Adaptation to Training and Performance in Elite Athletes

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Physical performance capacity is influenced by various factors such as the quality and quantity of metabolic processes and the function of cellular structures and organs. Nevertheless, for nearly all types of sport, the aerobic capacity (i.e., the potential to provide a maximum amount of oxygen to the working muscles) plays a very important role and is often the limiting factor. The aerobic capacity in elite athletes is determined by three major factors:

1. the acute ability to mobilize physiological and biochemical processes to achieve the maximum performance,
2. the intensity and duration of training and the related functional, morphological, and metabolic adaptations, and
3. the inherited potential.

The acute and repeated stimulus of training induces various adaptations within the athlete's organism and numerous metabolic processes. Likewise, anatomical structures adapt to physical exercise leading to improved performance (See Figure 1). Although the maximum performance level which can be achieved by the individual may be partly inherited, an increase of the aerobic capacity over the years is definitely a training result. Maximum oxygen uptake capacity is determined by the metabolic, cardiovascular, and respiratory systems and not by contractile processes. Consequently, an increased oxygen uptake capacity has to be mediated by structural and functional adaptations within metabolism or the cardiovascular system. This includes increased number of mitochondria, functional improvement of enzyme systems, augmented blood flow in the skeletal muscles due to increased diameters of the blood supplying arteries, as well as increased number of capillary vessels, etc. Despite our rising knowledge of acute regulation, as well as functional and structural adaptations, the factors which limit oxygen uptake capacity individually are not well understood.

Current research concerning the degree and mechanisms of interactions between the genotype, phenotype, individual response to training, and physical performance may bring promising results. The data concerning maximum oxygen uptake generally support a significant contribution of hereditary factors. It is concluded that the hereditary transmission of VO2max is between 40% and 60% of the total phenotypic variation. At this time there is no evidence to suggest that the phenotype for aerobic performance is influenced by a single gene only. Considerable individual differences exist in the response to exercise and training and the related processes of adaptation and improvement support the view for a polygenic heredity.

The present work summarizes aspects concerning physical performance in elite athletes and the related processes of adaptation. Special emphasis is placed on the maximum oxygen capacity since it is one of the most important factors determining physical performance.

Figure 1. Various inherited and external factors determine the athlete's performance.

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Acute Reactions to Exercise

When evaluating performance or training effects one must distinguish between the acute reactions of exercise and the long term chronic adaptation to training. After the “tuning” from rest to strenuous exercise, functions and activities of organs change in a large range and new levels of energy turn-over and aerobic and anaerobic processes are established corresponding to the energetic demands. The physiology of muscular work and exercise is basically the transformation of chemically bound energy into mechanical energy. The kind of substrates utilized for this transformation process depend on the intensity and duration of exercise. During short and intensive bouts of exercise (i.e., not more than 10 s), energy is supplied directly by the primary energy source, the pool of ATP and creatine phosphate (CP) stored in the muscles (Keul & Doll, 1973). If muscular work continues over a longer period of time, ATP and CP must be regenerated by metabolism of glucose or fatty acids. During mild or moderate exercise (up to a work load of approximately 65% of the maximum work capacity), energy is provided mainly aerobically by the oxidation of muscular glycogen and triglycerides as well as circulating blood glucose and free fatty acids (Holloszy, 1973; Keul & Doll, 1973). With prolonged duration of exercise and an improved endurance training condition, the relative proportion of fatty acids used for energy production increases (Holloszy & Coyle, 1984). Fatty acids are provided by augmented release of fatty acids from adipose tissue, or via hydrolysis of circulating lipoproteins and intramuscular triglyceride stores (Hurley et al., 1986). The main lipase involved in triglyceride hydrolysis is lipoprotein lipase (LPL) (Taskinen, Nikkilä, Rehunen, & Gordin, 1980). When the muscular triglyceride stores are mobilized during exercise, the synthesis of LPL in the muscular endothelial bed is enhanced, and the activity of adipose tissue LPL is elevated (Kiens & Lithell, 1989) leading to an increased catabolism of triglycerides and triglyceride rich lipoproteins. The transport of the liberated fatty acids across the muscle cell membrane plays a key role in supplying the mitochondria with a sufficient amount of fatty acids for β-oxidation. In contrast to former hypotheses of a passive, diffusion-mediated fatty acid uptake into the muscle cell, it is currently accepted that circulating fatty acids bound to albumin are incorporated into myocytes by a highly specific transport system, the fatty acid binding protein (Stremmel, 1988).

