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REVIEW: ADAPTATION TO SUBMAXIMAL PHYSICAL TRAINING

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SUMMARY

Submaximal training can induce adaptive changes in the skeletal muscles, cardiovascular system, respiratory system, endocrine system, and the like. Knowledge of the adaptation's signs and magnitude, the factors causing it, as well as awareness of the significance of the adaptation for the improvement of physical capacity to exercise is essential to developing an optimal training programme and by an adequate recovery process to achieving maximum positive effect at minimum negative effect on the athlete's health. Body adaptation resulting from regular aerobic training can involve enhancement of muscle enzyme systems, changes in muscle fibre types and vascularisation, metabolic changes in the trained muscle groups related to glucose, glycogen and fat utilisation as sources of energy. Furthermore, changes in the blood, the immune and coagulation status of the body accompany changes in parameters related to the 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 palmitoyl-transferase (CPT) and in the respiratory chain citochrome c oxidase (CCO). Submaximal training induces vascularisation in the muscles containing predominantly oxidative muscle fibres, and also enhances fibre type transformation. 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 physical working

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capacity. Permanent changes occur in humoral immunity, evidenced by the increase of serum IgA and IgG concentrations. The blood oxygen transport system adjusts itself to a more economical operation. As a final result, both the external oxygen delivery and the mitochondrial oxygen utilisation systems undergo adaptation leading to a moderate increase in maximal oxygen consumption, but to a greater increase in running economy.

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

ÖZET

DERLEME: SUBMAKSİMAL FİZİKSEL ANTRENMANA UYUM

Submaksimal antrenman iskelet kasları ile kalp-dolaşım, solunum, endokrin ve diğer sistemlerde uyumsal değişikliklere neden olur. Uyumun işaretleri, boyutu ve ilgili faktörlerin yanı sıra fiziksel egzersiz kapasitesinin geliştirilmesindeki öneminin kavranması optimum bir antrenman programının hazırlanması ve uygun bir toparlanma süreci tanınarak sporcunun sağlığı minimum olumsuzlukla karşılaşırken maksimum pozitif etkiyi gerçekleştirmek açısından elzemdir. Düzenli aerobik antrenmana uyum; kas enzim sistemlerinin gelişmesini, kas lifi tipi ve vaskülarizasyonunda değişimi, antrene kasta enerji kaynağı olarak glikoz, glikojen ve yağların kullanılmasına ilişkin metabolik değişiklikleri içerebilir. Ayrıca, organizmanın koagülasyon ve immün statüsü ile kandaki değişiklikler aerobik iş kapasitesine ilişkin parametrelerdeki değişikliklere eşlik eder. Düzenli submaksimal egzersizin metabolik yıkılım yollarında anahtar oksidatif enzimlerden karbonhidratlar için heksokinaz (HK) ve sitrat sentaz (CS)'ın, yağlarda ise 3-hidroksiacil-KoA dehidrogenaz (3-HAD) ve karnitin palmitoyltransferaz (CPT)'ın yanı sıra solunum zincirinde de sitokrom c oksidaz (CCO)'ın aktivitelerini arttırdığı saptanmıştır. Bu tip antrenmanla çoğunlukla oksidatif kas lifleri içeren kaslarda vaskülarizasyonu uyarılmakta, hatta kas lifi tiplerinde değişimler gerçekleşebilmektedir. Devreye sokulan kas hücrelerinde insüline bağlı glikoz transportu seçimli olarak artmaktadır. Organizmanın enerji kaynağı olarak karbonhidratlardan yağlara yönelmesi şeklinde metabolik bir uyum gerçekleşmekte, bunun sonucu olarak glikojen saklanımı fiziksel iş kapasitesini arttırmaktadır. Humoral immünitede serum IgA ve IgG konsantrasyonlarındaki artışla kanıtlanan kalıcı değişiklikler gerçekleşir. Oksijen transport sistemi daha ekonomik bir çalışmaya uyum gösterir. Son olarak, hem dış solunumla oksijen temini, hem de iç solunumda mitokondrial oksijen kullanımı sistemleri adaptasyona uğrayarak maksimum

oksijen kullanım kapasitesinde hafif bir artışa, buna karşın koşu ekonomisinde yüksek bir artışa yol açar.

