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THE PHYSIOLOGICAL AND BIOCHEMICAL BASIS OF AEROBIC AND ANAEROBIC CAPACITIES IN MAN; EFFECT OF TRAINING AND RANGE OF ADAPTION

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Introduction

In evolution the aerobic and anaerobic capacities of man have been essential components for survival. This is hardly the case to-day. Although this is true there is a great deal of interest in the energy yielding systems of the body, and their maximum rates. One reason for this is that the application of this knowledge has a bearing in sports, where energy turnover limits man when moving fast or in efforts of long duration. The measurements of aerobic and anaerobic energy delivery were made early by some of the pioneers in muscle and exercise physiology. Krogh and Linhard defined the oxygen deficit (1919/20) and A.V. Hill the maximal oxygen uptake (1923). The latter also elaborated on the interplay between these sources of energy in running and demonstrated that an even speed was most economical in a race (Hill, 1927). Although more than half a century has elapsed since these studies were performed many unsolved questions are still present. In this article an attempt is made to give an account of the various topics while identifying where the limitations to performance may be and point at the malleability of various systems in the body to overcome these limitations.

Anaerobic capacity

How to measure: An accepted method to measure a person's anaerobic capacity is not available. Several routes have been tried, but objections on theoretical grounds – can be made against the various trials to quantitate the anaerobic energy yield.

Measurement of lactate in blood after exhaustive exercise have frequently been used, and Margaria and associates have gone the furthest to use such a measure to estimate the anaerobic energy release (1963, see also Jacobs, 1986). There are, however, several difficulties. One is to identify, when there is an equilibrium between muscle and blood lactate concentration. Another is the variability of dilution space for lactate and a third is that lactate has a high rate of turn-over. Before an equilibrium is reached between muscle and blood and the lactate is evenly distributed in the various water spaces of the body a large fraction of the lactate has been metabolized. Thus although everybody would agree that lactate in the blood is an indication of glycolysis, it is equally true that it cannot give a fair estimate of the anaerobic energy yield, and is thus abandoned as a quantitative measure of anaerobic capacity.

It was shown early that the oxygen deficit accumulated during the exercise was paid off during recovery (Christensen and Høgberg, 1950). A measure of the recovery oxygen uptake above pre-exercise or resting value (oxygen debt) has also been proposed and used as a measure of the anaerobic capacity. Several drawbacks with this method limit its value. More energy appears to be needed to use lactate as substrate for a synthesis of glucose (glycogen) than is liberated when lactate is produced. Further, an unknown quantity of lactate is oxidized, which will then not be shown as an «extra» oxygen consumption. The largest problem is related to the fact that other factors than elevated lactate elevates the oxygen consumption of the body. Oxygen uptake measurements may still be used as an estimate of the anaerobic capacity using various factors (2 ↔) to convert the oxygen debt into an anaerobic energy yield but it is hardly recommended. The third route is to determine the oxygen deficit just as was done in the "old days". This approach has been used extensively; not always to obtain a measure of the maximal oxygen deficit that could be achieved but to quantitate the anaerobic energy contribution to the work performed. In short-term submaximal exercise there are no theoretical objections to use the oxygen deficit as a measure of the anaerobic energy release (figure 1). The problems arise at exhaustive exercise. If this work exhaust the sub-

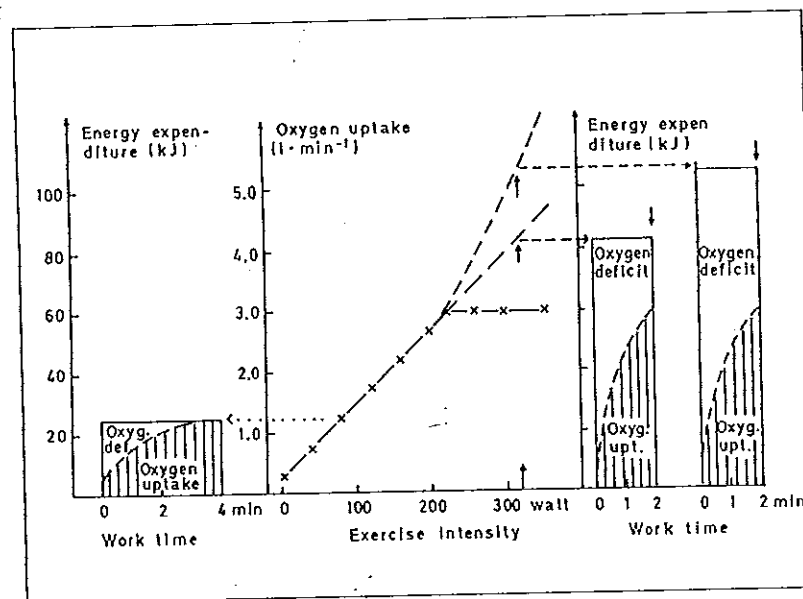


Figure 1. A schematic illustration is given for the relationship between work intensity and oxygen uptake. The typical linear increase is found until the oxygen uptake levels off (middle panel). An extrapolation of the linear relationship is also indicated and used to estimate the energy cost of a «supra» maximal work load (right panel). Included is an extrapolated relationship between a high work intensity and energy demand, which to the right is used to indicate that energy cost may be underestimated in very intense exercise. In the left panel is depicted the situation during submaximal work.

jects within short duration (2–6 min) it is likely that a maximal value for the oxygen deficit is reached (Medbø et al., 1986). However, the energy cost of the exercise must be accurately known to calculate the oxygen deficit. This is not difficult at submaximal work loads, where the steady state oxygen uptake represents the energy costs. At exhaustive exercise, however, the validity of the estimate of the true energy cost is less certain. This uncertainty relates to both methods used to estimate the energy costs, which are either to assume a given mechanical efficiency or to extrapolate from the submaximal relationship between work intensity and oxygen uptake. Such estimations are likely to underestimate the true energy expenditure during maximal work the reason being that mechanical efficiency is lower in exhaustive than in submaximal exercise. How much lower it is nobody knows. Further methods are not available to experimentally approach the problem. For bicycle work intensities which are close to those which elicit maximal oxygen uptake mechanical efficiency is in the range of 16–19% rather than the ordinary 20–25% (Saltin et al., 1972). The lowest mechanical efficiencies were found at the highest relative intensity, which may indicate that the higher the work load, the larger is the underestimation of the true energy cost. (In the above discussion, the significance of the lactate production during submaximal exer-

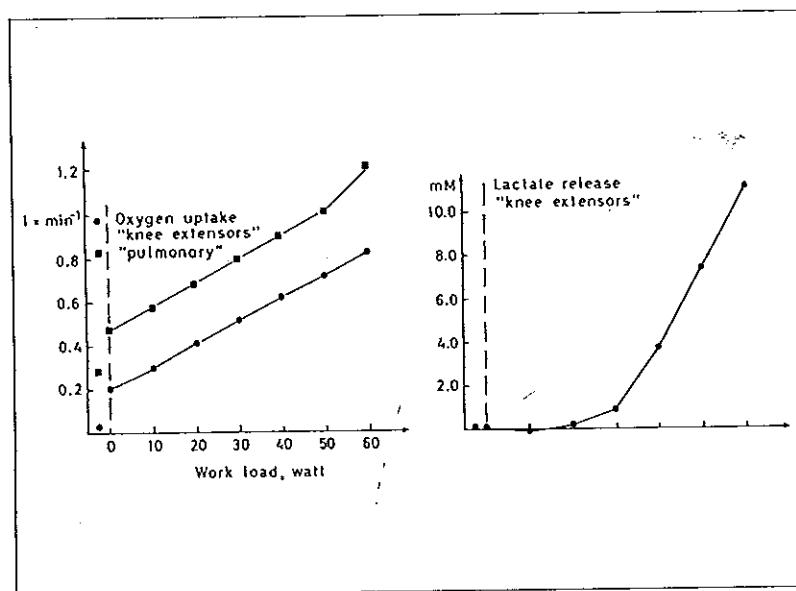


Figure 2. Data on pulmonary and knee-extensor oxygen uptake and knee-extensor lactate release at various work intensities performed with the knee-extensors of one leg (data are from Andersen and Saltin, 1985). It should be emphasized that muscle oxygen uptake increases linearly with work intensity, but at the higher work rates there is a rather significant lactate release indicating an energy yield from glycolysis. This may mean that already at high submaximal work intensities the mechanical efficiency is less than the expected 21–24%.

cise for calculations of mechanical efficiency or estimation of energy costs from extrapolations at submaximal work is not considered; see figure 2). In spite of all the objections of using the maximal oxygen deficit (or accumulated oxygen deficit as called by Medbø et al. 1986) as a measure of anaerobic capacity it is the only method available worthwhile to use. Its potential should be explored.

Components of the oxygen deficit: The energy derived to cover the oxygen deficit is not only originating from glycolysis but also from the phosphagens and an aerobic energy yield not measured as an oxygen consumption. At the start of the exercise muscle ATP and CP concentration is high (≥ 30 mmoles \times kg⁻¹) and at exhaustion they are low, reduced to 1/5 of the resting concentration. Depending on the muscle mass involved in the exercise the actual amount of this alactacid component of the oxygen deficit varies slightly. The reason why aerobic processes also are included in the oxygen deficit is that oxygen bound to hemoglobin and myoglobin is reduced from start to end of an exercise period. The magnitude of these two components are smaller than the lactacid component and amounts as a maximum to 1/3 of the total oxygen deficit (Table 1). More important is that training produces only very minor alterations in the absolute values for the contributions of these variables to the oxygen deficit. ATP and CP concentration in muscle is basically unaffected by any type of training and the degree of depletion during exercise is more a function of the relative intensity than the training status. Endurance training will enlarge the amount of hemoglobin (and myoglobin?) and then the oxygen stored, but again the magnitude means very little extra for the oxygen deficit.

Table 1. Components of the oxygen deficit. Estimations have been made for a sedentary subject (sed) and for two with larger anaerobic capacities (T: I and T: II). The exercise involves primarily the leg muscles.

Variable	O ₂ equivalents ml · kg ⁻¹			Percent of total		
	SED	T: I	T: II	SED	T: I	T: II
Oxygen stored to Hb and Mb	4	5	6	10	6	6
Phosphagens (alactacid oxygen deficit)	12	15	19	30	19	19
Glycolysis	24	60	75	60	75	75
Total MI-O ₂ Eq-KG ⁻¹	40	80	100	% 100	100	100

Magnitude of the anaerobic capacity (maximal or accumulated oxygen deficit): Although the concept of oxygen deficit is old it has not really been used systematically to evaluate the range of anaerobic capacities of man. Medbø and associates (1984, 1986a, 1986b) are just in the beginning of such investigations and can supply with data on a total of 19 subjects. These data together with some results obtained from the literature are summarized in Table 2.

Table 2.

Summary of some data in the literature on oxygen deficit in males and related variables including training status (sedentary (sed), endurance trained (et) and sprint trained (st)).

Reference	N	Age years	Training status	Weight kg	Oxygen deficit L	Work time minutes	Peak blood mM	Lactate
Eriksson et al., 1973	8	11,5	SED	44,7	1,48	~ 5	4,7	
Eriksson et al., 1973	8	12,1	ET	45,4	1,64	~ 5	5,9	
Åstrand-Saltin, 1961	4	29	ET	77	6,25	~ 3	14,8	
Karlsson-Satin, 1970	3	26	ET	74	4,95	~ 2,4	13,4	
Linnarson et al 1974	6	29	ET	75	5,75	~ 4	15,6	
Hermanssen-Medbø 1984	6	25	ET	70	3,15	~ 0,9	12,6	
Hermanssen-Medbø 1984	6	25	ST	75	4,06	~ 0,95	17,0	
Medbø et al., 1986	4	22	SED	74	4,72	~ 2	16,6	
Medbø et al., 1986	7	26	ST+ET	78	6,04	~ 2	16,6	

Sedentary subjects appear to have an anaerobic capacity of around 40–70 ml "O₂ eq" × kg⁻¹. The low and middle part of the range is rather well established. The high end contains only a few observations. None were champions ("national class") athletes, and those studied were not in peak training condition. A value of 100 ml "O₂ eq" × kg⁻¹ or more may be a likely estimate for good sprint trained athletes running or bicycling.

Of utmost importance is the fact that when more than the leg muscles are intensely involved in the exercise as in whole body exercise the maximal oxygen deficit is more than 50% larger (figure 3). This could be anticipated as the magnitude of the maximal oxygen deficit must be a function of the muscle mass engaged in the exercise. No measurements are available on top athletes performing whole body exercise. Thus the upper range for anaerobic capacity as in successful rowers or swimmers is presently unknown. If it is assumed that a 800 m runner of top class has the equivalent of 120 ml "O₂ eq" × kg⁻¹ in anaerobic capacity while running, a top rower should have close to 200 ml "O₂ eq" × kg⁻¹ in an all out rowing performance.

Young pre-puberty children appear to have a much lower anaerobic capacity than adults, as judged by Eriksson et al.'s data (1973). These 11–12 years old boys had an anaerobic capacity of only 35 ml "O₂ eq" × kg⁻¹ and endurance type training had only a very minor influence on this capacity. Both muscle and blood lactate concentrations were also low in these boys. These findings have frequently been interpreted as anaerobic events are unsuitable for young kids, but that is not a proper interpretation. It only means that they perform less well. However, they probably have the advantage to recover more quickly as less lactate has to be dispersed. After puberty adolescent children have similar glycolytic enzyme levels as adults and they then also exhibit "normal" blood lactate concentration after exhaustive work.