Although it is unknown whether this transport system increases during exercise, the diminished concentrations of circulating fatty acids observed during and after exercise may be an indirect marker for an increased cellular uptake of fatty acids in addition to the known shift in energy balance toward a higher intracellular fatty acid oxidation (Koivisto, Hendler, Nadel, & Felig, 1982). With increasing exercise intensity, glucose is preferentially metabolized resulting in lactic acidosis and symptoms of muscular fatigue. Glucose uptake into the muscle cell is increased via an exercise-induced translocation of glucose transport proteins (GLUT-4) from the microsomal pool to the cell surface (Marette, Atgie, Liu, Bukowiecki, & Klip, 1993). The composition of membrane bound and circulating fatty acids also seems to be important factors for muscular glucose metabolism (Borkman et al., 1999; Vessby et al., 1994) as a high content of long-chain polyunsaturated fatty acids improves insulin sensitivity and muscular glucose uptake.

The amount of oxygen which can be delivered to the muscles and utilized as energy fuel is decisive for endurance stamina at higher intensities (70-75% VO₂max). The increased oxygen transport is mediated mainly by acute vasodilatation of arterioles and opening of existing capillaries. Thereby, the arterial vascular resistance falls in exercising muscle groups which results in an increase of regional blood flow, and a consequently higher venous return from the active muscle groups (Saltin, 1986). The reduction of local arterial vascular resistance and an increase in venous blood leads to a reduction in cardiac afterload and to a rise in preload (Blomquist & Saltin, 1983). Under these conditions, the diastolic filling and systolic emptying of the heart are augmented, resulting in a higher stroke volume. However, only small changes of the pO₂ or pCO₂ are measurable in the arterial blood. This is due to an increased transport of O₂ and CO₂ across the alveolar capillary membrane and an improved peripheral oxygen utilization. The additional activation of the sympathetic nervous system results in a rise in heart rate and an increase in myocardial contractility. In the arterial vascular system, selective vasoconstriction occurs in non-active muscle groups and in the splanchnic bed (Saltin, 1986). This results in a redistribution of blood with a further increment in arterial blood in favor of exercising muscle groups.

Chronic Adaption to Endurance Training

For best performance comprehensive adaptations of morphological, physico-chemical, and metabolic functions have to be established to meet the challenges of competitive exercise (Viru, 1993). The degree of these adaptations is related to the intensity of training, the athlete’s genotype and phenotype but also factors such as age, nutrition and lifestyle, and many more, are responsible for the interindividual expression of functional and metabolic improvements (See Figure 1). The morphological changes, especially the training induced adaptations of the heart and circulatory system have to be considered as they are important for increased blood
flow to muscle cells leading to a higher oxygen supply and substrates and augmented elimination of metabolic waste products.

The Cardiopulmonary System. Verzar (1912) first proposed that prolonged muscular exercise may be aerobically limited by oxygen availability. A.V. Hill and his coworkers later suggested that pulmonary and circulatory limitations restrict the in vivo oxygen delivery to the muscle cell (Hill, Long, & Lupton, 1924). An increased oxygen supply can be mediated by morphological and functional adaptations of the heart and the circulatory system, leading to an economization of heart function, cardiac output, and peripheral blood provision. However, size and hemodynamics of the athlete's heart are directly related to the quality and quantity of training. The influence of extensive endurance training, involving more than 1/6 of the skeletal muscle mass for more than 5 hours a week, is characterized by dimensional changes of the heart, such as increased heart volume and muscle mass (See Figure 2). Longitudinal studies have shown that an increase in the diameter of the cardiac chambers of 20% and a rise in cardiac muscle mass of 70-80% are still within physiological limits. A doubling of the heart volume is accompanied by a doubling of cardiac output and oxygen uptake. The increase in the cardiac stroke volume is responsible for the improved cardiac performance and maximal oxygen uptake in athletes since the heart rate in trained and untrained individuals at maximal work load is not different. Therefore, the stroke volume, or the oxygen-pulse, as an indicator of the stroke volume size, is closely and linear related to the heart volume and left ventricular volume (See Figure 3). Furthermore, the sympathetic drive is inversely associated with heart size (Lehmann, Dickhuth, Schmid, Porzig, & Keul, 1984). The larger the athlete's heart, or rather the more pronounced the endurance aspect of training is, the lower the catecholamine serum concentrations are at rest or at comparable work loads. The endurance trained heart shows an improved efficiency and in consequence, the energy turnover in relation to the cardiac output is decreased (See Figure 4). The prolonged diastolic time-interval, as result of the reduction in heart rate and the changed compliance, allows a longer diastolic perfusion of the coronary arteries thereby improving the supply of oxygen and substrates (Heiss et al., 1976).