Anahtar sözcükler: *Submaksimal egzersiz, adaptasyon, sporcu, aerobik iş kapasitesi*

Training exercise, performed properly considering methodology and conducted regularly at submaximal levels, is a major approach in establishing structural and functional grounds to achieve high aerobic working capacity. This type of training causes adaptive changes in the skeletal muscles, in the cardiovascular, respiratory, and endocrine systems which lead to greater aerobic working capacity and hence to improvement of sports results (7). To understand these changes, their scope, and the direct factors causing them gives a powerful means to obtain maximum results, through an optimal training plan and adequate recovery under the guidance of a trainer and medical staff, with minimum adverse effect on the health of athletes.

1. Enzyme adaptation in muscles

Endurance training induces distinct adaptation in the recruited muscles. Muscle tissue can be evaluated both morphologically and biochemically by using biopsy techniques. It is well known that the major pathways involved in energy supply to the working muscle 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 enzymes in question. 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 remaining practically unchanged (25).

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 changes in the quantity (acitivity) 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).

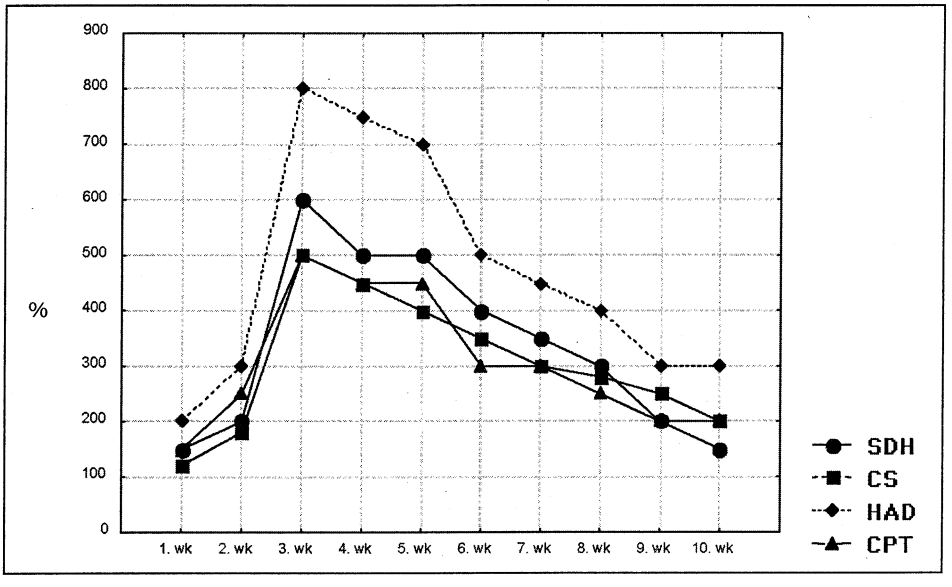

Figure 1. Enzyme activity changes (% of initial level) as a result of muscle electrostimulation in experimental rabbits.

Choosing a group of enzymes to use in the analysis depends largely on the biochemical techniques mastered for their detection in muscle homogenates; basically, the following enzymes are used:

- Hexokinase (HK), phosphofructokinase (PFK) and lactate dehydrogenase (LDH) in the assessment of the glycolytic pathway,
- 3-hydroxyacyl-CoA dehydrogenase (3-HAD) and carnitine palmitoyltransferase (CPT) in the analysis of beta-oxidation of fatty acids,
- Citrate synthase (CS) and oxoglutarate dehydrogenase (OGDH) in the analysis of the 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 three to five

weeks (8). After cessation of training, their activity drops to baseline levels after five to six weeks.

Though studies on muscle tissue enzymes of experimental animals had been performed by 1970, human studies of biopsy material had not been performed until then (24,39). From 1979 to 1980, the techniques for obtaining biopsy material as well as the methods for its processing (buffering, organelle disintegration, etc) were perfected. The changes are differently expressed in experimental animals and humans, but in all cases they are similar (Figure 2 and Figure 3).