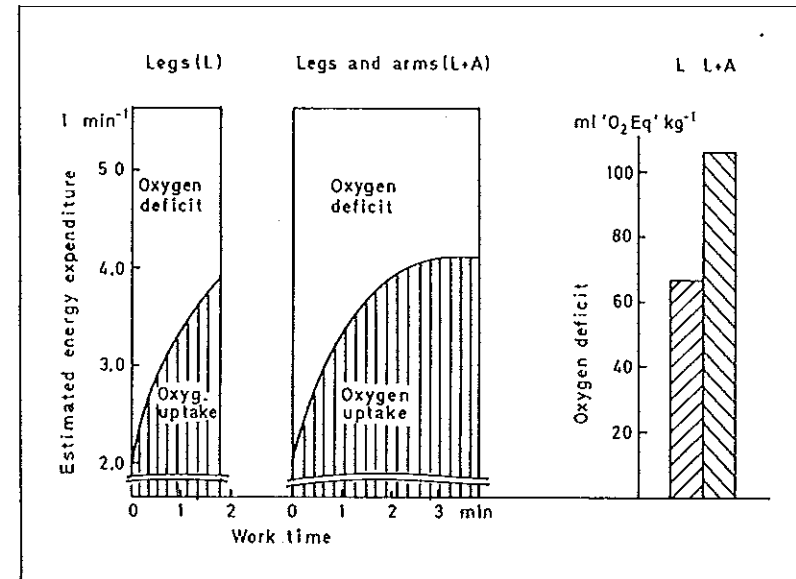


Figure 3. The oxygen uptake and the estimated oxygen deficit are illustrated when exercising with the legs (left) and with arms and legs (right) in one subject. These data demonstrate that the fraction of the muscle mass involved in the exercise is decisive for the magnitude of the maximal oxygen deficit. Adding arms to leg bicycle exercise increase the oxygen deficit with a factor of 0,5–1,0 (Åstrand and Saltin, 1961).

Apparently, there are no systematic observations of anaerobic capacity in females. Thus, it can only be speculated as to whether or not they can achieve the same maximal oxygen deficit as men. The only factor speaking against this being the case is that at a given body weight the muscle mass will be slightly less in women who thus will most likely possess a smaller anaerobic capacity per kg body weight.

Rate of contraction of the anaerobic capacity: It is of note that an oxygen deficit is a capacity not a rate as maximal oxygen uptake, but it is utilized with a certain rate. This rate varies during a measure of the oxygen deficit (see figure 1) just as it does during a race. The unloading of oxygen from hemoglobin and myoglobin is rapid and the new lower equilibrium concentrations for ATP and CP in the contracting muscles is also reached within 15–20 seconds. The rate of the lactate production can be extremely high and 1–2 mmoles × kg⁻¹ can be accumulated in the muscle over some few seconds (figure 4). Further in contrast to earlier belief the glycolysis starts also in dynamic exercise at onset of the exercise. With this in mind it could be anticipated that the whole oxygen deficit is utilized within a minute or less. This may not be the case. According to Medbø et al. (1986) only 50–60% of the total oxygen deficit was utilized after one minute of the exercise but after two minutes the whole capacity was exhausted (figure 5). This would mean that in a 100 m swim event or 400 m dash a high lactate accu-

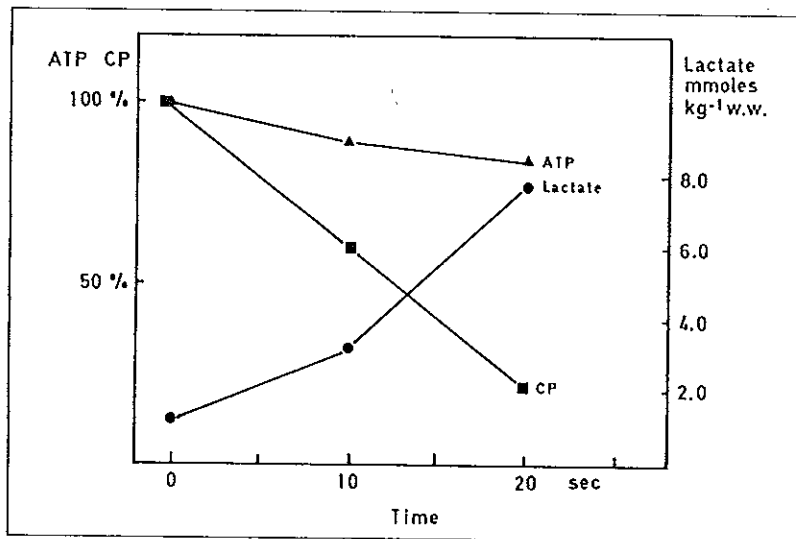


Figure 4. The reduction of ATP and CP and the increase in lactate of contracting muscles are depicted after onset of exercise (Saltin et al., 1971).

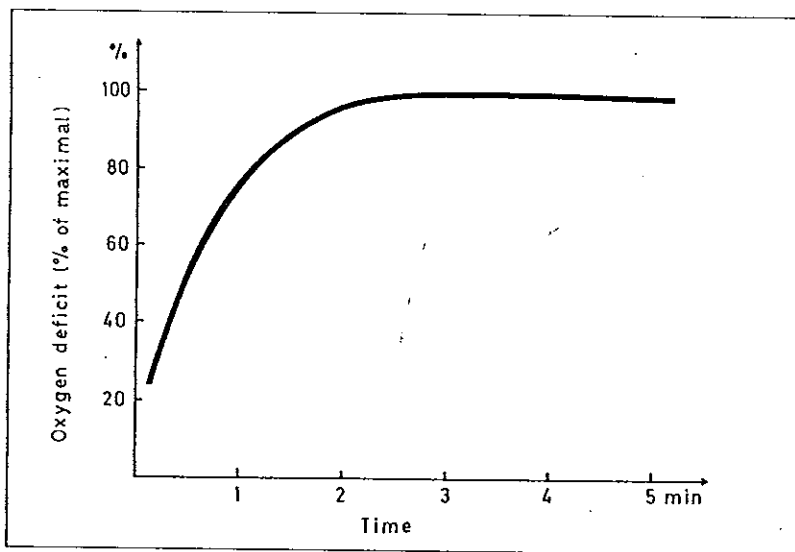


Figure 5. Data taken from Medbø's et al. study (1986) of the fraction of the maximal oxygen uptake that can be utilized in exhaustive exercises lasting various times.

mulation will not be limiting. Events like 200 m swimming or running 800 m are then the shortest distances where the full anaerobic capacity can be utilized.

It is of note that none of the subjects studied were trained to perform optimally for one minute. The rate by which the oxygen deficit can be contracted may be trainable. If there is a limitation in using the full oxygen deficit in less than 2 minutes it cannot be due to the "aerobic" or the alactacid components of the oxygen deficit, rather the rate of glycolysis would be the component that is limiting.

Another possibility relates to the above discussion of the difficulties to precisely estimate the energy costs at high exhaustive work rates. It was noted that the energy costs of so called supra-maximal efforts may be underestimated, and if that is the case it will be more so the shorter the maximal work time (see figure 1). The result of this would be a larger underestimation of the true oxygen deficit in a one minute exercise bout than one lasting two minutes or more. This could in part explain why Medbø et al. (1986) found such a low fraction of the anaerobic capacity used in one minute.

If the intense exercise can be tolerated for more than 2 minutes, both Medbø et al.'s and earlier data demonstrate that, if the subjects are motivated, the same maximal oxygen deficit is achieved in exhaustive exercise lasting between 2 and 16 min (figure 6). Of note is also that exercise performed at low or high barometric pressures result in the same anaerobic capacity (figure 7). In more prolonged exercise the role of the anaerobic capacity is small. This is not only due to its minor relative role for the over-all energy de-

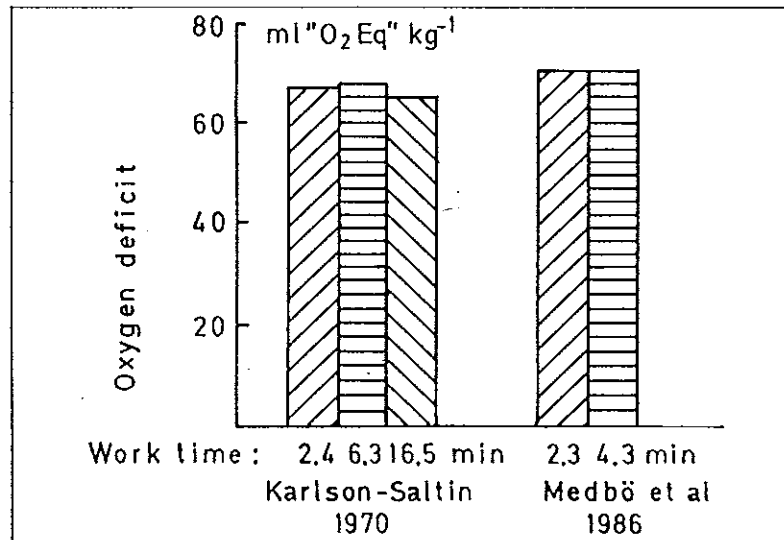


Figure 6. Mean values for the maximal oxygen deficits determined in exhaustive exercise of various duration (Åstrand and Saltin, 1961; Karlsson and Saltin, 1970; Medbø et al., 1986).

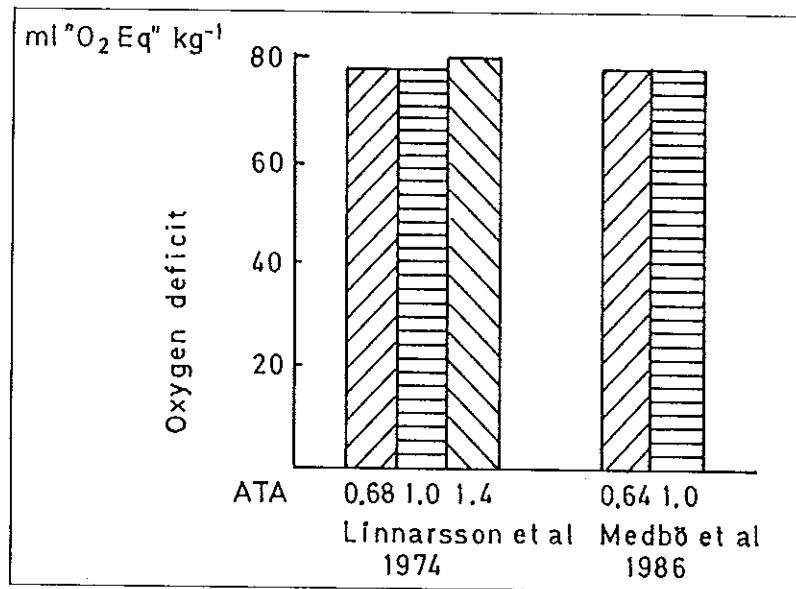


Figure 7. Mean values for maximal oxygen deficit achieved at different barometric pressures (hypoxia, normoxia and hyperoxia). Maximal work time was shorter at 0.68 and 0.64 ATA and longer at 1.4 ATA as compared with sea level (1.0 ATA). (Linnarsson et al., 1974; Medbø, 1986).

livery but also in the finish. The reason for this is that it may not be any glycogen left in the muscle fibers. Without such a supply the rate of glycolysis cannot be very high and lactate formation is small. Already after 45–60 min of intense continuous exercise a large fraction of the fast twitch muscle fibers are glycogen depleted (Ander and Sjøgaard, 1976).

Relative role of the anaerobic energy yield: Many tables or graphs have been published on this topic. The data in Table 3A are based on the assumption that champion athletes in typical anaerobic events like 400–800 m running have an anaerobic capacity of 100 ml "O₂ eq" × kg⁻¹ and can use it all. Their assumed maximal oxygen uptake is less impressive or 60 ml × kg⁻¹ × min⁻¹. At the other end the assumed values for the endurance trained athletes are 80 ml "O₂ eq" × kg⁻¹ and 80 ml × kg⁻¹ × min⁻¹, respectively. At the shorter duration of the exercise the anaerobic energy yield dominates. Although aerobic processes starts to dominate already at a work time of 2 min it should never be forgotten that the anaerobic capacity is so decisive at the finish and thus for the placing in major events.

In whole body exercise like swimming and rowing the relative importance of the anaerobic events are more dominant as the anaerobic capacity is larger (Table 3b). Not until competition time exceeds 3–4 minutes does the aerobic energy yield dominate, but 1/3 of the energy still is derived from anaerobic processes at 6 min exercise.

Table 3. Absolute and relative (within paranthesis) contribution of anaerobic processes for the energy yield in exhausting exercise with various durations. It is of note, that Table A relates to running or bicycling and the values for the estimations are based on a maximal oxygen uptake of 60 ml · kg⁻¹ and an anaerobic capacity of 100 ml "O₂ eq" kg⁻¹ for the shorter durations. The respective values for the longer exercise periods are 80 ml · kg⁻¹ and 90 "O₂ eq" ml⁻¹

In Table B the data relate to whole body exercise and the assumed maximal capacities are 6 · l min⁻¹ in maximal oxygen uptake and 18 L "O₂ eq" ml⁻¹.

