), and of IgG - by 10.7% higher (р<0.05) than those of the untrained individuals (Table 1, data presented as mean ± SEM) (Taralov, Boyadjiev, & Georgieva, 1999).

**Adaptations in the oxygen transport system - red blood**

Biancotti, Caropreso, Di Vincenzo, Ganzit & Grisaudo. (1982) and Hasibeder, Schobesbarger and Mairbaur (1987) demonstrated that intense training, which includes also submaximal training, could lead to a “suboptimal” hematologic status of athletes, with evidence even in some cases of sports anemia. In a previous study we examined 230 rowers (122 boys and 108 girls) of sports schools in Bulgaria (age 14.01±0.06 yrs; 56.24±0.52 kg; 3.52±0.07 yrs of training, training 5 days per week, two times a day) and 350 untrained controls (168 boys and 182 girls – 14.58±0.09 yrs, 57.75±0.67 kg). The findings of the study indicate that with the number of years of increased training the athletes undergo certain adaptations which create the conditions for a greater economy and effectiveness of metabolism.

**Table 1. Serum immunoglobulin profile in submaximal trained and non-trained pubescent subjects (mean±SEM)**

<table>
<thead>
<tr>
<th>GROUP</th>
<th>VARIABLE</th>
<th>IgA (g/l)</th>
<th>IgM (g/l)</th>
<th>IgG (g/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. rowers</td>
<td></td>
<td>2.05±0.07</td>
<td>0.96±0.03</td>
<td>12.24±0.32</td>
</tr>
<tr>
<td>2. controls</td>
<td></td>
<td>1.39±0.10</td>
<td>1.23±0.09</td>
<td>11.06±0.44</td>
</tr>
<tr>
<td>P1-P2</td>
<td></td>
<td>0.001</td>
<td>0.001</td>
<td>0.05</td>
</tr>
</tbody>
</table>

**Table 2. Red blood cell variables in submaximal trained boys (rowers) compared with non-trained controls (mean±SEM)**

<table>
<thead>
<tr>
<th>GROUP</th>
<th>VARIABLE</th>
<th>RBC (x 10^12/l)</th>
<th>Hct (l/l)</th>
<th>Hb (g/l)</th>
<th>MCV (fl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. rowers</td>
<td></td>
<td>4.66±0.03</td>
<td>0.400±0.003</td>
<td>136.21±0.94</td>
<td>85.59±0.39</td>
</tr>
<tr>
<td>2. controls</td>
<td></td>
<td>5.01±0.03</td>
<td>0.425±0.003</td>
<td>146.16±0.82</td>
<td>84.94±0.40</td>
</tr>
<tr>
<td>P1-P2</td>
<td></td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
<td>NS</td>
</tr>
</tbody>
</table>

RBC, red blood cell count; Hct - packed cell volume; Hb - haemoglobin; MCV - mean corpuscular volume.

**Table 3. Red blood cell variables in submaximal trained girls (rowers) compared with non-trained controls (mean±SEM)**

<table>
<thead>
<tr>
<th>GROUP</th>
<th>VARIABLE</th>
<th>RBC (x 10^12/l)</th>
<th>Hct (l/l)</th>
<th>Hb (g/l)</th>
<th>MCV (fl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. rowers</td>
<td></td>
<td>4.32±0.04</td>
<td>0.384±0.003</td>
<td>124.27±0.93</td>
<td>86.00±0.45</td>
</tr>
<tr>
<td>2. controls</td>
<td></td>
<td>4.51±0.03</td>
<td>0.384±0.002</td>
<td>134.26±0.64</td>
<td>85.51±0.44</td>
</tr>
<tr>
<td>P1-P2</td>
<td></td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
<td>NS</td>
</tr>
</tbody>
</table>

RBC, red blood cell count; Hct - packed cell volume; Hb - haemoglobin; MCV - mean corpuscular volume.
Adaptation in the blood coagulation system

Physical exercise and training induce changes in the hemostasis of healthy people. A single bout of exercise usually causes transient activation of the coagulation system indicated by a shortening of the activated partial thromboplastin time (APTT) (Bartsch, Haeberli, & Straub, 1990; El-Sayed, Sale, Jones, & Chester, 2000; Prisco et al., 1998) or by activation of the fibrinolytic mechanisms (Wallen, Goodall, & Hjemdahl, 1999). There are few studies on the long-term effects of doing different types of exercises on coagulation. We conducted a study of 37 active training athletes exposed to submaximal workloads (age 15.49±2.02 yrs, 4.83±2.20 yrs of training); changes in the parameters of aerobic work capacity

Aerobic work capacity depends on the ability of the body to provide energy for muscle activity through the aerobic mechanisms of oxidation; it is characterized by:

- aerobic power (oxygen consumption under physical stress or VO2max),
- submaximal endurance.