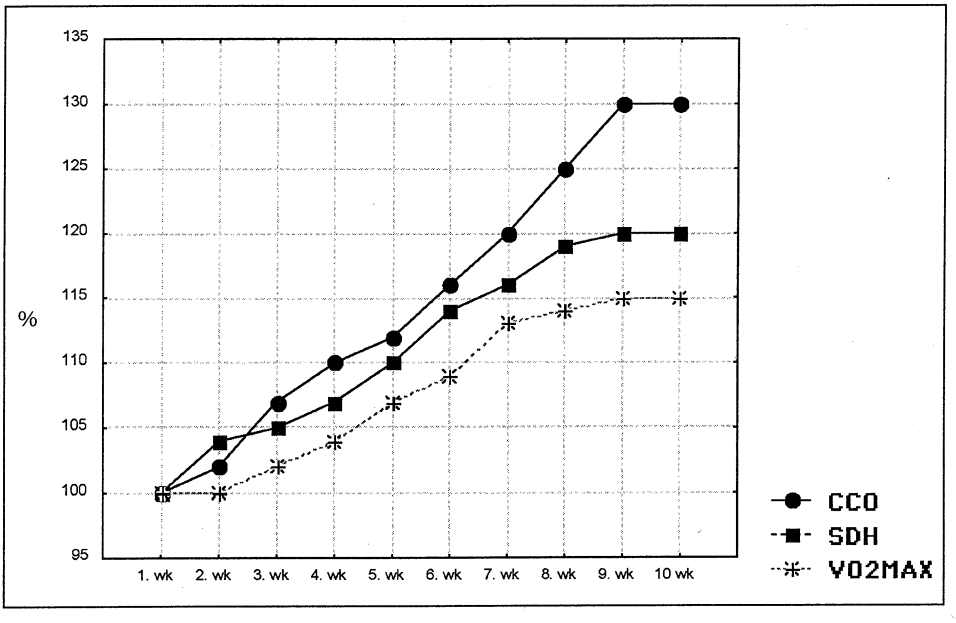

Figure 2. Enzyme adaptations as a result of submaximal training in m. vastus lateralis in humans.

The explanation that enzyme adaptation occurs as a result of submaximal training can be accounted for by the fact that the enzymes have a specific "life cycle", and accordingly, each has a specific half-life, which varies from a day (for the glycolytic enzymes) to a week (for the mitochondrial enzymes). The cellular content (metabolic activity) of the enzyme is always a result of the balance between its synthesis and its disintegration. Submaximal workload affects the intensity of enzyme synthesis (41). The following is believed to be at the basis of enzyme adaptation:

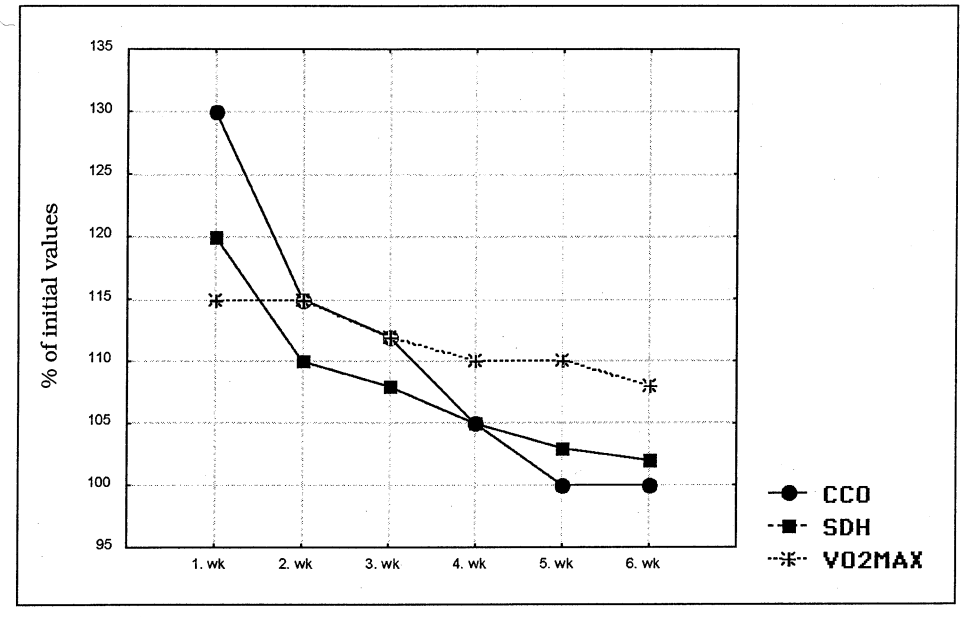

Figure 3. Changes in the activity of CCO, SDH and maximal oxygen uptake after ceasing training in humans.