A (Running or bicycling)					
Time, min	1/2	1	2	4	10
Energy yield anaerobic ML "O ₂ eq" kg ⁻¹ (%)	60 (75)	90 (64)	100 (48)	90 (26)	90 (10)
Aerobic ML "O ₂ eq" kg ⁻¹ (%)	20 (25)	50 (36)	110 (52)	260 (74)	770 (90)

B (Rowing or swimming)					
Time, min	1/2	1	2	4	6
Energy yield anaerobic l "O ₂ eq"; (%)	9 (82)	16 (76)	18 (62)	18 (43)	18 (33)
Aerobic l "O ₂ eq"; (%)	2 (18)	5 (24)	11 (38)	24 (57)	36 (67)

Training of the anaerobic capacity

From the above it may be valid to conclude that the training of the anaerobic energy yield can be divided in 1) elevate the maximum rate of glycolysis, i.e. to be able in a minimum of time to utilize the whole oxygen deficit, and 2) improve the anaerobic capacity, i.e. to tolerate the lactate accumulation.

Rate of glycolysis: It must be emphasized that the knowledge in this particular topic is almost non-existing. That does not mean that the basic mechanisms in the regulation of the rate of the glycolysis are not understood, rather the limitation is a lack of knowledge about the actual changes during extreme exercise of the various factors which are known to activate the glycolysis in muscle of man (figure 8).

Basically there are three conditions, which may govern an elevated rate of glycolysis, the content of glycogen, glycolytic enzymes and activation of key enzyme activators. Proper diet and training affect the muscle glycogen content. The glycolytic enzymes are found at very high level in muscle of man and only a partial activation of the enzymes cause a very rapid rate of glycolysis and lactate production. Very intense training with high speed has been shown to produce a further increase in the glycolytic enzy-

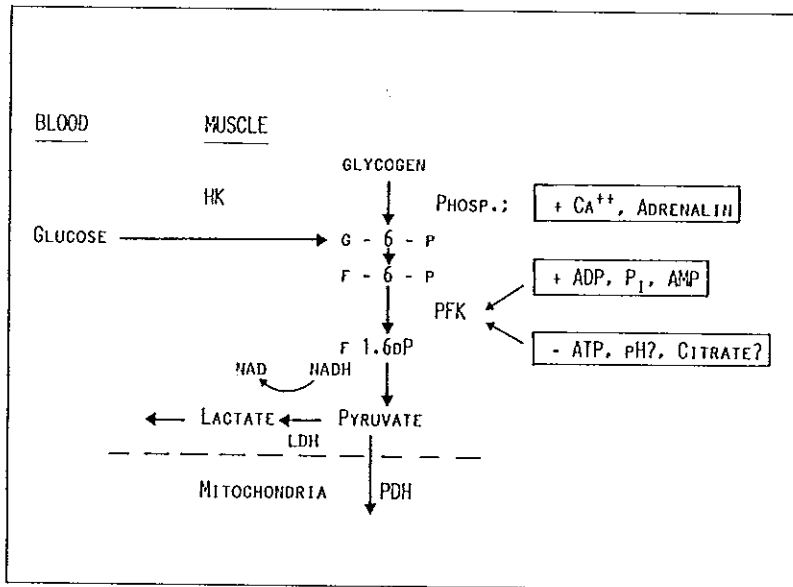


Figure 8. Some of the principle steps in glycolysis with an indication of factors activating or inhibiting some of the regulatory enzymes. A plus (+) indicates activation and a minus (-) inhibition. Phosphorylase is then activated by an elevated free intracellular Ca^{++} , but adrenalin can via its 2:nd messenger cyclic AMP add to this activation. A low ATP and an elevated ADP concentration are the key factors to activate the enzyme phosphofructokinase (PFK).

mes. The importance of such a change is that a smaller alteration in an activator of one of the regulatory enzymes will result in a larger flux through that particular pathway. Further inhibitors of that enzyme will have less effect.

Whether the activation of the key enzyme can be "trained" is unknown. If they are it is most likely that also in this case very high speed would be most favourable, as alterations probably are the largest at the very highest rates of contraction. Of special interest in this respect is that the sympathetic nervous system may be involved. Newsholme (1983) has gone the furthest in such speculations postulating that a rapid onset of the glycolysis depends upon the existence and right training of futile cycles which are regulated by the sympathetic nervous system (figure 9). If such a system operates in the muscle of man, can it then be tuned?

Fate of the lactate produced. The other aspect of glycolysis is that as the rate of pyruvate and NADH production in intense exercise surpass the capacity of the mitochondria to handle all that is formed, lactate is accumulated in the muscle. Of note is that the content of the LDH₄₋₅ fraction of the enzyme primarily catalysing the reaction is found in

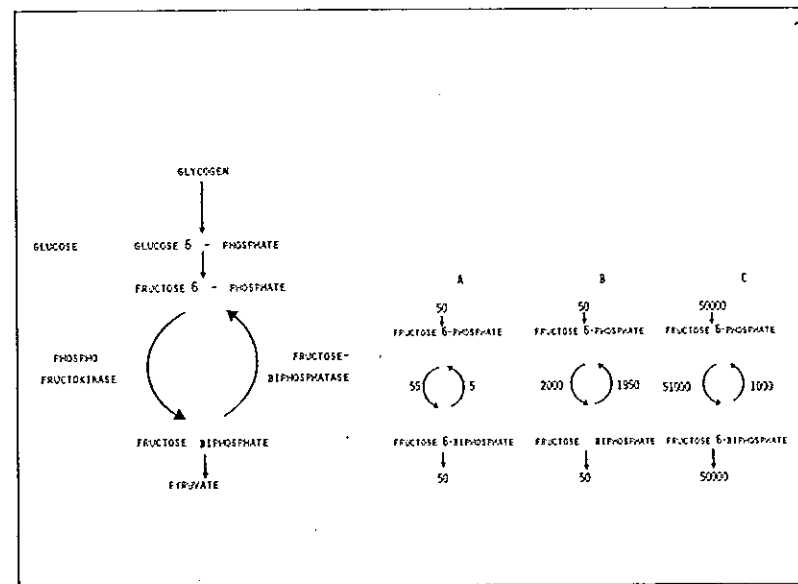


Figure 9. A tentative example of where a substrate cycle may operate according to Newsholme and Leech (1983). He proposes that the well controlled reaction from 6-phosphofructokinase to fructose biphosphate is a site for "cycling", i.e. some of the formed fructose biphosphate is transformed to 6-phosphofructokinase catalyzed by the enzyme fructose biphosphatase. The advantage with this coupling is that you can increase the precision in the rate control and also more quickly increase the rate of the glycolysis when needed, as for example at the start of a 100 m race. Such an example is illustrated here with

- resting before race
- on starting block
- after 6 sec sprint

The cycling rates indicated are speculative. In (b) the rate of cycling is raised by the influence of the sympathetic nervous system, by which a 25 fold increase in the rate of 6-phosphofructokinase and a 50% decrease in that of fructose biphosphatase causes a 1000 fold increase in flux.

very high amounts in muscle of man in all fiber types. Lactate dissociates and will affect the pH of the muscle. A certain drop in pH can occur without too much disturbances of basic processes in the muscle but there is a definite lower limit. Presently this lower limit is not known. With the nuclear magnetic resonance (NMR) technique a pH below of 6.0 has been reported in skeletal muscle of man at exhaustion, whereas with the more conventional methods pH values below 6.4-6.5 seldom are found (Sahlin, 1978; Sjøgaard et al., 1985). It is likely that training would result in the ability to withstand slightly lower pH (more enzymes would contribute to this), but just as impor-

tant would be if the buffering capacity of the muscle is increased and if a large fraction of the formed lactate can leave the muscle fiber in which it has been produced. To deal with the various possibilities in a systematic manner the more important factors are listed in table 4.

Table 4.
Fate of lactate produced in skeletal muscle during exhaustive exercise.

Event	Limiting factor
Accumulation in the muscle fiber	Buffer capacity
Transport to adjacent muscle fibers (FT → ST fibers)	Own lactate formation LDH ₁₋₂ mitochondrial capacity
Transport to interstitial space and blood	Capillary density muscle perfusion uptake by other tissues

From this list it is apparent that in addition to lactate being accumulated in the muscle some of the lactate escape to adjacent muscle fibers and some to the interstitial space and the blood stream. The relative magnitude of these possibilities can only very roughly be estimated. Medbø et al. (1968) calculated that 11% of total oxygen deficit out of $66 \text{ ml} \times \text{kg}^{-1}$ could be ascribed to lactate transferred to the blood (ECF) which was $\frac{1}{4}$ of the lactate produced (Table 1). Such estimates can be made from data from two minutes of exercise. An increase in muscle lactate of 15 up to 20–25 $\text{mmoles} \times \text{kg}^{-1}$ may occur in sedentary and sprint trained individuals, respectively. In the same time periods some 15–30 mmoles of lactate may be released from the exercising legs (Wahren et al., 1974). If it is assumed that the lactate release comes from a muscle mass of 10–12 kg approximately 1.5–3.0 mmoles of lactate has left each kg of muscle, or only 10% of the lactate produced. This value is a factor of 3 lower than the one given by Medbø et al. (1986). They based their calculations on increase in blood lactate and assumed that it was in equilibrium with the ECF of the whole body. That may not necessarily be the case in short exercise periods with a very rapid increase in the blood lactate concentration. Of note is, however, that even in the more prolonged exhaustive exercise the same oxygen deficit is obtained, supporting the notion that the lactate released from the contracting muscles to the blood constitutes only a small fraction of the oxygen deficit.

In view of Brooks and associates (1985) data postulating that a large turn-over of lactate occurs in the body during exercise one would expect to find a greater release of lactate from the muscles. As this appears not to be the case it must be concluded that lactate is transferred from the muscles to the blood during short-term exhaustive work but the amount released is a rather small fraction of what is produced. Further the lactate that is released can account for the elevation in blood lactate concentration observed at the end of the exercise but the amount is hardly large enough to be distributed evenly throughout the whole ECF. It is noteworthy that the individual variation is large and the subjects involved in the experiments were far from especially trained. It is possible then that the above given value for lactate released during the exercise could

be larger, as the result of training. A larger gradient for lactate between muscle and blood, more capillaries in muscle and a higher muscle perfusion would contribute to this. Medbø et al. (1986) did not discuss the possibility that some lactate is transported to adjacent muscle fibers in the same muscle and not only accumulated there but also is metabolized. This may not be a reasonable assumption in sedentary subjects but in endurance trained muscles ST fibers may be able to convert the lactate to pyruvate and oxidize it with a rate that is not only of theoretical interest. From this account it is apparent that, although some lactate may escape from the contracting muscles during the exhaustive exercise, the fraction that remains in the muscle represents by far the largest portion. Questions are then related to which consequences this lactate accumulation has for the function of the muscle cell. As mentioned above, the pH will drop, but it appears that the buffer capacity of muscle can be altered, i.e. in sprint type trained muscle a given concentration of H^+ (the lactate is dissociated) causes a slightly less marked reduction in pH (Sahlin and Henriksson, 1985). Such alterations in the buffer capacity of a muscle have been demonstrated to occur in young horses undergoing training as well as in man (Sharp et al., 1986; Fox et al., 1986). It is believed that some

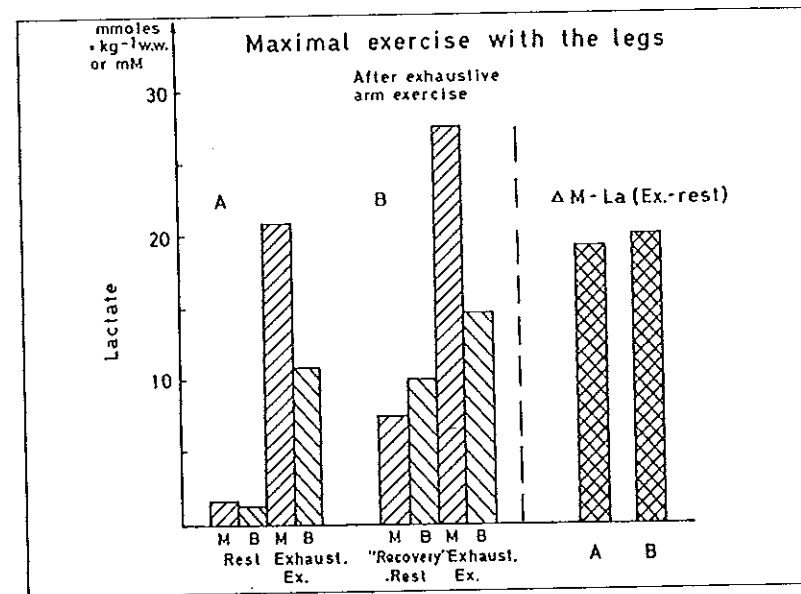


Figure 10.
Muscle and blood lactate in experiments where the subjects first exercise with their arms to exhaustion. After ten minutes of rest when some of the lactate is transferred to the blood and further to other tissues such as the leg muscles, maximal exercise is performed with the legs. In spite of higher initial leg muscle lactate concentration the rate of lactate formation during the exhausting leg work is the same as in control experiments demonstrating that although lactate concentration is high the rate of lactate accumulated is constant (Knuttgen et al., 1973).