In contrast to endurance training, strength trained athletes have smaller hearts, even when they train intensively several hours per day. Static training leads to a concentric thickening of the myocardial walls and thus to an increase of the cardiac muscle mass at the cost of the ventricular volume (See Figure 2). In these athletes the myocardium is clearly thicker in relation to the end diastolic diameter, resembling a concentric adaptation of the heart. This results in a reduction of stroke vol-

Figure 2. A strong correlation exists between enddiastolic volume (EDV) and left ventricular muscle mass (LVMM) for untrained subjects (U) as well as for highly trained athletes (T) of both sexes (cyclists = C; runners = R; cross country skiers = S; weight lifters = W; triathletes = T; body builders = B; paraplegics = P; tetraplegics = Te).

Figure 3. Linear regression between heart volume, stroke volume and O₂-pulse.
during exercise to the same extent as in the untrained heart. In conclusion, the ratio of the end diastolic volume to muscle mass is increased in strength trained athletes whereas it is unchanged in endurance trained athletes. In accordance with the rule of La Place the relationship between the dimensions of the cavities of the heart and the cardiac muscle mass remain roughly constant. The training-induced enlargements of the inner dimensions of the heart cavities are followed by an increase in the thickness of the walls rather than the cardiac muscle mass. This can be used as a clinical criterion to differentiate between a physiologically adapted athlete's heart and a pathological heart (Auch-Schwelk, Dickhuth, Keul, Lehmann, & Meinertz, 1987; Huston, Puffer, & Rodney, 1985; Keul et al., 1981, 1982; Reindell et al., 1960). Hearts, which are enlarged as a result of myocardial damage, show no increase in shortening fraction and only an insufficient increase in the circumferential shortening velocity during exercise. Today there is no indication that the athlete's heart syndrome (even after extreme intensive training lasting decades) could be connected to pathological processes in any way.

The Respiratory System. In contrast to the cardiovascular system, no current research findings indicate that respiratory functions are adaptable because of increased physical exercise. Oxygen uptake in the lung is determined by oxygen pressure, anatomy of the airways, and diffusion capacity of the lung. The direct relationship between tracheal dimensions and maximum oxygen uptake across species suggests that, especially in obstructive pulmonary diseases, the airways of the lung can limit aerobic performance (Leith, 1983) but an improvement of airway function or respiratory muscles with training could not be demonstrated (Guyatt, Keller, Singer, Halcrow, & Newhouse, 1992). No significant difference between trained and untrained individuals could be observed with pulmonary oxygen transfer (Dempsey, 1986). Although lung capacity is more than adequate for normal individuals, elite athletes may be limited by pulmonary O2 transfer, particularly at high altitude or after prolonged strenuous exercise when transient changes in the structure of the alveolo-capillary membrane occur (See Figure 5) (Guenard, Manier, Moinard, Techoueyres, & Varene, 1991; Keul & Doll, 1973; Sutton, 1992). Nevertheless, increased peripheral oxygen demand can not be satisfied by adaptations within the pulmonary oxygen diffusion or transport capacity.