- Reduced content of ATP and/or of other high-energy phosphates in the cell,
- Reduced O_2 pressure in the muscle tissue,
- Increased sympathoadrenal stimulation of the muscle cell,
- Ca-induced release of diacylglycerol with ensuing activation of protein kinase C.

2. Changes in the vascularisation of trained muscles

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

The effect exerted by aerobic exercises on muscles differs for the different types of muscle fibre (14). In fast oxidative glycolytic fibres (type IIa or FOG) the effect enhances with the increase of training intensity up to intensities reaching 80% VO_2max. Fast glycolytic fibres (type IIb or

FG) can be affected by regular exercises of intensity over 80% VO_2max. The effect on the slow fibres of type I is more pronounced in higher intensity of training up to 80% VO_2max. In intensities exceeding this level, the effect is less pronounced.

3. Changes in the type characteristics of muscle fibres

There are basically two types of muscle fibres - type I (slow-twitch or ST) and type II (fast-twitch or FT). Type I muscle fibres have high oxidative capacity, that is, high activity of the oxidative enzymes. Type II fibres are characterised with relatively higher glycolytic capacity and lower resistance to fatigue than type I fibres. In submaximal exercises, the lactate produced in the muscles is oxidised by type I fibres but utilised by type II fibres for glycogen resynthesis. This difference in the metabolic "behaviour" of the two types of muscle fibres 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, with systematic endurance training the activity of the H-LDH (heart specific isoenzyme) in type I fibres increases.

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

While enzyme activity of recruited muscles can change relatively rapidly as a result of regular training, the distribution of fibres within the active muscle probably remains constant. However, this is at present a controversial matter. Noakes (1992) reported that the gastrocnemius muscle is composed of about 79% of type I fibres in long distance runners, of about 62% in middle distance runners, and of about 58% in untrained subjects (26). Other authors also report similar data. Differences in fibre distribution is genetically determined according to Komi et al. (1997) and Komi & Karlsson (1979) - Figure 4 (21,22).

We should keep in mind that type I fibres (the slow-twitch fibres) are initially activated in continuous submaximal exercises. When, however,

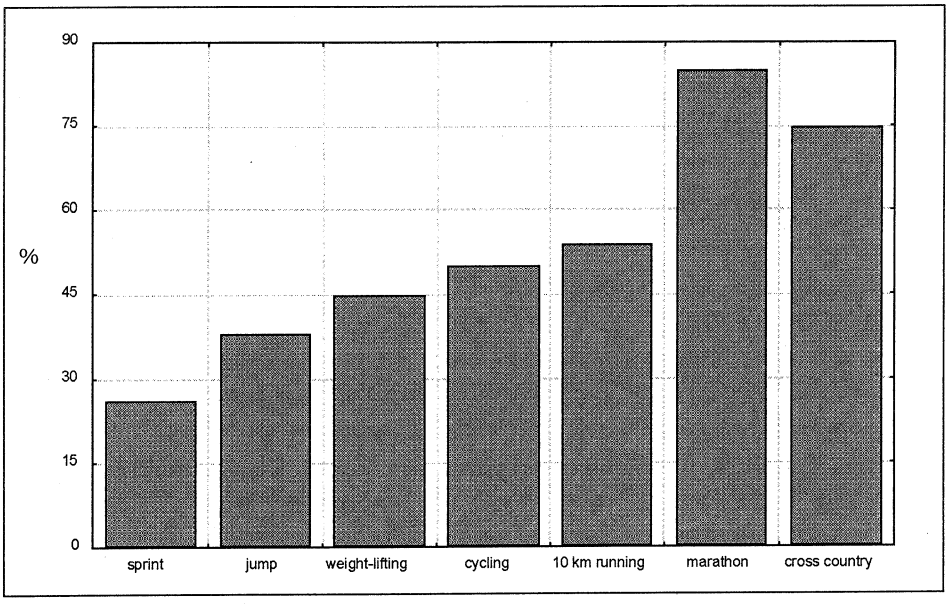

Figure 4. Muscle fibers of type I in m. gastrocnemius (%) in athletes of different sports.

their energy stores are close to depletion, a larger number of fast-twitch fibres (type II) begins to be recruited, starting with type IIa, then type IIb (33).