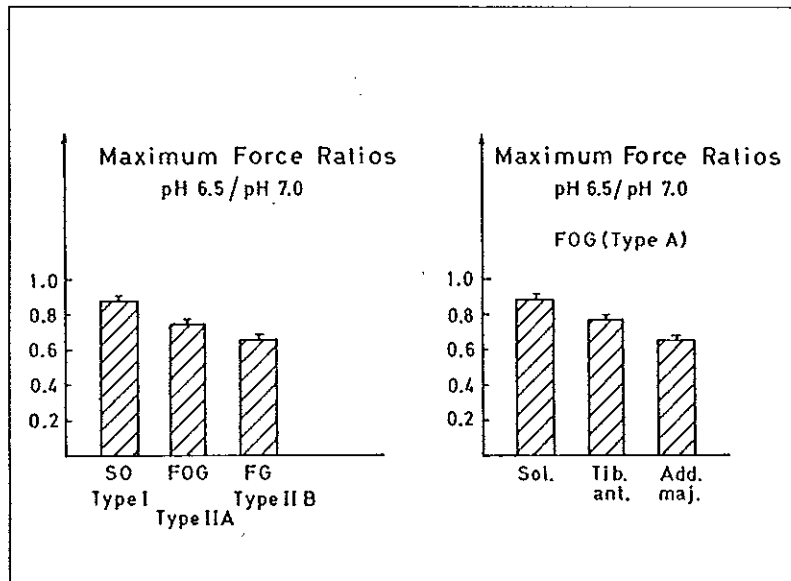


Figure 11. Results from experiments where the free Ca^{++} concentration in the muscle (rat) could be controlled as well as the pH. The force generated by the muscle fibers was reduced at a given Ca^{++} concentration when pH was 6.5, and more so in the fast twitch than slow twitch fibers (Donaldsson, 1983).

proteins (amino acids and / or peptides) in the muscle are increased, which have the property of buffering H^+ . Although the buffering can be improved, lactate is accumulated and causes the pH to become reduced. The enzymes catalyzing the various metabolic reactions in the muscle have a pH optimum. It has been postulated that the pH should become so low that the enzymes in the glycolytic pathway are inhibited, with the result that the rate of the glycolysis would be retarded (Bergström and Hultman, 1973). However, as depicted in figure 10, high muscle lactate concentration does not affect the rate by which lactate is formed. Instead, the reason for the reduced force development is likely to be that the low pH reduced the affinity of the contractile proteins for Ca^{++} (figure 11).

Practical guidelines. From the above discussion it should be clear that there may be two modes of training to improve anaerobic capacity (Table 5). One to obtain highest possible rate of the glycolysis, and the other is to tolerate and handle the lactate produced. Optimal training pattern may be different for these two variables. Speed or intensity are essential elements for the training of rate of glycolysis. Duration of the effort cannot, however, be too short as the energy costs during the first 5–10 seconds of the exercise to a large extent will be covered by aerobic means and the reduction of the phosphagens. Although glycolysis is turned on from the very first second of the exerci-

se its maximum rate may not be reached until 5–15 seconds of exercise. The length is limited by how long the high exercise intensity can be maintained. Periods of 30–40 seconds may be the longest. These should be repeated 3 or more times. If the rest between work bouts is long (≥ 5 min) then the speed in each work bout may be maintained longer than if the rest period is made short. Either way would do, as long as work intensity is not reduced and the duration of each work bout is longer than 15 sec.

Table 5. General principles for training of anaerobic capacity.

I. Training to elevate glycolytic enzymes in skeletal muscle.

Speed	Duration	Repetitions
Highest possible	30 – 40 sec	3 → 5 → 8

II. Training to improve buffering capacity of skeletal muscle.

Speed	Duration	Repetitions
a. Close to top speed max. hr.	1,5 → 2,0 → min	2 – 4 – (6) long rest: 10 min.
b. Top speed	30 – 40 sec.	3 – 5 – 7 short rest: 1–2 min.

To continue to exercise with high intensity is the important point in the training to tolerate the lactate. Again is high speed required. The duration should be larger than 45 seconds but just as important is that when it is difficult to maintain speed one should continue with highest possible intensity another 5, 10 to 15 seconds. Long or short rest periods can be used between work bouts. In the latter case the duration of the effort will quickly be shortened, while with longer rest periods the duration of the exercise can be better maintained. Number of repetitions could be 3 or more.

One aspect of this training which is common to both variants is that the adaptation taking place, which is the basis for the improvement, is local, i.e. enzyme levels only increase in muscle fibers engaged in the exercise. The same is of course true for the buffer capacity. Thus, this training should be performed in movements identical to or very similar to those in actual competitions.

In all anaerobic training it is of value to have active rest, i.e. low intensity exercise not exceeding a work intensity demanding above 60% of the maximal oxygen uptake (figure 12). Lactate is then quicker transferred from the fatigued muscles because the perfusion of the muscle is maintained at a higher level than at rest. A prerequisite is that the exercise intensity is low enough not to cause further lactate formation.

Measurements of anaerobic work performance: The above emphasized specificity in the training of the anaerobic energy yield relates also to the testing of this capacity. A

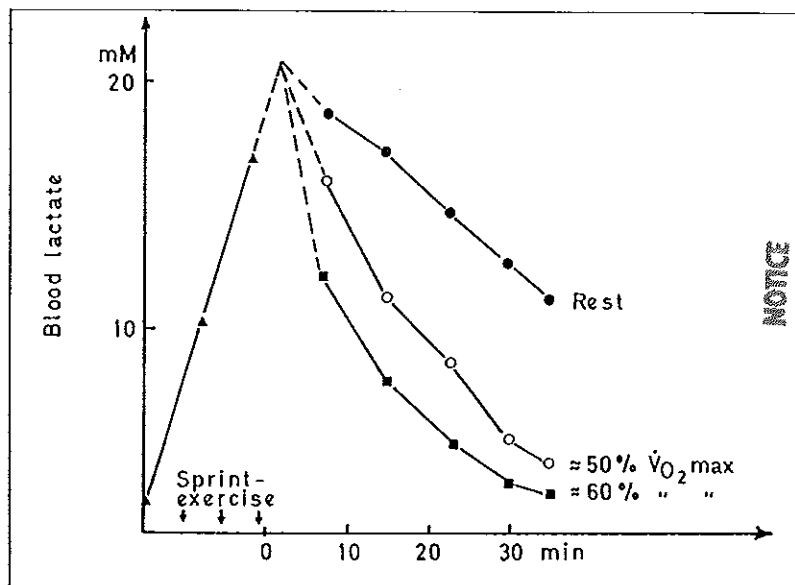


Figure 12. Blood lactate in recovery from sprint exercises, when light or moderate exercise is performed in the recovery period (modified from Hermansen and Stensvold, 1971). Even faster return to pre-exercise blood lactate response could be noticed at relative work loads up to 80% of maximal oxygen uptake in endurance trained individuals.

runner should be tested on the treadmill or on a track. It is of importance that the running is on the flat, as the relative stress on the various muscles is different running uphill. A cyclist should pedal the bicycle, a rower should row etc. The length of the test should be well above one minute and may be close to two or three minutes. The idea that a test for anaerobic capacity should only last 30–40 sec is not well founded. The argument for a short test is that the aerobic energy yield then only covers a small fraction of the total energy expenditure. However, that helps little when such a small part of the maximal oxygen deficit has been utilized. If such a short test is used it is *not a measure of anaerobic capacity*, but may be a measure of how fast the alactacid part of the oxygen deficit and the rate of glycolysis can deliver energy.

Training at altitude: Reduced availability of oxygen as at altitude could be advantageous in the training of the anaerobic energy yield as it may favour glycolysis and lactate formation. Recently this has been shown to be the case. There are indications that the buffering capacity of the muscle does increase when the training was performed at altitude (1,500–2,700 m.a.s.l.). Interestingly, this adaptation took place although the training was not geared towards anaerobic training, but rather to improve technique in c.c. skiing and aerobic capacity. However a maximal oxygen uptake test was performed

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on each subject each day at altitude. With the larger buffer capacity followed a larger maximal oxygen deficit and improved short term performance. Of note was that maximal oxygen uptake was unaltered (see below).

The glycolytic enzymes were not elevated, but no very high intensity training was included. Theoretically, it appears that altitude training ought to be effective in this regard as well, but in a recent study, when high intensity training was performed at altitude (low pressure chamber $\sim 2,300$ m.a.s.l.), the glycolytic enzymes studied were reduced (Terrados, personal communication).

Time for an adaptation to occur: The point to consider is how long time is needed for a significant change in anaerobic capacity to occur. Sad to say no such measurements are really available in man. With an optimal stimulus (very high intensity) a change in the enzyme level may occur after some few days or a week (Figure 13). After 2–4 weeks one can assume that the adaptation that can be obtained has been achieved. Less is known about the buffer capacity. The fact that two weeks of training at altitude was suf-

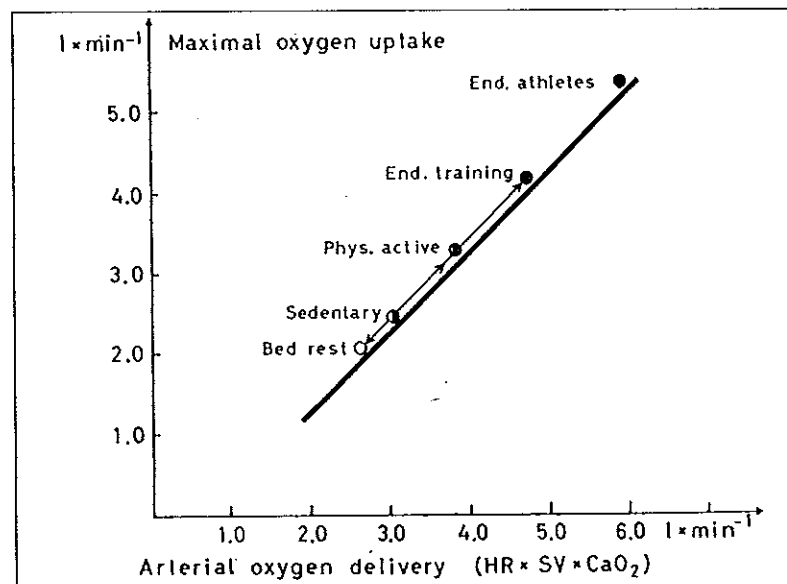


Figure 13. Schematic summary of studies where the volume of oxygen offered to the tissues of the body during maximal exercise is related to the observed maximal oxygen uptake. The O_2 volume offered is a function of arterial oxygen content (CaO_2), maximal heart rate and stroke volume. Of these, only the stroke volume is more markedly affected by the degree of physical activity, and thus the variation on the x-axis is almost exclusively related to the stroke volume. These data are taken from studies by Saltin et al., (1968), Ekblom, (1968) and Ekblom-Hermansen (1968).

ficient to increase the buffer capacity indicate that also this variable can vary with a rather short time-constant, however, it is definitely longer than for the glycolytic enzymes.

Aerobic capacity

In contrast to the anaerobic capacity the aerobic fitness has been studied extensively, with the first more systematic investigations performed by Robinson and associates on sedentary and trained subjects as well as endurance athletes (1937, 1938). Methodological problems are small when assessing a person's maximal oxygen uptake. This statement may be surprising in view of the lengthy discussion related to the value of bicycle vs treadmill exercise and the critical role of the magnitude of the muscle mass involved in the exercise. This debate has been on-going for many decades and persists today. As discussed below the basis for using two-legged exercise to establish an individual's aerobic capacity is well founded and although bicycle exercise may give some few percent lower values than treadmill exercise, the observed values closely reflect the aerobic capacity of the subject.

Maximal oxygen uptake in endurance athletes. The highest reported values are around $90 \text{ ml} \times \text{kg}^{-1} \times \text{min}^{-1}$ for males and just above $75 \text{ ml} \times \text{kg}^{-1} \times \text{min}^{-1}$ among female endurance athletes. In the respective sex values down to 75 and $65 \text{ ml} \times \text{kg}^{-1} \times \text{min}^{-1}$ are common to find among successful athletes in endurance events. Sedentary young adult men have close to $50 \text{ ml} \times \text{kg}^{-1} \times \text{min}^{-1}$ in maximal oxygen uptake and their female counterpart closer to $40 \text{ ml} \times \text{kg}^{-1} \times \text{min}^{-1}$. Thus, for each sex a close to two-fold difference exist between sedentary and endurance trained individuals. What is not known is to what extent a sedentary person can elevate his maximal oxygen uptake by extreme training. Most sedentary people will improve 20–30 percent may be more but only a very small minority will achieve values above 60 () or 70 () $\text{ml} \times \text{kg}^{-1} \times \text{min}^{-1}$.

What limits maximal oxygen uptake: The question what limits maximal oxygen uptake has been addressed since the first measurements were performed in the 1920's. A central factor was thought to be the most likely candidate limiting maximal oxygen uptake of man. Support for this view was obtained from elaborate studies in the 1950's and 1960's (for ref., see: Bevegård and Shephers, 1967; Rowell, 1974). Further, longitudinal studies with training and detraining revealed a close relationship between changes in the performance of the heart and maximal oxygen uptake (figure 13). Later a similar conclusion was reached by Ekblom and associates (1972, 1976) based on results from studies showing that reinfusion of red cells momentarily increased maximal oxygen uptake, a finding similar to the one observed when oxygen enriched gas mixtures are inhaled during maximal exercise (Nielsen, 1937; Margaria et al., 1961).