The Circulatory System. Similar to the heart, the arterial vascular system also adapts to chronic exercise. The nature of these training induced adaptations in arterioles and capillaries remains a subject of controversy. Most investigators have used the "shear stress hypothesis" to explain chronic vascular adaptations. According to this hypothesis, rising intravascular shearing forces, resulting from the increase in regional arterial blood flow during dynamic exercise, stimulate the vascular endothelium, but metabolic (particularly hypoxic) factors have also been suggested to play a role in structural adaptations of the vascular system induced by muscular training (Huonker, Halle, & Keul, 1996). The majority of studies presently available indicate that regular muscular training induces changes in basal arterial tone, peripheral vascular resistance and increases the cross-sectional area of medium sized and small arteries (Huonker, Simons, Schumacher, & Keul, 1994). Investigations of vascular dimensions in highly trained cyclists (Kool, Struijker-Boudier, Wijnen, Hoeke, & Van Bortel, 1992) have revealed that the femoral arteries of these athletes have a larger cross sectional area than the femoral arteries of sedentary individuals (See Figure 6).

![Figure 4](https://example.com/figure4.png)

**Figure 4.** The oxygen consumption of the myocardium at rest and during exercise is significantly reduced in athletes compared to untrained in relation to cardiac output. Vcor is myocardial blood flow in ml/min * 100 g myocardium. *p < .05, **p < .01, ***p < .001

![Figure 5](https://example.com/figure5.png)

**Figure 5.** During maximal exercise at altitude elite athletes show a greater reduction of P(O2) than sedentary individuals. This indicates that pulmonary function shows no apparent signs for adaptation to training and increased oxygen demand (black bars = athletes; grey bars = sedentary controls). *p < .05; **p < .01; ***p < .001

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and that the brachial and carotid arteries show similar dimensions in trained cyclists and untrained men (Huongker et al., 1994). Similar results are found in regularly exercising swimmers and triathletes. Swimmers, primarily using their arms for movement, show larger vascular dimensions of the arteries supplying the arm muscles whereas triathletes, using arms and legs, show increased vascular dimensions in all extremities. In contrast, elite weight lifters, including Olympic champions and world record holders who train several hours every day, show no increase of vessel dimensions or blood flow. Blood flow is reduced relative to muscle mass.

Side differences of the cross sectional areas of arteries are observed in tennis players (see Figure 6). The dimensions of arteries of the dominant arm are larger than the size of the contralateral arm, which means that these arteries have adapted to the regular exercise of the single arm. Overall, these findings show that there are regional structural adaptations of vessels due to an increased muscular demand for blood during exercise. Interestingly, the capability to adapt is only evident for peripheral arteries that supply skeletal muscles with blood. Central vessels show no signs for adaptation even in highly endurance trained athletes. Therefore, their size and function and their ability to increase may be limiting factors for blood flow and oxygen.

The Muscular Metabolism. Skeletal muscle mitochondria are responsible for the consumption of more than 90 percent of the oxygen uptake during exercise at maximal oxygen uptake level. The higher aerobic capacity of athletes can be achieved with an increase in size and number of mitochondria and enzyme mass. The whole body maximal oxygen uptake is related to the total volume of the body's skeletal muscle mitochondria. The ratio of VO₂max to total mitochondrial volume is an indicator of the maximal in vivo respira-

tion rate per unit of mitochondrial volume (Hoppeler et al., 1985; Hoppeler & Turner, 1989; Lindstedt, Wells, Jones, Hoppeler & Thorson, 1988). The calculated mitochondrial respiration rate is rather constant and ranges about 5 ml O₂ min⁻¹ml⁻¹ for all mammalian species. These findings suggest that in vivo mitochondrial maximal oxygen consumption is variable in mammals. Athletes achieve their higher aerobic capacity by increasing the muscle mass with a greater volume density of mitochondria. In this cellular compartment, ATP for muscular contraction is derived from the oxidation of glucose and fatty acids and oxyhydrogen reactions. Triglyceride and fatty acid metabolism can be improved with regular physical exercise by increasing the activity of lipid enzymes primarily lipoprotein lipase (LPL) (Kiens & Lithell, 1989). Thereby triglycerides of VLDL particles can be mobilized more rapidly to meet the demands for fatty acid oxidation within the exercising muscle cells (Berg, Frey, Baumstark, Halle, & Keul, 1994). In addition, other lipid enzyme activities such as cholesterol ester transfer protein (CETP) and hepatic triglyceride lipase (HL) are reduced in athletes, whereas, that of lecithin-cholesterol-acyl transferase (LCAT) or transfer proteins is reduced (Durstone & Haskell, 1994; Seip et al., 1993). As CETP induces the exchange of HDL cholesterol esters for VLDL triglycerides, an impairment of CETP activity reduces the loss of triglycerides from VLDL particles, which is important for the peripheral fatty acids supply during exercise.