Some authors indicate that conversion from one type of muscles to another as a result of training is impossible to occur in humans (17,34). Other researchers have opposite findings suggesting that the metabolic and physiologic potential of muscle fibres permits such transformation (1,4,28,37). There are also authors that consider as possible only the conversion of type IIb fibres to type IIa (2,20).

4. Changes associated with glucose utilisation in muscle cells. Role of insulin

It is well known that transmembrane glucose transport is realised by facilitated diffusion in the muscle cells, adipocytes and fibroblasts. This is a passive process (glucose is taken up from a high concentration medium and is transported to a low concentration one) mediated by a carrier molecule which facilitates 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 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 increased contractility (leading to elevated release of intracellular Ca^{++}) and insulin secretion. The direct effect of Ca^{++} persists several hours following exercise, and that of insulin lasts one or two days. It is interesting to note that the insulin effect applies to the group of muscles that are recruited in the training (16). It has been found that glucose transport during submaximal exercise is selectively increased in the slow-twitch muscle fibres (29). Rodnick et al. (1990) have found that aerobic training leads to the increase of glucose transporters in the membrane of slow-twitch muscle fibres (32). In already trained people, the response to insulin is markedly more pronounced in this respect. Therefore, regular submaximal exercises cause the sensitivity of muscle cells to insulin to increase.

5. Changes in muscle glycogen

In trained people at rest, muscle glycogen concentration is higher compared with untrained subjects (140 - 230 mmol/g ww vs 70 -110 mmol/g ww). Identical correlation is found when comparing the trained and untrained extremities of the same individual. In the detraining process, the glycogen content becomes equal in both extremities. Mikines et al. (1984) have found that muscle glycogen synthase activity is high in trained subjects, while Bogardus et al. (1984) and Devlin & Horton (1985) have demonstrated that the activity of this enzyme is enhanced under the effect of insulin (6,13,23). Therefore, the repletion of glycogen stores following submaximal exercise is associated with an increased sensitivity of the muscle cell to insulin. According to Gulve et al. (1990), this intensifies the process of glucose uptake into the cell (18).

When exercise performed at 60 to 80% of the maximal oxygen consumption lasts for more than 60 minutes, the initial glycogen concentration in the muscles is undoubtedly crucial for submaximal endurance (which is an element of aerobic working capacity of the body). The rapidity with which fatigue is felt is the major limiting factor for submaximal endurance. This is directly proportional to the degree of muscle glycogen depletion (DG) in the process of 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 submaximal endurance. The glycogen sparing effect usually occurs after six weeks of regular aerobic training and is mainly caused by enzyme (CPT) adaptation

in the beta oxidation pathway. Increased capillarisation additionally facilitates this process. Thus it turns out that trained individuals can better utilise fatty acids during submaximal exercise as a source of energy than untrained subjects, which leads to preservation of glycogen supply and hence to greater endurance in identical work loads. The respiratory exchange ratio (RER) in trained people is lower than that in untrained people; the former also having a higher degree of muscle triglyceride utilisation (Fig. 5).