The idea that the central circulation limits maximal oxygen uptake in man and that all the oxygen offered to the exercising skeletal muscle could be consumed was challenged by a series of investigations in the late 1960's and early 1970's. Kaijser (1970) was unable to demonstrate an increase in maximal oxygen uptake in subjects breathing air at 10 atmospheres. He concluded that the extra oxygen delivered to skeletal muscles could not be utilized because of a limitation in the muscle, that is, the capacity of the respiratory chain was saturated. Further work from Holloszy (1967), and later from ot-

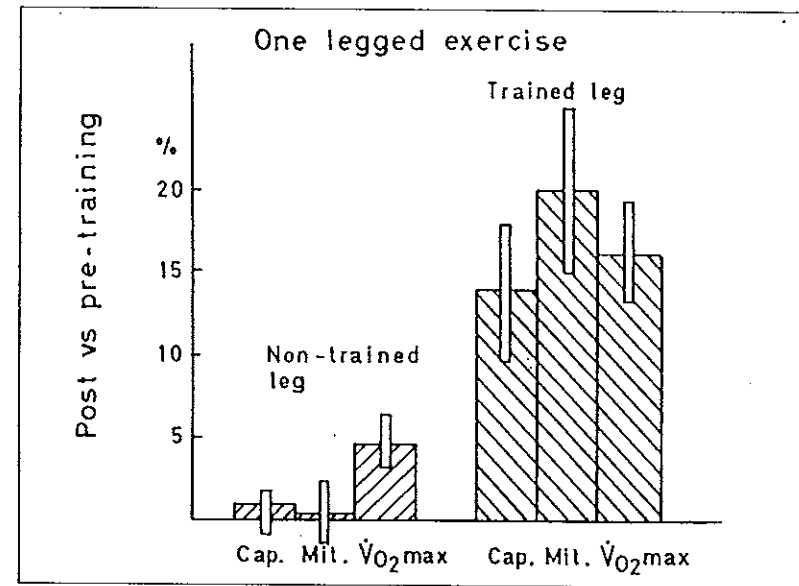


Figure 14. Muscle adaptations to one-legged training. Depicted are relative changes in the untrained and the trained leg for muscle capillarization, mitochondrial enzyme activity (*vastus lateralis*) and peak oxygen uptake during one-legged short-term exercise (data are from Saltin et al., 1976, Henniksson, 1977).

hers (for ref., see: Saltin and Gollnick, 1983), confirmed that the mitochondrial enzyme activity of skeletal muscle increased during training. Chronic and enhanced use of muscles for many months results in a 2- to 3-fold increase in mitochondrial enzyme activities, and it was believed by some that this elevation had significant importance for the rate at which oxygen could be utilized (Hoppeler et al., 1985).

The strongest support for a peripheral circulatory and metabolic limitation was obtained when 1-legged training studies revealed that, when exercised, the trained leg could reach a peak oxygen consumption rate that could not be attained when the untrained leg was used. In the trained leg, in contrast to the non-trained leg, capillaries proliferated and mitochondrial enzymes increased (figure 14).

Several others who have worked in the area of possible limitations of maximal oxygen uptake have the notion that the links in the transport of oxygen from the ocean of oxygen we live in to its ultimate function as a hydrogen acceptor in the respiratory chain of the mitochondria are so closely linked and matched that one factor cannot be singled out as limiting (Holmgren and Åstrand, 1966), a view recently reiterated and elaborated upon by di Prampero (1985).

To pinpoint one single factor limiting maximal aerobic power on a cellular – subcellular – or molecular level can hardly be done today. The question is, however, whether or not data are available on a organ level to support the concept that central (lungs – heart) rather than peripheral (vascular tree, muscle aerobic potential) factors are limiting.

In the past the lungs have been excluded as a limiting factor for maximal oxygen uptake at sea level as oxygen saturation with few exceptions has always been found to be above 90% at peak exercise in sedentary subjects as well as in champion endurance athletes (Åstrand et al., 1964; Ekblom and Hermansen, 1968). Recent reports, however, have convincingly shown that in some endurance athletes desaturation of arterial blood may occur during exercise at sea level (Dempsey et al., 1984; Terrados et al., 1985). This is in part due to a reduced ventilatory drive, but it is also most likely a demonstration of less adaptability of the lungs, including its diffusing capacity, to endurance training as compared to the plasticity of the cardiovascular system or the skeletal muscles. In untrained man and most endurance trained individuals the maximal capacity of the lung to transfer oxygen is far from taxed during exhaustive exercise eliciting maximal oxygen uptake.

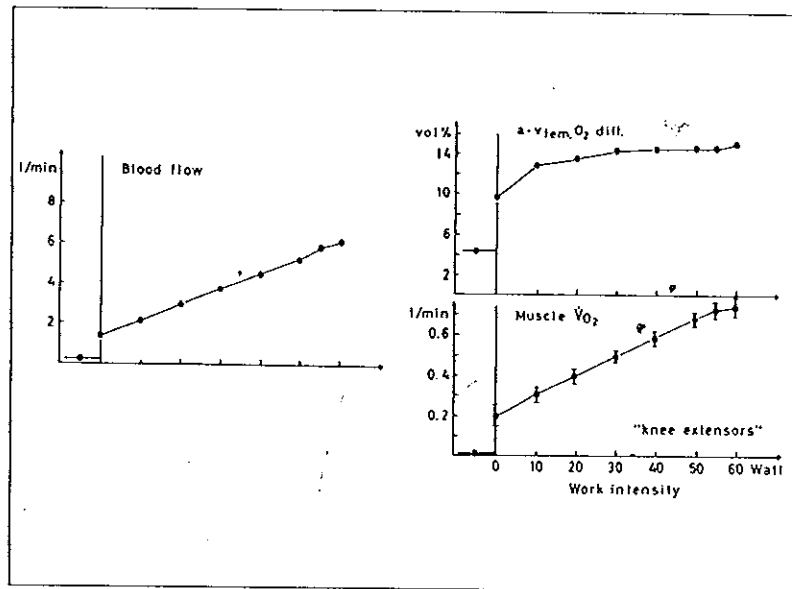


Figure 15.

From above are given the thigh blood flow, the a-v oxygen difference over the exercising limb and the oxygen uptake of the knee-extensor muscle at rest and at various work rates including one exhausting the subjects in 4–6 minutes. The work is performed with the knee-extensors of one-leg by «kicking». The ankle is coupled via a bar to an ordinary Krogh bicycle ergometer. In no-load exercise the lower part of the limb is lifted (weight 3 kg?) which explains the high values at 0 watt.

A series of experiments has been performed in an attempt to differentiate between central cardiovascular and peripheral factors limiting maximal oxygen uptake, transfer, and utilization. The capacity of the heart to supply the body with a blood flow is well established (for ref. see Blomqvist and Saltin, 1983). What is lacking is the knowledge of the true capacity of the skeletal muscle to accommodate a high blood flow and its peak oxygen uptake.

To obtain such data an exercise model had to be established in which the muscle mass performing the contraction was well defined and the weight of which was possible to estimate. This was accomplished using the knee-extensors of one leg for the exercise, measuring flow in the femoral (iliac) vein with a cuff inflated just below the knee (Andersen and Saltin, 1985). In contrast to earlier reports on muscle blood flow and exercise (Klausen, 1976), blood flow in the femoral vein increased linearly with work intensity and in sedentary subjects it reached $5-7 \text{ l} \times \text{min}^{-1}$ at a work load which exhausted the knee-extensors of the exercising limb within 5–6 minutes of work (figure 15).

Oxygen uptake by the knee-extensors also increased linearly with work load and was $0.6-0.9 \text{ l} \times \text{min}^{-1}$ depending upon the magnitude of the work load at exhaustion. The mass of the knee-extensors was 2–3 kg, which, when allowing for 10% of the observed blood flow as coming from parts other than the knee-extensors (30), gave a perfusion of $200-250 \text{ ml} \times \text{min}^{-1}$. This value surpasses earlier estimates by a factor of 2 or more (for ref., see: Mellander, 1981).

These data indicate that, if most of the muscle mass of the body was intensely engaged in the exercise, the heart would have to pump $70 \text{ l} \times \text{min}^{-1}$ or more to avoid a drop in blood pressure (figure 16). Such a high cardiac output has never been reported in man, not even in champion endurance athletes (Ekblom and Hermansen, 1968). Making the same calculations for oxygen uptake, it is obvious that the 30–35 kg of skeletal muscle of a 70 kg man would be able to consume $9-10 \text{ l} \text{ O}_2 \times \text{min}^{-1}$ or $110-130 \text{ ml} \times \text{kg}^{-1} \times \text{min}^{-1}$, values close to double those reported for endurance athletes and 3–4 times higher than what is observed in sedentary man. Consequently, the performance of the heart sets an upper limit to the cardiac output and thus to the maximum oxygen uptake of man.

Comparisons with other species. In a way it can be said that man is not really designed for exercise using both upper and lower extremities simultaneously, at least not from the standpoint of the capacity of the heart as a pump. The system functions with the baroreceptors serving as the safety feed back. The sympathetic vasoconstrictor activity overrides any metabolic factors causing local vasodilatation, when the pump capacity of the heart is surpassed by the needs of the contracting muscles. The role of the sympathetic system is increased in relation to the fraction of the muscle mass involved in the exercise.

Man probably differs from other species by having a low central circulatory capacity in relation to the size of vascular beds of the muscles. This is verified by the fact that man has a lower heart volume/body size ratio than other mammals using all four extremities in locomotion and dependant upon their endurance capacity to survive. In man the weight of the heart is only 0.3–0.35% of the body weight, whereas it approaches or

Muscle mass		Muscle blood flow		Flow to non-exercising tissue		Cardiac output
kg		$l \cdot kg^{-1} \cdot min^{-1}$		$l \cdot min^{-1}$		$l \cdot min^{-1}$
30	×	2.2	+	4	=	70
30	×	0.33	+	0.2	=	10.1
kg		$l \cdot kg^{-1} \cdot min^{-1}$		$l \cdot min^{-1}$		$l \cdot min^{-1}$
Muscle mass		Muscle oxygen uptake		Oxygen uptake non-exercising tissue		Maximal oxygen uptake

Figure 16.

In this graph theoretical values for maximal cardiac output and oxygen uptake in man are obtained based on the observed values for leg muscle blood flow and oxygen uptake per kg of muscle performing maximal knee-extensor exercise. Estimations are based on including 30 kg of muscle mass in the exercise as in rowing and that the heart can honour the demands of the muscles. For discussion see text.

surpasses 1% in dogs and horses (Table 6). The latter two species also have 2–3 times larger maximal oxygen uptakes ($100\text{--}160 \text{ ml} \times \text{kg}^{-1} \times \text{min}^{-1}$). Thus in these species there is a more equal balance between the capacity of the heart to supply with a flow and oxygen and the skeletal muscle to accommodate the flow and utilize the oxygen. This difference between species should be viewed in the light of the mass of muscle engaged in normal locomotion of the particular species. Man moving in the upright posture has adapted to only use part of the muscle mass for locomotion with the result that also the cardiovascular dimensions are scaled to only support a share of the muscles when exercising maximally.

With this in mind, it would be interesting to estimate the mass of muscle exercising intensely that can be supplied with a "sufficient" blood flow in man. Using the present data, it appears that in sedentary man having a maximal cardiac output of 18–22 l/min , 7–9 kg of muscle actively working could tax the capacity of the heart. Another way of expressing this would be to say that $\frac{1}{4}$ – $\frac{1}{3}$ of the total muscle mass of a sedentary person can consume $2.5\text{--}4.0 l \times \text{min}^{-1}$ of oxygen, or the equivalent to the maximal oxygen uptake of a sedentary person. In accordance with these estimates is the well-known fact that maximal oxygen uptake of a person is achieved in two-legged exercise (figure 17).

Table 6.

Heart weight to body weight ratios and maximal oxygen uptake of some species. Data are from Saltin – Åstrand, 1967 and Grande-Taylor 1965 (Man), Mackie Terjung, 1983, Grande-Taylor 1965, Gleeson, Baldwin, 1981 (Rat), Grande-Taylor 1965, Borger et al. 1956, Musch et al. 1985 (Dog) and R. Rose, personal communication (horse).

	Heart/body weights ratio %	Maximal oxygen uptake $ml/kg \times min^{-1}$
Man:	0.3 – 0.4	40 – 80
Rat. lean:	0.5 – 0.6	70 – 90
Dog:	0.7 – 0.8	90 – 130
Horse:	0.8 – 1.0	100 – 160

In an endurance athlete having a cardiac output of 30–40 $l \times \text{min}^{-1}$ either a larger muscle mass can be perfused reasonably well as in rowing or swimming or if only the legs are intensely involved in the exercise as in bicycling or running they will obtain a larger flow. What should be apparent is that maximal vasodilatation cannot occur in all

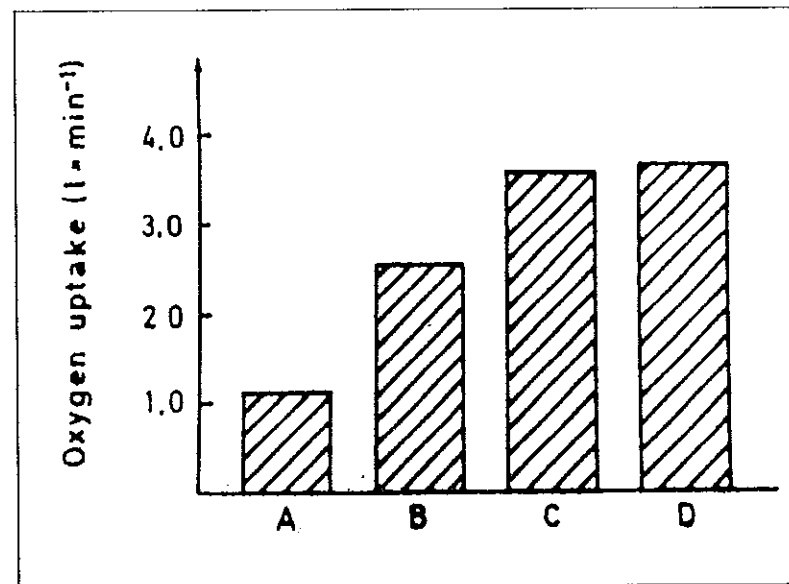


Figure 17.

