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Tittel: Trainabilty of anaerobic capacity

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Sammendrag:

Trenbarheten av anerob kapasitet er under søkt hos 13 frivillige forsøks-personer som tre ganger i uka i en periode på 6-8 uker dreiv systematisk trening med stort innslag av anerob energiomsetning. Forsøkspersonenes anerobe kapasitet, uttrykt som det maksimale akkumulerte oksygenunderskud-det, ble bestemt før og etter treningsperioden, og en gjennomsnittlig framgang på 6% ble funnet. Resultatene viser at den anerobe kapasiteten kan økes i løpet av få ukers trening.

Stikkord: Anerob kapasitet, Trenbarhet, Melkesyrekonsentrasjonen i blodet, Løpsøkonomi

Key words: Anaerobic capacity, Trainability, Blood lactate concentration, Running economy

FORORD

Denne undersøkelsen ble gjennomført ved Muskelfysiologisk institutt høsten 1986. Arbeidet ble gjennomført som en del av Simone Burgers medisinerstudium ved Universitetet i Leiden, Nederland. Rapporten som følger er den prosjektrapporten hun skreiv om undersøkelsen.

Oslo, juni 1987.

Jon Ingulf Medbø

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I INTRODUCTION

Much research has been done on training aerobic capacity (long distance training). Literature on training anaerobic capacity however, is rare.
In my research project I have tried to find an answer to the most fundamental question concerning this topic:

- Is it possible to improve the anaerobic capacity by means of (specific) physical exercise, by so called anaerobic training ?
  Anaerobic capacity can be defined as the maximal amount of ATP (energy) formed by anaerobic processes (processes that do not use oxygen) during exercise.

Other questions are:

- What is the best form of training once it turns out to be possible to improve the anaerobic capacity ?
- And how specific are the different forms of training ?

This project does not intend to be more than a pilot study about training anaerobic capacity.

I.1 BIOCHEMICAL BACKGROUNDS

In order to move, the human body needs energy. The only source of energy that is immediately transformable into exercise is ATP.(1)
In the first few seconds of exercise the muscles get energy from small ATP-stores in the muscle cells. Despite the paramount importance of ATP as a coupling agent for energy transfer, this substance is not the most abundant store of high energy phosphate bonds in the cells. On the contrary, creatine phosphate, which also contains high energy bonds, is several times as abundant, at least in muscle. In about 30 seconds of muscle exercise all creatine phosphate in the muscle cells is used up.
After 10 seconds of exercise, glucose breakdown is the main source of energy (ATP). If the exercise is intense and short, leading to exhaustion within a few minutes, the major supply of energy is formed by the anaerobic breakdown of glucose, a process in which lactate is formed. In exercise of lesser intensity and longer duration aerobic glycolysis is the main source of ATP formation. See fig.1 and fig.2.

Fig.1: Energy production in skeletal muscle:

I anaerobic : 1. ATP stored in the muscle cells

2. $\text{creatine-P} + \text{ADP} \longrightarrow \text{creatine} + \text{ATP}$

3. $\text{glucose} \longrightarrow \text{lactate}$

$2\ \text{ADP} + 2\ \text{Pi} \qquad 2\ \text{ATP}$

II aerobic : $\text{glucose} + 6\ \text{O2} \longrightarrow 6\ \text{CO2} + 6\ \text{H2O}$