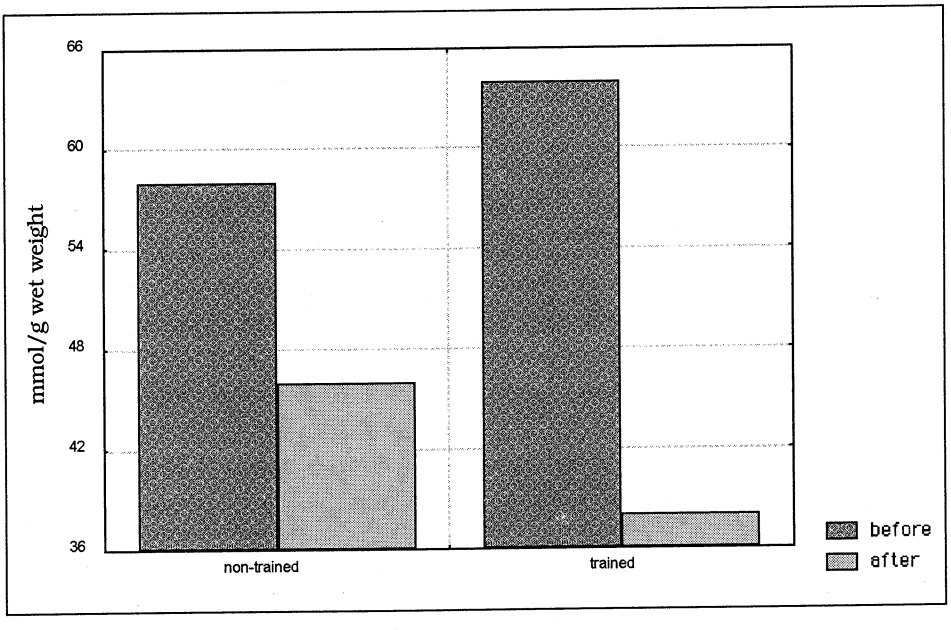

Figure 5. Muscle triglycerides before and after workload (64% of VO_2max) in the trained and non-trained state.

In a previous study on 233 rowers training for more than three years we found that total serum cholesterol levels (means ± SD) were 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 triglyceride levels of these groups were observed (36).

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 into the mitochondria,
- Increased number of mitochondria in the muscle cells,

- Increased activity of the mitochondrial enzymes of beta-oxidation (3-HAD and CPT),
- Increased sympathoadrenal activity.

6. Immunity

It is well known that physical activity affects the body immune system. Submaximal exercises are considered to stimulate both specific and non-specific immunity, which reduces the risk of inflammatory diseases. There are studies, however, which demonstrate that immuno-suppression can be caused by intensive exercise training and is accompanied by increased morbidity of infectious diseases, especially acute respiratory infections (27,31).

It is widely thought that a single-bout of exercise can reduce immune reactivity, while keeping regular submaximal exercise training habits for many years can have a heterogeneous effect on immunity parameters and systemic inflammatory response. In a study including 143 rowers from sports schools in Bulgaria (age 14.0 ± 0.1 yrs; 56.4 ± 0.5 kg; 3.44 ± 0.06 yrs of training, training five days per week, two times a day) and 61 untrained controls (age 14.1 ± 0.1 yrs; 57.0 ± 0.2 kg), we found that the mean serum IgA concentration in athletes was higher by 47.5% ($p<0.001$), that of serum IgM was lower by 22.0% ($p<0.001$), and that of serum IgG was higher by 10.7% ($p<0.05$) than the respective means of untrained individuals (Table 1) (35).

Table 1. Serum immunoglobulin profile in submaximally trained and untrained pubescent subjects (mean ± SEM).

Variable Group	IgA (g/l)	IgM (g/l)	IgG (g/l)
1. Rowers (n=143)	2.05 ± 0.07	0.96 ± 0.03	12.2 ± 0.3
2. Controls (n=61)	1.39 ± 0.10	1.23 ± 0.09	11.1 ± 0.4
P1-P2	0.001	0.001	0.05

7. Adaptations in the oxygen transport system - red blood

Biancotti et al. (1982) and Hasibeder et al. (1987) demonstrated that intense training, which also includes submaximal training, could lead to a "suboptimal" hematologic status in athletes, with even some cases of sports anaemia (5,19). In a previous study we examined 230 rowers (122 boys and 108 girls) of sports schools in Bulgaria (age 14.0 ± 0.1 yrs; 56.2 ± 0.5 kg;

3.52 ± 0.07 yrs of training, training five days per week, two times a day) and 350 untrained controls (168 boys and 182 girls, 14.6 ± 0.1 yrs, 57.8 ± 0.7 kg). The findings of the study indicate that with increasing number of training years, athletes undergo certain adaptations that create conditions for greater economy and efficiency of the oxygen transport with blood. Smaller number of red blood cells (RBC), smaller amount of haemoglobin (Hb) and lower haematocrit (Hct) in peripheral blood meets the increased demands of athletes practising submaximal exercises. For boys, these parameters are respectively lower by 7.5%, 7.5% and 6.2% than the corresponding figures in untrained subjects ($p<0.001$). For girls, they are lower by 4.5%, 8.0% and 5.5%, respectively ($p<0.001$). The mean corpuscular volume (MCV) in both groups and both genders was identical (Tables 2 and 3) (9).