Peak oxygen uptake when performing short-term exhaustive exercise with various muscle groups going from one-arm to whole body exercise. The values for muscle mass are rough estimates. Adapted from data by Lewis et al. (1983), Hermansen (1973), Saltin et al. (1976), Saltin and Hermansen (1969), Andersen and Saltin (1985).

muscular beds at the same time maintaining normal blood pressure. If more muscle mass is involved in the exercise vasoconstriction must occur in vessels feeding contracting skeletal muscles. Indeed this has been observed when arm work is added to leg exercise or when during one-legged exercise the other leg is included in the exercise (Secher et al., 1977; Klausen et al., 1982).

Local muscle adaptation (capillaries – oxidative enzymes) with endurance training, functional significance?: Endurance training causes a proliferation of capillaries and elevation of the content of mitochondrial enzymes. It was first believed that these adaptations were a necessity for raising maximal oxygen uptake. As this appears not to be the case, the pertinent question is what role these alterations in the muscle may have? The increased number of capillaries elevate capillary blood volume, which in turn means that at the same muscle blood flow mean transit time is lengthened ($MTT = \text{capillary blood volume} / \text{muscle blood flow}$). This is not the only positive effect of more capillaries. As the capillary surface area is enlarged and if the increase in capillarization is larger than any increase in muscle fiber size, the area for diffusion is also smaller. All these factors contribute to improve the conditions of exchange between blood and muscle and will have some bearing on the extraction of oxygen, but its largest role is probably making it possible to elevate the uptake of substrates (especially FFA) from the blood stream (figure 18).

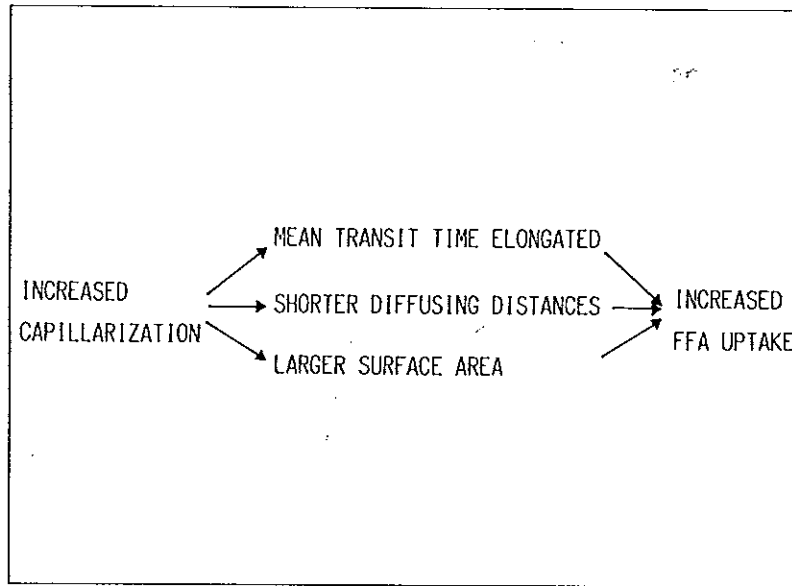


Figure 18. An increased number of capillaries in the muscles affect several conditions of significance for an exchange over the capillary wall. In the figure is highlighted the effect of the FFA uptake from the blood stream, but gas, substrate and metabolite exchange are also affected by muscle capillarinity.

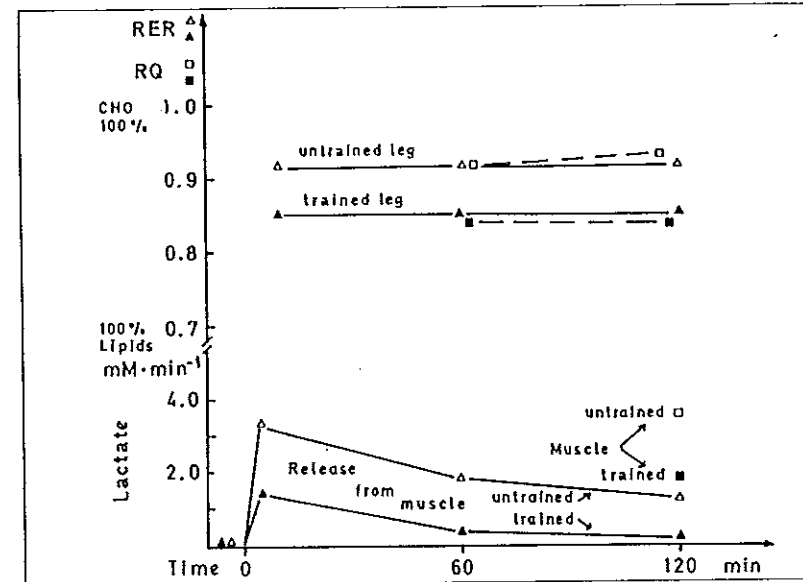


Figure 19. Illustrated is the marked effect endurance training of skeletal muscle has on the RQ (respiratory quotient) determined over the exercising limb and RER (respiratory exchange ratio). Further is illustrated that muscle lactate and lactate release are also lower in the trained leg therefore the production of lactate is clearly less. The exercise performed is by the knee-extensor of one leg (trained or untrained). Modified from data by Kiens et al. (1986).

With endurance training the metabolism during exercise is markedly altered. Respiratory exchange ratios (RER) are lower at a given and also at the same relative work load after endurance training, and the lactate production in muscle is reduced (figure 19) which is reflected in lower lactate level at a given absolute, but also at the same relative work load in the very well trained endurance athlete. This knowledge is not new. In the 1930's Christensen (1931) and Bang (1936) (figure 20) made similar observations. What is new is that we better understand how it is brought about. Table 7 presents in summary some of the factors which bring about the more efficient use of lipids as substrates for the muscle and concomitantly spares the glycogen break down. Of note is that more lipids or oxygen are not really offered to the trained muscles rather it is the "quality" adaptations within the muscles which makes them superior in gearing the substrate choice from CHO to lipids and further saving the limited glycogen stores by a reduced rate of lactate formation. This is accomplished not only by careful control of the rate of glycolysis but also by reducing the LDH_{4-5 (m)} isozyme, increasing the LDH_{1-2 (H)} isozyme and increasing the capacity of one of the shuttle systems for NADH (Shantz et al., 1985) as well as enlarging the mitochondrial volume (figure 21). The over all effects of these metabolic regulations with enhanced oxidation of lipids and sparing of glycogen are exemplified in Table 8.

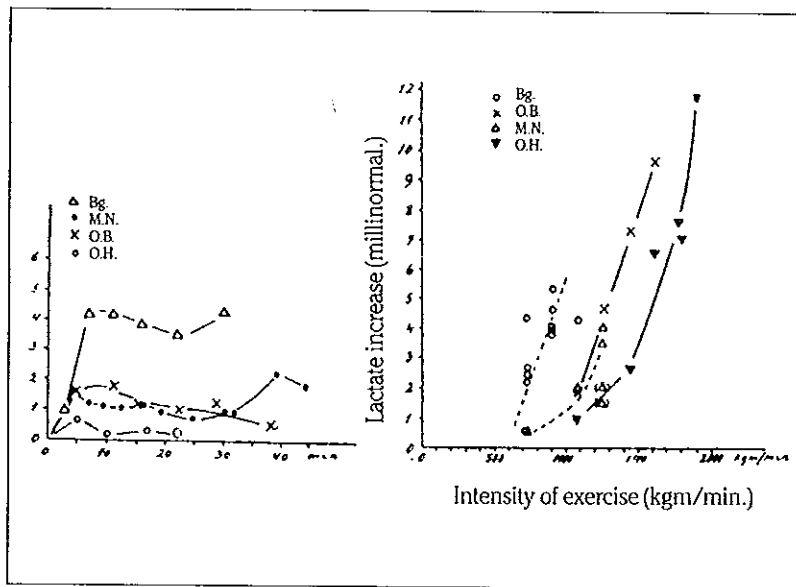


Figure 20. This is the data that Bang published (1936) demonstrating that at the same work load blood lactate varied between subjects in relating to their training status. To the right data on the blood lactate concentrations at various work intensities are depicted for the same subjects. Note that today this mode of presentation has become very common.

Table 7. List of factors which have a bearing on the regulation of various steps of the muscle metabolic pathways. A comparison is made between endurance trained and untrained muscles. A high ADP/ATP ratio activates phosphofructokinase (PFK). High concentrations of fatty acids inhibit PFK and in the mitochondria pyruvate dehydrogenase, whereas the β -oxidation is increased. A high citrate concentration has been proposed to inhibit PFK.

Variable	MUSCLE		Effect
	Trained	Untrained	
ADP/ATP	Low	High	Activates PFK
Cytoplasmatic FA	High	Low	Inhibits PFK?
Mitochondrial FA	High	Low	a) Inhibits PDH b) Increases flux through β -oxidation
Cytoplasmatic citrate	High	Low	Inhibits PFK

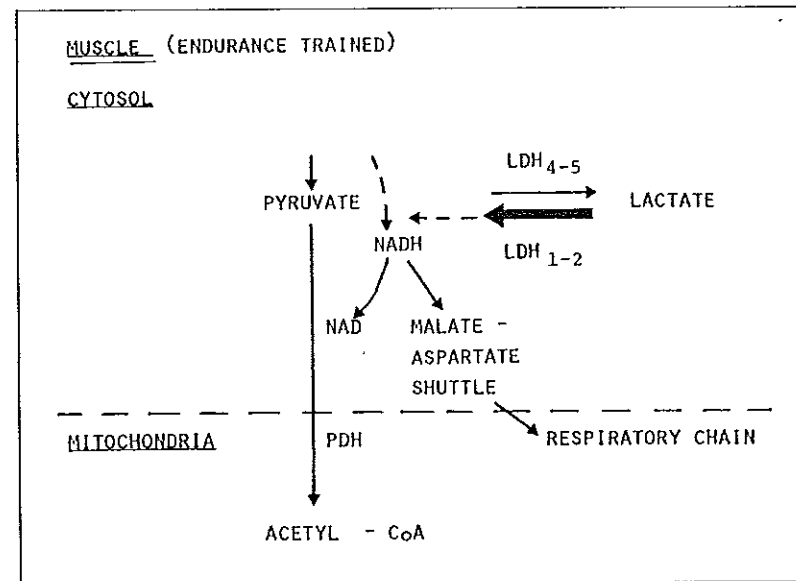


Figure 21. A schematic illustration of the important adaptation which occurs in the skeletal muscle with endurance training in regard to LDH isozymes and enzymes in the malate - aspartate shuttle. The two alterations together with an enlarged mitochondrial surface area bring about a minimum of lactate production in endurance trained muscles. In fact these trained muscles have a good capacity to convert lactate to pyruvate.

Test of aerobic performance: A measure of maximal oxygen uptake gives a value of the pump capacity of the heart if large enough muscle mass has been engaged in the exercise. Usually running is quite sufficient. Lately it has been shown that such measure is not always a very precise estimate of endurance performance. This is understandable, because the peripheral component may not be adapted in parallel with the heart. There are different ways to improve the prognostic value of a test. From the above discussion it should be apparent that a measure of the effectiveness in the metabolic response to exercise would do. RER-values are then one possibility, another is lactate in muscle or blood. Most experience has been obtained with lactate. Its accumulation in blood is a good predictor of the capacity for prolonged exercise.

It is common to determine the blood lactate concentration at several work intensities (figure 22a). As shown by Costill et al. (1985) (Table 9) this is not really needed. A one point test will do just as well. Of utmost importance is, however, that the test involves the proper muscles in the right movements. It is of little interest to test a runner on a bicycle or vice versa. Like Costill et al. (1985) the best to do is to let the athlete perform over a given distance with a given speed in his/hers sport. Too much of the accuracy of this test is lost if it is not performed in the athlete's own mode of exercise.

Table 8.

Two examples are given from experiments when trained and untrained subjects exercise to exhaustion.

In case A the work is the same and in case B the relative exercise intensity is the same. Note that at the same work rate time to exhaustion is much longer for the trained than for the untrained subjects, whereas at the same relative exercise intensity time to exhaustion is not much different, but total amount of work is markedly different just as in case A. In both instances the more work performed by the trained subjects relates to the enhanced lipid utilization. Further, in all situations an almost complete glycogen depletion was noted (unpublished work, S. Runesson).

Subjects	Work rate intensity (watt)	Rel. exercise (%)	Oxygen uptake (l·min ⁻¹)	Work time of work (min)	Tot. amount (kJ)	R	Fat (G)	Carbohydrate (G)
Case A								
Ut	150	78	2.46	118	5 960	0.95	25	320
T	150	52	2.43	240	11 851	0.87	396	
Case B								
Ut	200	69	2.92	275	16 650	0.88	122	387
T	350	68	4.63	291	27 380	0.84	271	429

Table 9.