Besides its function in triglyceride lipolysis, LPL also plays an important role for the transfer of triglycerides into the muscle cell (Simoneau, 1995). As its activity is enhanced in athletes, a better supply of triglycerides can be expected in trained individuals. Furthermore, carnitine palmitoyl transferase, the limiting step of the transfer of long-chain acyl CoA esters into the mitochondria, and the B-hydroxyacyl CoA dehydrogenase, a marker enzyme of intramitochondrial fatty acid oxidation, is increased in trained individuals (Tremblay, Simoneau, & Bouchard, 1994). Therefore, athletes have an increased triglycerides hydrolysis in the circulation as well as an increased transport of fatty acids into the muscle cell and the mitochondria for oxidation. These training-induced changes lead to an increased caloric energy expenditure from lipids in well-trained endurance athletes.

Inherited Potential

The degree and nature of interactive effects between genotype and physical performance or training induced adaptations remains largely unknown (Bouchard & Malina, 1983). In regard to maximum oxygen uptake the current literature presents an enormous range of different data. The postulated size of genetic effects for VO₂max differs between 10% (VO₂maxFFW) (Bouchard et
More concordant data are available concerning the heritability levels for endurance performance. A study with twins showed a 70% genetic effect for aerobic performance measured by a 90 minute endurance test and a 93.4% genetic determination of maximum aerobic power determined by treadmill running to exhaustion (Klisouras, 1971).

From these data it can be concluded that the genetic effect for endurance performance is more pronounced than for VO2max. Therefore, the potential to improve aerobic capability seems to be dependant on genetic factors. In training studies, training gains from 0-41% (Prud'Homme, Bouchard, Leblanc, Landry, & Fontaine, 1984) and > 60% (Bouchard et al., 1986; Bouchard & Malina, 1983) were detected after endurance training and strong correlations were found between achievable results and heredity. There seems to be an inherited potential in regard to low and high responders to training and it can be estimated that the heritability of VO2max is between 20% and 60% of total phenotypic variation (See Figure 7) and the range to improve VO2max by training is between 20-40%. Three mitochondrial DNA morphs, two in the NADH dehydrogenase gene and one in the tRNA for threonine, have been found and carriers of these genes had significantly higher VO2max than noncarriers, even in the untrained state (Dionne et al., 1991). Dionne also found a lower response to training for carriers of a variant in subunit 5 of the NADH dehydrogenase and the authors concluded that sequence variation in mitochondrial DNA may contribute to interindividual difference in VO2max and its response to training. At the moment there is no evidence to suggest that aerobic performance phenotype is influenced by a single gene only. Furthermore, the heredity of aerobic performance seems to be a polygenic approach with a more or less huge panel of genes involved in the expression of a wide range of physiological control mechanisms.

Implications

Ideal structures resulting from an optimal structural adaptation form the basis for an ideal function. The variability of these structural and metabolic functions are determined by the genotype and the individual phenotypic expression. The factors regulating the degree of phenotypic expression and the related training induced adaptations are far from known. Despite training induced adaptations, there are some fixed factors (e.g., maximum heart rate, diameter of central elastic arteries) that do not change even in elite athletes, while changeable characters such as heart size, peripheral artery diameters, and cardiac output may increase in capacity by up to 100 percent. As training and energy requirements increase, the adaptable characters change in order to fit the augmented demands. The maximal degree of adaptation seems to be partly inherited, but is also influenced by external factors such as nutrition, training conditions, environment, and many more. Only regular, long-term challenge of the limiting compartment responsible for oxygen transport and energy production will lead to the greatest possible adaptation and for the realization of maximal performance during competition.

References


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