Table 2. Red blood cell variables in submaximally trained boys (rowers) compared with untrained controls (mean ± SEM).

Variable Group	RBC (x 10^{12}/l)	Hct (l/l)	Hb (g/l)	MCV (fl)
1. Rowers (n=122)	4.66 ± 0.03	0.400 ± 0.003	136.2 ± 0.9	85.6 ± 0.4
2. Controls (n=168)	5.01 ± 0.03	0.425 ± 0.003	146.2 ± 0.8	84.9 ± 0.4
P1-P2	0.001	0.001	0.001	NS

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

Table 3. Red blood cell variables in submaximally trained girls (rowers) compared with unrained controls (mean ± SEM).

Variable Group	RBC (x 10^{12}/l)	Hct (l/l)	Hb (g/l)	MCV (fl)
1. Rowers (n=108)	4.32 ± 0.04	0.364 ± 0.003	124.3 ± 0.9	86.0 ± 0.5
2. Controls (n=182)	4.51 ± 0.03	0.384 ± 0.002	134.3 ± 0.6	85.5 ± 0.4
P1-P2	0.001	0.001	0.001	NS

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

8. Adaptation in the blood coagulation system

Physical exercise and training induce changes in the haemostasis of healthy people. A single bout of exercise usually causes transitory activation of the coagulation system indicated by shortening of the activated partial thromboplastin time (aPTT) (3,15,30) or by activation of the fibrinolytic mechanisms (40). There are few studies on the long-term practice of

different types of exercise on coagulation. We conducted a study on 37 actively training athletes exposed to submaximal workloads (age 15.5 ± 2.0 yrs, 4.83 ± 2.20 yrs of training). When findings from these athletes were compared with those of 67 age-matched controls (15.8 ± 2.7 yrs) no differences were found between the basal values of major coagulation parameters, namely the number of thrombocytes (PLT), fibrinogen (FGN), prothrombine time (pT), activated partial thromboplastin time (APTT) and thromboplastin time (TT). Unlike these subjects, the athletes training for anaerobic sports for a long time reveal parameters suggestive of blood coagulation mechanism activation, which is persistently more intense, a characteristic consequence observed in untrained people exposed to acute work loads (Table 4) (10).

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

Variable Group	PLT (x 10^9/l)	FGN (g/l)	pT (%)	APTT (sec)	TT (sec)
1. Strength (n=46)	279 ± 53	2.79 ± 0.69	86.1 ± 10.5	31.3 ± 2.2	20.6 ± 1.2
2. Endurance (n=37)	257 ± 45	2.65 ± 0.61	81.0 ± 9.5	31.5 ± 2.8	21.3 ± 2.8
3. Controls (n=67)	271 ± 50	2.47 ± 0.58	80.8 ± 10.1	32.0 ± 2.7	21.5 ± 1.3
P1-P2	0.05	NS	0.05	NS	NS
P1-P3	NS	0.02	0.02	NS	0.001
P2-P3	NS	NS	NS	NS	NS

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

9. Changes in the parameters of aerobic working capacity

Aerobic working capacity depends on the ability of the body to provide energy for muscle activity through the aerobic mechanisms of oxidation, and is characterised by:

- Aerobic power (oxygen consumption under physical stress or VO_2max),
- Submaximal endurance.

Although it is a very important parameter, VO_2max 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 identical performance, with the one having high VO_2max and relatively poorer oxygen utilisation, and the other having lower VO_2max but displaying

more economical oxygen use under physical stress. It has been found that athletes revealing similar results can have rather different VO_2max scores. Therefore, important as this parameter can be, it is not crucial for a sportsman's achievement. The factor that matters here is the percentage of the athlete's own VO_2max that can be used in performing a specific physical work. Daniels J (1974) and Costill D (1979) established independently that different athletes have identical running speed in a competition but at a different percentage of their maximal oxygen consumption (11,12). Better athletes run using smaller amounts of oxygen, that is, they have greater running economy.

VO_2max reflects to a large extent oxygen transport and delivery to working muscles. While it can be in the range of 40 to 55 ml $O_2.kg^{-1}.min^{-1}$ in untrained subjects, it can reach values of up to 77 ml $O_2.kg^{-1}.min^{-1}$ and even 90 ml $O_2.kg^{-1}.min^{-1}$ in elite athletes. VO_2max is genetically determined and with the development of the individual, it can be raised through training by 5-15% (Figure 6).