Although it is a very important parameter, VO2max should not be considered in isolation when assessing aerobic working capacity. It is quite possible, for instance, for two elite long distance runners to have an identical performance, with the one having high VO2max and relatively poorer utilization of oxygen, and the other - lower VO2max but more economical use of oxygen under physical stress. It has been found that athletes showing similar results can have rather different VO2max. Therefore, important as this parameter can be, it is not crucial for a sports person’s achievement. The factor that matters here is what percentage of the athlete’s own VO2max can be used in performing specific physical work. Daniels (1974) and Costill (1979) established independently that different athletes have an identical running speed in a competition but at a different percentage of their maximal oxygen consumption. Better athletes run using smaller amounts of oxygen, that is, they have greater running economy.

VO2max reflects to a large extent the oxygen transport and oxygen delivery to working muscles. While in untrained subjects it can be in

Table 4. Haemocoagulation parameters in strength and endurance athletes compared with non-trained controls (mean±SD)

<table>
<thead>
<tr>
<th>GROUP</th>
<th>VARIABLE</th>
<th>PLT (x 10^9/l)</th>
<th>FGN (g/l)</th>
<th>pT (%)</th>
<th>APTT (s)</th>
<th>TT (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. strength (n=46)</td>
<td>PLT</td>
<td>278.8±52.9</td>
<td>2.79±0.69</td>
<td>86.12±10.47</td>
<td>31.25±2.21</td>
<td>20.63±1.24</td>
</tr>
<tr>
<td>2. endurance (n=37)</td>
<td>PLT</td>
<td>256.9±44.9</td>
<td>2.65±0.81</td>
<td>81.00±9.50</td>
<td>31.46±2.76</td>
<td>21.30±2.75</td>
</tr>
<tr>
<td>3. controls (n=67)</td>
<td>PLT</td>
<td>270.6±49.7</td>
<td>2.47±0.58</td>
<td>80.78±10.09</td>
<td>31.96±2.69</td>
<td>21.53±1.30</td>
</tr>
<tr>
<td>P1-P2</td>
<td>PLT</td>
<td>0.05</td>
<td>NS</td>
<td>0.05</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>P1-P3</td>
<td>PLT</td>
<td>NS</td>
<td>0.02</td>
<td>0.02</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>P2-P3</td>
<td>PLT</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>0.001</td>
</tr>
</tbody>
</table>

PLT - platelet count; FGN - fibrinogen; pT - prothrombin; APTT - activated partial thromboplastin time; TT - thromboplastin time.

when the findings from these athletes were compared with those of 67 age-matched controls (15.81±2.73 yrs) no differences were found between the basal values of the major coagulation parameters - number of thrombocytes (PLT), fibrinogen (FGN), prothrombine time (pT), activated partial thromboplastin time (APTT) and thromboplastin time (TT). Unlike these subjects, the athletes doing anaerobic sports for a long time show the parameters suggestive of activation of the blood coagulation mechanisms which is persistently more intense, a characteristic consequence in untrained people exposed to acute workload (Table 4) (Boyadjiev & Taralov, 2002).

Changes in the parameters of aerobic work capacity

when the findings from these athletes were compared with those of 67 age-matched controls (15.81±2.73 yrs) no differences were found between the basal values of the major coagulation parameters - number of thrombocytes (PLT), fibrinogen (FGN), prothrombine time (pT), activated partial thromboplastin time (APTT) and thromboplastin time (TT). Unlike these subjects, the athletes doing anaerobic sports for a long time show the parameters suggestive of activation of the blood coagulation mechanisms which is persistently more intense, a characteristic consequence in untrained people exposed to acute workload (Table 4) (Boyadjiev & Taralov, 2002).
What changes take place in aerobic capacity with age have been shown in the longitudinal studies of Trappe, Costill, Vukovich, Jones and Melham (1996) on a contingent of elite sports people. The survey started in the 1960s and lasted till the last decade of the millenium. A marked reduction of VO\textsubscript{2max} (over a period of 20 years) was found in a group of athletes that had stopped training - decrease by 18% in the absolute maximal oxygen consumption and by 34% in the relative consumption because of weight gain. In those that kept on training this reduction was 9% and 14%, respectively. Biopsy specimens were taken from m. gastrocnemius in all subjects. The findings showed that the former active athletes who went on training intensively had high activity of the mitochondrial enzymes CS and SDH while those who had ceased to train had a considerable decline of the activity of the oxidative enzymes which was most probably the cause for the pronounced reduction of VO\textsubscript{2max} in them.

It is important to bear in mind the fact the maximal oxygen consumption is dependent on two systems:

- an "external" system of oxygen delivery which includes the cardiorespiratory system and blood - it delivers oxygen from the air to the muscle cell,
- an internal (mitochondrial) oxygen utilization system realizing the aerobic production of energy.

What is the restricting factor for VO\textsubscript{2max}? This issue is quite controversial among sports physiologists. It is believed that muscle capacity for oxygen utilization is several times as high as (or at least it acquires this characteristic as a result of aerobic training) the capacity of the cardiorespiratory system to meet its demands. For this reason the changes occurring in the cardiovascular system and especially the increase of the stroke volume and the cardiac output are very important for the enhancement of the aerobic capacity of the body as a result of regular submaximal exercise.