Data from a study by Costill et al. (1985), where swimmers were followed before (0) and after 5 months of training (5) aiming at top performance in a major competition. The swimmers were also followed immediately after, 1, 1, and 4 weeks the event.

	Blood lactate (mmoles × L ⁻¹) after swimming 200 yds at 90% of top performance							
	Training months				Detraining weeks			
	0	5	0	1	2	4		
Swimmers:	14.7	5.3	4.2	6.3	6.8	9.7		

The appearance of lactate in blood with a reduction in blood pH is associated with increased ventilation (figure 22b). The ventilatory coefficient (ventilation per liter of oxygen uptake) is increased which has been used by many instead of measuring the lactate concentration in blood. In experienced subjects and with good laboratory facilities using bicycle or treadmill exercises, this method works well. However, there are several drawbacks. They include that several exercise intensity levels need to be used in the test and it is hardly feasible to apply in the field.

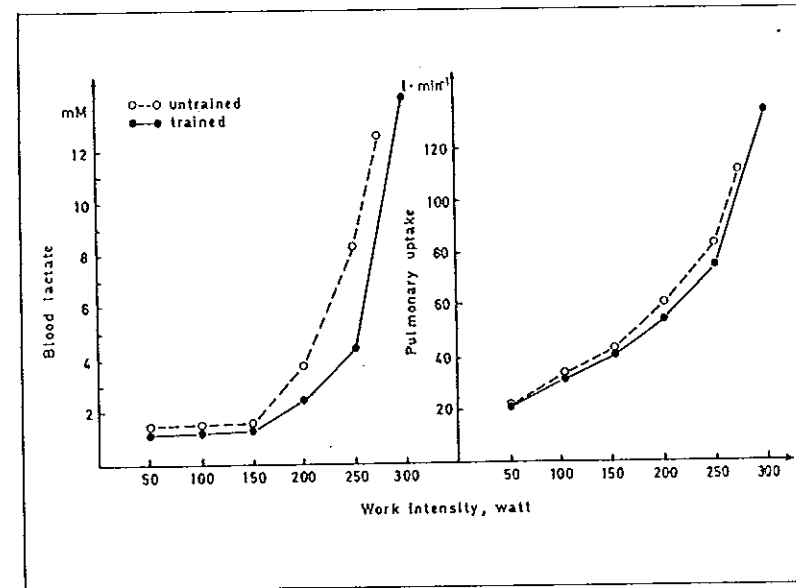


Figure 22. To the left is illustrated increase in blood lactate concentration at increasing work intensities in an untrained and trained individual. From such plots onset of blood lactate accumulation (OBLA) can be determined and used as indicator of training adaptations. To the right is a similar plot but using a ventilatory variable. The pulmonary ventilation is elevated in a linear fashion until lactate starts to appear in the blood. A small drop in pH causes ventilation to increase with an accelerated rate. This point of deflection can be used instead of the OBLA estimate. (Karlsson & Jacobs, 1982; Holman, 1985; Hughson, 1985).

Practical implications: One important practical implication of the above discussed findings is that training of endurance can be divided into two components aiming at the improvement of the pump capacity of the heart which is then closely related to the maximal oxygen uptake of the subject (figure 23). The peripheral adaptation with increased capillarization and elevated oxidative potential in the muscles is the other component whose functional significance mainly is to optimize substrate utilization of the muscle and thereby improve endurance capacity.

This dissociation between maximal oxygen uptake and muscle adaptation for performance has been nicely demonstrated experimentally. If road-runners stop their training for two weeks maximal oxygen uptake only drops by 2-3%, but performance measured as the time to exhaustion at a given speed was reduced with 24% (figure 24). Incidentally there was also a reduction in one of the oxidative enzymes used as a markers for the local adaptation. Recently Coyle et al. (1986) have added to this and demonstrated that the reduction in maximal oxygen uptake, although small, is reversible by expanding the plasma volume, which in turn made the short term work performance (4 min) to return to predetraining level.

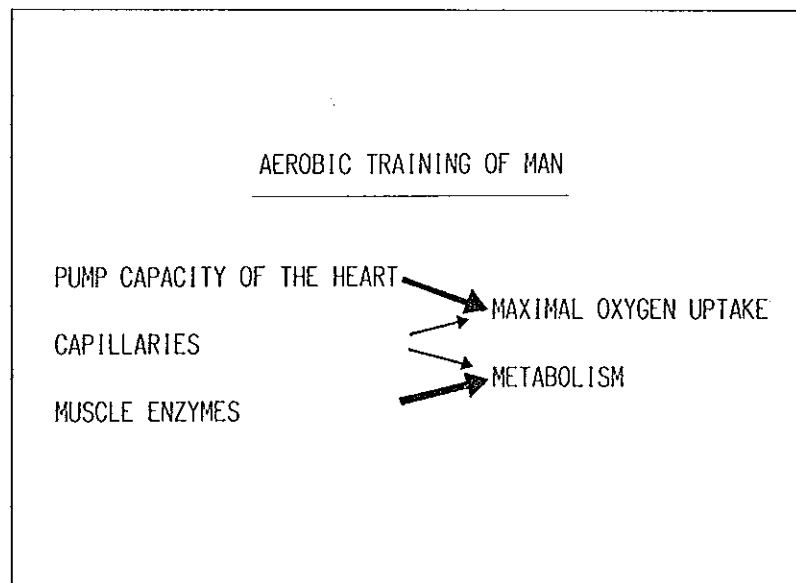


Figure 23. Schematically is illustrated the coupling between some of the adaptations which occur with endurance training and their functional significance.

Another example can be taken from the preparation for Tour de France. When the bicyclists were studied in February they had a maximal oxygen uptake of $74 \text{ ml} \times \text{kg}^{-1} \times \text{min}^{-1}$, which was only increased with 5% over the five months of intensive training up to the start of the Tour de France in July (Table 10). Oxidative enzymes in the limb muscles on the other hand doubled during the same time period, resulting in four fold higher mitochondrial enzyme level in these bicyclist than found in muscle of sedentary men. An adaptation most needed for optimal fuel economy during such a long race.

Table 10. Maximal oxygen uptake and some mitochondrial enzymes in a limb muscle of professional bicyclists in February one year at the start of the more intense preparation for the season and a week ahead of the start of Tour de France (July). Data from Sjøgaard et al. 1981.

	Professional bicyclist	
	February	July
$\dot{V}O_2 \text{ max. ml} \times \text{kg}^{-1} \times \text{min}^{-1}$	74	77
Enzymes:		
CS $\text{mmoles} \times \text{kg}^{-1} \times \text{min}^{-1} \text{ d.w.}$	69	112
HAD $\text{mmoles} \times \text{kg}^{-1} \times \text{min}^{-1} \text{ d.w.}$	66	104

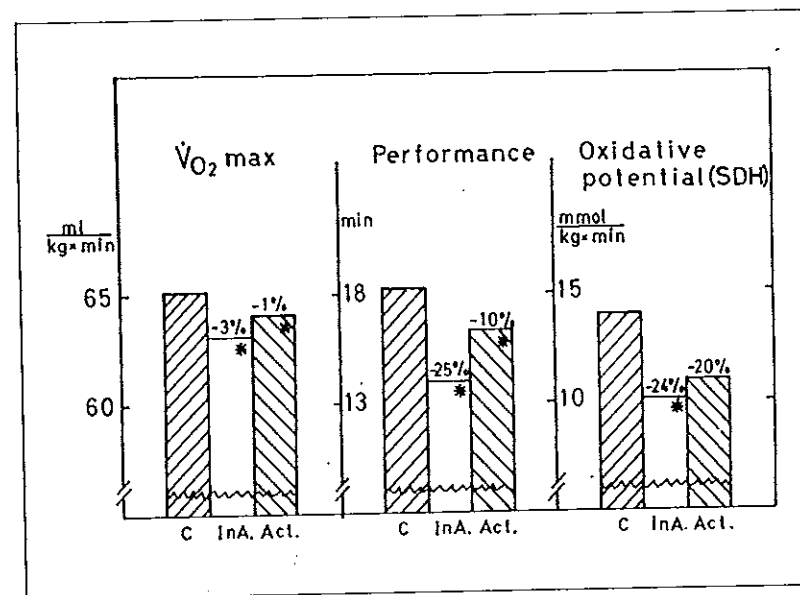


Figure 24. An oxidative enzyme (succinate dehydrogenase) in limb muscles of runners has been determined performance capacity (running to exhaustion on treadmill at a fixed speed) and maximal oxygen uptake a) when in training, b) after two weeks of complete inactivity and c) after the training had been resumed for two weeks (modified from data by Houston et al., 1979).

Another implication of the above results relates to an optimal training pattern of the heart. It is apparent that a minimum muscle mass must be involved in the exercise to train the heart. One legged training (7-8 kg muscle) is not sufficient even if the training sessions are quite long (Saltin et al., 1976) or repeated with each leg (Klausen et al., 1982). Two-legged exercise is a minimum, but whether the inclusion of arm work improving training stimulus, is not known. It is of note that rowers and c.c skiers have the very highest reported values for maximal oxygen uptake (in $\sim 7.50 \text{ l} \times \text{min}^{-1}$ and $\sim 90 \text{ ml} \times \text{kg}^{-1}$). These high values can be achieved when the athletes exercise only with their legs running.

Practical guidelines. One reason for emphasizing the central vs. peripheral factor for aerobic performance is that it has a bearing on the training, i.e. the optimal way to train the heart and the optimal training of the muscles for endurance work may not necessarily be the same. It was just stated that a minimum fraction of the muscle mass should be included in the exercise to improve the pump capacity of the heart. The intensity of the exercise is also of extreme significance. Extended duration of the training sessions can be used in exchange of the intensity, but the prize paid in time is quite marked. This is exemplified in Table 11, and although these results relate to sedentary and moderately trained individuals, the basic principle most likely applies to most athletic training.

Table 11.

Data from a study by Nordesjø (1971) where he studied young men and had them trained with different intensity, duration and frequency. From these results can be selected three different levels of intensity which gave the same (15–20%) improvement in maximal oxygen uptake after three months training, provided that the duration and frequency of the training were properly increased.

- | | |
|----|--|
| A. | Top intensity (HR ~ 190 bpm); 15 min; once/week |
| B. | Moderate intensity (HR ~ 150 bpm); 1 hr; 2–3 times/week. |
| C. | Low intensity (HR ~ 110 bpm); 2 hrs; 5 times/week. |

For the athlete with unlimited time and only the aerobic capacity to improve low intensity training performed for many hours ($\geq 2-3$ hrs) a day is an alternative. However, increasing the intensity the same training effect will be achieved in half or one third of the time.

For the muscle adaptation to occur, duration of the training appear to be essential. This does not imply that high intensity training would not elevate capillary count and mitochondrial volume in the muscles engaged in the training, but if duration is too short (≤ 30 min?) the local adaptation is far from optimal in spite of good increase in maximal oxygen uptake.

There is another difference and that is that any exercise which involves a large muscle mass can be used to train the heart. The skeletal muscle adaptation, however, has to be in the muscles used in the competition. This will only occur if these muscles are engaged in the training. The specificity is greater than one might expect. One example is from data when bicyclists, track runners and orienteers are compared. They all use their legs, but the enzymatic adaptation varies between them in the three leg muscles portions studied (Table 12). The specificity of the training has been emphasized repeatedly. A last example will be given, although it is in part outside the scope of this article, and related to energy costs of running. In the preparation for the London marathon a runner performed upto 70% of all training with the same pace as the one anticipated to be maintained in the race. This resulted in a lowered energy expenditure at that particular speed with 4–5% (H. Larsen personal communication; similar findings have been obtained in the world famous Norwegian female marathon runners, Strømme and collaborators, personal communication).

Aerobic and anaerobic training combined: In many sports both an aerobic and anaerobic energy yield are required for optimal performance. It would then be feasible to combine the training or at least balance the training so that any beneficial effects of one mode of training does not reduce the effects on the other. Anaerobic training should result in an increased rate of glycolysis and lactate tolerance which goes together with an enlarged muscle fiber cross sectional area. Aerobic training should improve the capacity of the heart, the various qualities of the muscle as well as large capillary to fiber area ratio and mitochondrial volume.

Table 12.

Data on succinate dehydrogenase in limb skeletal muscle of successful endurance athletes in three different sports, where the legs are heavily engaged in the exercise, biopsies were taken from three different muscle portions in each athlete (unpublished observations).

Sport	N	Succinate dehydrogenase (mmoles · min ⁻¹ · kg ⁻¹ w.w.)		
		M. Gastrocnemius Lat. Head	M. Quadriceps femoris Vast. lateralis	Rect. femoris
Runners, track	2	24	16	22
Orienteers	5	22	21	22
Bicyclists	3	17	24	23
Sedentary	8	7	6	6

The conflicts or problems are two and related to 1) muscle fiber area and no. of capillaries and 2) high lactate accumulation and development of mitochondria. To the first problem can be said that one has to compromise. It is impossible to perform real high intensity training without enlarging the size of the muscle fibers, but properly balanced with endurance training the no. of capillaries found around a fiber will become increased. However, the distance from the capillary to the middle of the fiber is affected only by the size of the fiber. Thus the more extreme endurance athletes (c.c. skiers, marathon runner, orienteers, bicyclists) should avoid top speed exercises (and of course explosive type strength training). In events lasting less than 30–40 minutes it is unavoidable to include very high intensity work in the training.