Changes that take place in aerobic capacity with age have been shown in the longitudinal studies of Trappe SW et al. (1996) on a contingent of elite sportsmen (38). The survey started in the 60s and lasted till the end of the 90s. A marked reduction of VO_2max (over a period of 20 years) was found in a group of athletes that had stopped training - decrease by

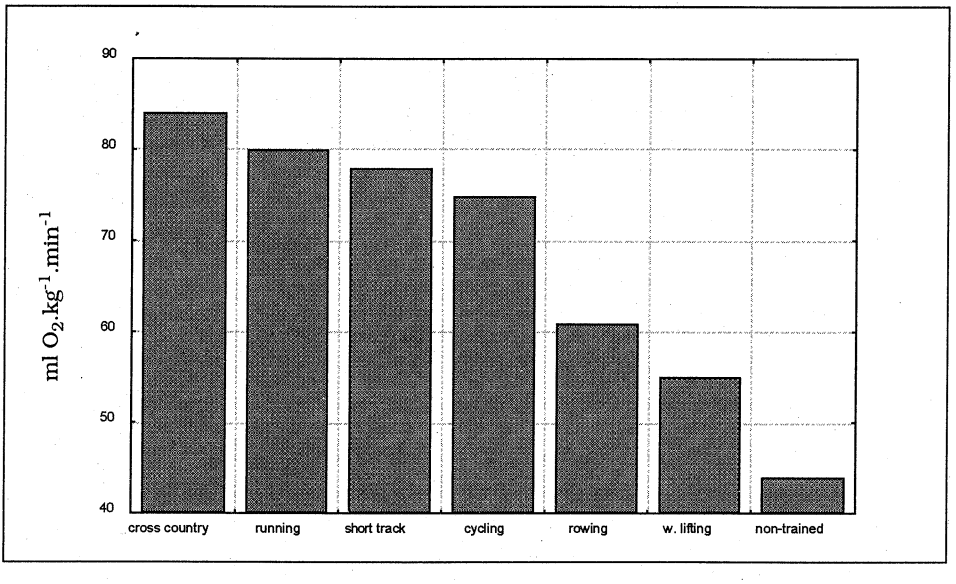

Figure 6. VO_2max of elite sportsmen of different sports.

18% in the absolute maximal oxygen consumption and by 34% in the relative consumption because of weight gain. In those who kept on training, this reduction was 9% and 14%, respectively. Biopsy specimens were obtained from m. gastrocnemius in all subjects. The findings revealed 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 in the activity of those oxidative enzymes, which was most probably the cause for their pronounced reduction in VO_2max.

It is important to bear in mind the fact that 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 air to the muscle cell,
- An internal (mitochondrial) oxygen utilisation system realising the aerobic production of energy.

What is the restricting factor for VO_2max? This issue is quite controversial among sports physiologists. It is believed that muscle capacity for oxygen utilisation 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 increases in stroke volume and cardiac output are very important for the enhancement of the aerobic capacity of the body as a result of regular submaximal exercise.

Conclusions

Adaptive changes in the body that result from submaximal exercise can be summarised as follows:

1. Key oxidative enzymes in the carbohydrate breakdown pathway (HK and CS), in the fat breakdown pathway (3-HAD and CPT) and in the respiratory chain (CCO) increase their activity.
2. Selective vascularisation in the muscles containing predominantly oxidative muscle fibres is induced.
3. A type change of the muscle fibres is possible to occur (including changes from type II to type I) in the submaximally recruited muscle groups.

4. Glucose transport into the recruited muscle cells is selectively increased with direct insulin mediation.
5. A metabolic adaptation occurs which consists in a shift of energy substrates from carbohydrates to fats, and a subsequent glycogen sparing effect, which guarantees fatigue delay and work capacity increase. Serum levels of total cholesterol decrease permanently.
6. Permanent changes occur in the humoral immunity, expressed as increases of serum IgA and IgG levels.
7. The oxygen transport system 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 utilisation occurs, which results in a non-significant increase of the maximal oxygen consumption and a significant increase of the oxygen running economy.

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