**Conclusions**

The adaptational changes in the body we and other authors have found that result from submaximal exercises can be summarized as follows:

1. Key oxidative enzymes in the carbohydrates breakdown pathway (HK and CS), in the fat breakdown pathway (3-HAD and CPT) and in the respiratory chain (CCO) increase their activity.
2. A selective vascularization in the muscles containing predominantly oxidative muscle fibers is induced.
3. A type change of the muscle fibers is possible to occur (including changes from type II to type I) in the submaximally recruited muscle groups.
4. Glucose transport in the cells of the recruited muscles is selectively increased with direct insulin mediation.
5. A metabolic adaptation occurs which consists of a shift in the energy substrates from carbohydrates to fats and a subsequent glycogen sparing effect which guarantees a delay of fatigue and an increase of the work capacity. The serum level of the total cholesterol decreases permanently.
6. Permanent changes occur in the humoral immunity expressed as an increase of the level of serum IgA and IgG.
7. The system of oxygen transport in blood adapts to a more economic operation. The coagulation capacity of blood does not differ from that of untrained subjects.
8. Eventually, adaptation of the external system of oxygen delivery and the mitochondrial system of oxygen utilization occurs which has as a result an insignificant increase of the maximal oxygen consumption and a significant increase of the oxygen running economy.
References


Boyadjiev, N. (1996a). Increasing of the aerobic capacity of the organism as a result of submaximal training and dietary influences. (Unpublished doctoral thesis, Faculty of Medicine, Medical University, Plovdiv, Bulgaria).


Sažetak

Submaksimalan trening može potaknuti adaptacijske promjene skeletnih mišića, kardiovaskularnog sustava, respiratornog sustava, endokrinog sustava itd. Poznavanje obilježja i veličine adaptacije, poznavanje čimbenika koji je uzrokovali, kao i svijest o važnosti adaptacije za poboljšanje fizičkih sposobnosti neizostavni su za oblikovanje optimalnog trenažnog programa te, uz adekvatan proces oporavka, za postizanje maksimalnog pozitivnog učinka uz minimalan negativni učinak na zdravlje sportaša.

Adaptacija, kao posljedica redovitog aerobnog treninga, može uključivati adaptaciju enzimskih sustava mišića, promjene u vaskularizaciji i tipu mišićnih vlakana, metaboličke promjene treniranih mišića i mišićnih skupina povezane s korištenjem glukoze i uporabom glikogena i masti kao izvora energije, promjene u krvi, immunološki i koagulacijski status, kao i promjene parametara aerobne radne sposobnosti organizma.

Adaptacijske promjene u tijelu, za koje smo i mi i drugi autori utvrdili da su posljedica submaksimalnih vježbi, moguće je sažeti ovako:

1. Pojačava se aktivnost ključnih oksidacijskih enzima u procesu razgradnje ugljikohidrata (HK i CS), u procesu razgradnje masti (3-HAD i CPT) u respiracijskom lancu (CCO).

2. Inducirana je selektivna vaskularizacija u mišićima koji pretežno sadrže oksidacijske mišićna vlakna.

3. Kod submaksimalno aktiviranih mišićnih skupina može nastati promjena tipa mišićnih vlakana (uključujući prelazak iz tipa II u tip I).

4. Direktnim djelovanjem inzulina selektivno se povećava prijenos glukoze u stanice aktiviranih mišića.

5. Dolazi do metaboličke adaptacije koja se očituje u premještanju energetskih procesa s ugljihidrata na masti s posljedicom uštede glikogena, što jamči odgodu pojave zamora i porast radne sposobnosti. Nastupa i trajni pad razine ukupnog kolesterola u serumu.

6. Trajne promjene humoralnog imuniteta izražene su u vidu porasta razine IgA i IgG u serumu.

7. Sustav prijenosa kisika krvlju prilagođava se u smislu povećanja ekonomičnosti. Koagulacijski kapacitet krvi ne razlikuje se od istog kapaciteta netreniranih osoba.

8. Konačno, prisutna je i adaptacija vanjskog sustava za dopremu kisika i mitohondrijskog sustava iskorištenja kisika, što za posljedicu ima neznačajan porast maksimalnog primitka kisika i značajno poboljšanje ekonomike potrošnje kisika (energetske učinkovitosti).

Submitted: September 1, 2002
Accepted: April 20, 2004

Correspondence to:
Prof. Nickolay Boyadjiev, MD, PhD
Department of Physiology,
Faculty of Medicine,
Medical University,
15-a, V. Aprilov Blvd.,
4000 Plovdiv, Bulgaria
Phone: + 359 (88) 8802 590
Fax: + 359 (32) 644 675
E-mail: boyad@plovdiv.techno-link.com