The risk that high intensity training would cause lengthened diffusion distances may not be so crucial, because, when muscle fibers enlarge, they may not maintain a true circular shape. Instead, they may enlarge in a more ellipsoid or rectangular form (Sillau and Banchemo, 1978). Thus, large size muscle fibers and short diffusion distances may go hand in hand.

Now, what about lactate and density of mitochondria? One of the reasons to determine the anaerobic threshold is to find a work intensity where lactate accumulates only slightly. The exercise can then be performed for quite long. This all may well be true, but the proof that it is the low lactate that allows mitochondria to increase in number (volume) is lacking. It may well be that it is the long duration of the exercise that is the crucial factor.

To test whether high intensity interval training is in fact decremental to the aerobic power and muscle oxidative enzymes, good runners were divided in groups – one performing high speed running 3 days a week (T-group) for 7 weeks in addition to distance training amounting to 40–70 km per week (Okkels, personal communication). The other group (I-group) performed fast running but not at top speed. Blood lactate levels reached above 16 mM in each special training in the T-group and around 10 mM in the

I-group. Both groups maintained their maximal oxygen uptakes and oxidative enzyme levels. In fact the group with the lower intensity (I-group) did demonstrate some increases in aerobic power. As anticipated, those training with the highest intensity elevated their glycolytic enzyme levels and anaerobic capacity. In the short running test (~ 2 min) improvement was up in both groups but for different reasons. The T-group had higher anaerobic capacity and the I-group had a higher aerobic capacity. In the longer running test only the I-group improved as could be anticipated, but it is of note that the T-group maintained their endurance performance reasonably well.

The conclusion of this is then that anaerobic and aerobic training effects can be obtained if properly trained and that they may not, to any greater extent, negatively influence upon each other. The reason why they may in practice be most likely that the athlete's time does not permit optimal training of both capacities.

Training the aerobic capacity at altitude. With the successful appearance on the world arena of runners from the highlands in Africa speculations started about special beneficial effects of training at altitude. This was supported by some studies in connection with the Olympic Games in Mexico City (Faulkner et al., 1967), although other studies failed to demonstrate any positive effects (Saltin, 1966; Buskirk et al., 1967). In fact, in some of the studies a reduction in aerobic work performance was noticed upon return to sea level (Saltin, 1966). Since then the discussion has continued, but surprisingly few studies have been performed to elucidate in detail the physiological changes. A recent trial has been made with Danish cross country skiers as discussed above. They were tested regularly from May until the day before they departed in early October for a 2 weeks stay at altitude. They stayed at 2,100 m.a.s.l. and trained at 1500-2700 m.a.s.l.; (mainly at the highest altitude). Maximal oxygen uptake was stable from August to departure and no change was observed as the result of the training at altitude (figure 25). Capillary counts did not change significantly in either the gastrocnemius muscles or the triceps brachii. The two oxidative enzymes studied were essentially unchanged with a trend towards reduction. These findings conform with results from studies on rats exposed to hypoxia (Sillau et al., 1980). Further, sedentary men, living at altitude, do not show high oxidative enzyme levels and the no of capillaries per fiber is not increased (Saltin and Gollnick, 1983).

All these data do not speak in favour of any special positive effect of training at altitude for aerobic performance as rather on the contrary. It may in fact be difficult for a top-trained endurance athlete to maintain his / hers fitness during a more prolonged stay at altitude, although the training is kept at highest possible level. In less than optimally trained subjects training at altitude may result in improvements. The question is, however, whether the same increases may not have been achieved by training at sea level.

A last point on training at altitude is that in the above discussion the elevation of the hemoglobin concentration has not been considered. It could be anticipated to have an effect. The reason why this is not always apparent is probably due to the fact that the Hb concentration rather quickly approaches «normal» levels upon return to sea level.

Training of adolescents. It was once thought that training at a young age would be superior to training at any later age. The idea came from studies of young girl swimmers

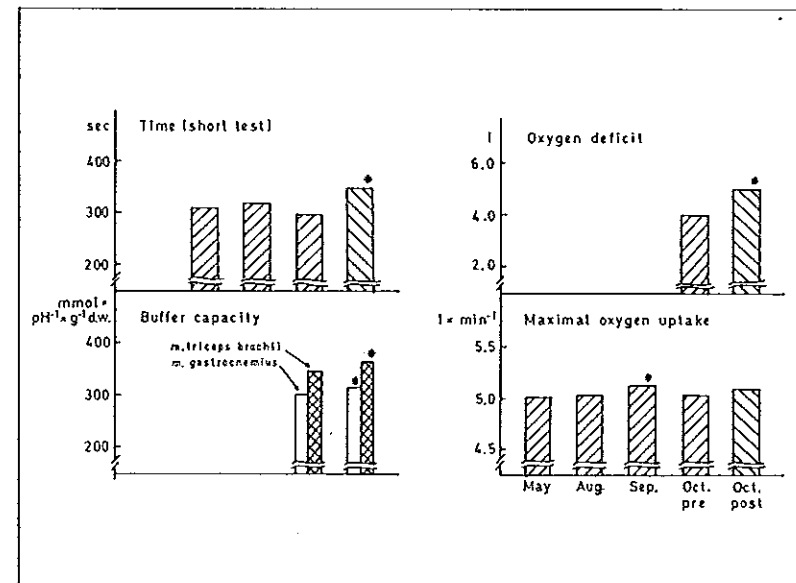


Figure 25. Summary of findings from a study of c.c. skiers training during the summer and early fall and then after two weeks at altitude (modified from data by Mizuno et al., 1987).

(Åstrand et al., 1963), and appeared at first confirmed by the findings in a well-controlled longitudinal training study of young boys (Ekblom, 1968). However, when the observed improvements were adjusted for the dimensional growth due to the concomitant increase in height, the training response was more normal, and similar to what is found when older teen-agers or young adults perform similar training (Eriksson, 1972). Several later studies have confirmed that training of the aerobic capacity in the adolescent children gives far from exceptional results. Further, during the year of the most pronounced growth spurt training per se may not have any effect at all (Klissouras, 1972; Lammert, 1976).

It is important to interpret these findings right. There is no risk or harm with training of aerobic capacity at a young age, but nothing is lost to wait until a more mature age. Further, as shown in a 5 year follow up study of Finnish teen-age endurance runners, an early start is no «short-cut» to maximal oxygen uptakes of 80 ml × kg⁻¹ × min⁻¹ and above (Elovanino, Sundberg, 1983).

Detraining - retraining: A myth is that if you have trained once and achieved a good capacity it is easier to return to the same level if - for one reason or the other - the athlete has been away from regular training for some time. This is not true which has been demonstrated for sedentary people undergoing training as well as for former very successful athletes (Jørgensen, Pedersen, 1977; Eriksson et al., 1975). It is true that

dimensions such as of the lungs and the heart does not change so rapidly. Of greater importance is, however, that the functional capacity relates to quality of the various organs or tissues. The content of enzymes in a tissue or the degree of capillarization of skeletal muscle do change more rapidly than the volume or size of a tissue or organ (Booth, 1977; Henriksson and Reitman, 1977; Klausen et al., 1981). In Table 13 is an attempt made to give an idea of the time – constants for a change of certain variables to occur. The size of the heart develops slowly (Rost and Hollman, 1983). In the Finish study no difference was observed in the 14 years old between runners (with at least 2 years of endurance training) and controls, but in a follow-up study after 5 years a clear-cut difference was observed (Elovanio, Sundberg, 1983). In adults faster changes have been seen, but the changes in the heart size must be regarded as rather slow. Blood volume is changed faster than the heart size, but it is dependent upon the dimensional development of the cardiovascular system for an optimal adaptation.

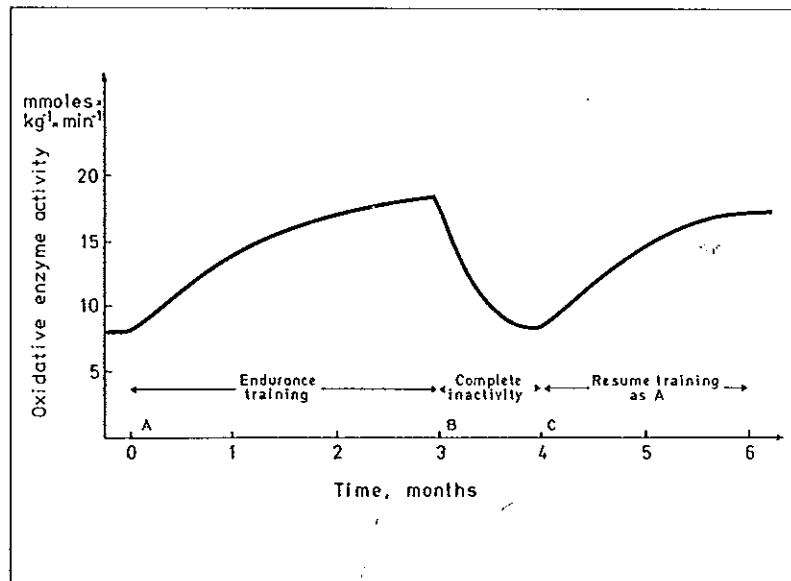


Figure 26. A schematic illustration of adaptations in the skeletal muscle at onset of a training program, resulting after some months in a given optimal adaptation for that particular training. when this training is terminated, the adaptation disappears with a rate, which is faster than observed during training. There are two reasons for this. One is that an optimal stimulus cannot abruptly be applied at start of training, i.e. the training volume and intensity is gradually increased, whereas when training is terminated the stimulus for the adaptations disappears completely. Another reason is that changes occur in relation to the absolute magnitude of the adapted variable. Thus, after the training the reduction occurs from a higher absolute value and the difference is then larger. Of note is that when training is reassumed the adaptation occurs at the same rate as during the first training period.

Table 13. Summary of approximate times for an adaptation to occur (for details see text).

Variable	Time for a change
Heart size	Months – years
Blood volume	Months
Skeletal muscle capillaries	Months
Skeletal muscle mitochondrial enzymes	Weeks
Skeletal muscle buffer capacity	Weeks?
Skeletal muscle glycolytic enzymes	Days – weeks

The alterations that take place in muscle can occur quite fast if the optimal stimulus (training pattern) is present. If it is removed the return to control level goes fast (figure 26; Booth, 1977). Capillary proliferation or reduction can occur with a time constant of weeks (Andersen & Henriksson, 1977; Klausen et al., 1981), which is similar to the mitochondrial enzymes (Henriksson, Reitman, 1977), whereas glycolytic enzymes may change within days or in a week (Pette and Dolken, 1975). The optimal training pattern for an adaptation to occur is far from known. There is also a lack of knowledge about the minimum of training required to maintain a certain level of adaptation, but it is likely to be less than that needed to achieve an improvement.

Based on the information given in Table 13 it can be said that to improve the pump capacity of the heart (= maximal oxygen uptake) there is a need for training over a long period of time (years). Muscle capillaries and oxidative enzymes can be brought to quite high levels with regular training, lasting 6 to 12 months. The glycolytic enzymes should become markedly elevated within a month of appropriate training. Too little is known about buffer capacity (= lactate tolerance) to make any firm comments. The

Table 14. A simple overview of the need for regularity in the training of various components of importance for aerobic and anaerobic work capacity.

Aerobic training	Regularly; Year round; Every week; $\dot{V}O_2 \text{ max}$ – \dot{M} Metabolism
«Buffer capacity»	Regularly; Year round; Once a week; Last month before competition period twice or three times a week.
«Glycolytic enzymes»	Last month before competition period gradually increasing up to 4–5 times per week; During competition period once or twice a week.

data in figure 26 reveal that it always take a longer time to elevate an enzyme or capacity than it takes to lose the adaptation. Thus, it must be of value to maintain some regularity in the training with some few training sessions also in the period between seasons. For one thing it is easier to resume training and the possibility for an improved performance the coming year is larger. A simplified summary of the above considerations are found in Table 14, where the regularity of various components of the training for an event (sport) demanding both an aerobic and an anaerobic energy yield are given.

Final remarks: The primary factors setting the athlete's pace in an endurance event are maximal oxygen uptake and technique (energy costs for a given speed). The time this speed can be maintained is a function of glycogen storage in the body (muscles) and efficiency in fuel utilization, which in essence is how effective the stored glycogen can be spared. The latter factor sets the relative work rate, and the glycogen stores how long it can be maintained (Table 15).

Table 15.

Summary of the factors which set the pace and determine the duration that this pace can be maintained.

Endurance exercise	
Speed (work rate)	Maximal oxygen uptake Efficiency (technique) (relative work rate (% of VO_2 max))
Duration (maintain speed)	Glycogen stores. Efficiency in usage of CHO (*glycogen saving) relative work rate (% of VO_2 max)

In an anaerobic event the rate of glycolysis sets the pace and the handling of the lactate determines how long the high work intensity can be continued. Buffer capacity of the muscles and the ability to withstand a low pH are the two critical factors, but the transfer of lactate from the contracting muscles fibers and its turnover in other tissues also play a role. Thus aerobic metabolism contribute in high intensity exercises both by supply of oxygen for an aerobic energy yield but also by using lactate via pyruvate which can serve as substrate.

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