$38\ \text{ADP} + 38\ \text{Pi} \qquad 38\ \text{ATP}$

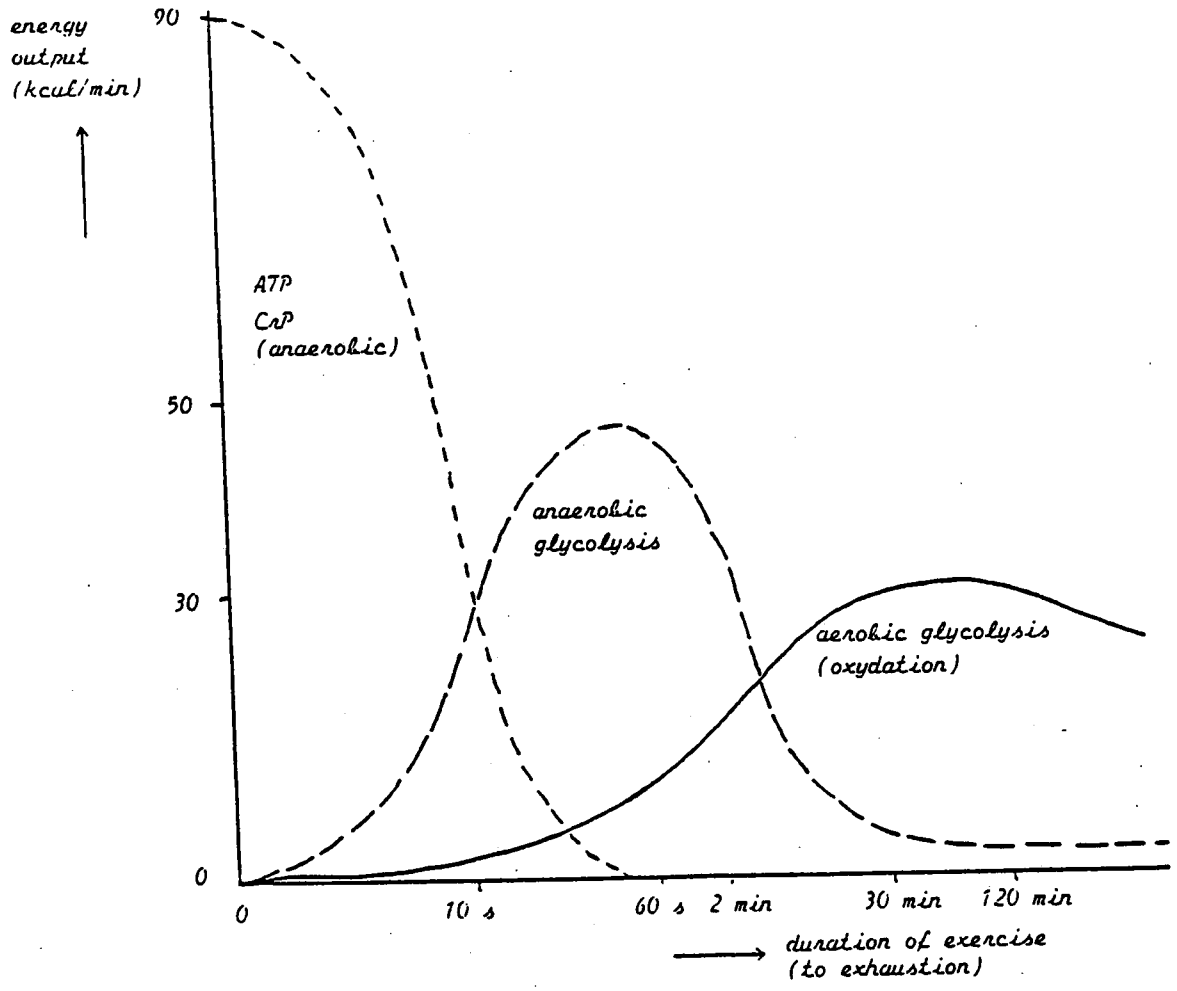

Fig. 2 : Sequence of metabolic pathways supplying energy to the working skeletal muscle. Time is given on a logarithmic scale so that very short bouts of exercise can be compared with marathon running.(8)

The anaerobic energy production is dependent on several factors:

1. The energy stores in the muscles (ATP, creatine-P and glycogen).
   The amount of glycogen stored in the muscle cells does not limit the anaerobic energy production. In fact, immediately after an exhausting exercise, there is still about 70 % left of the glycogen that was stored in the muscle cells.(1)
2. The buffering capacity of muscle and blood.
   When lactate is produced, the pH in muscle decreases, which results in a decrease in enzyme activity and in the interaction between actine and myosine. Thus the muscle gets stiff and painful and further exercise becomes increasingly difficult.(1) If muscle and blood have a larger buffering capacity, the pH will not decrease so fast when lactate is produced, and so the muscle can continue to work for a longer time.
3. The blood circulation
   The better the blood circulation in the muscle, the faster lactate is removed, and the longer the muscle can continue to work.
4. The CO2-removal.
   Increased breathing causes a faster removal of carbondioxide (CO2), which results in a slower decrease of blood pH and probably also of muscle pH.
5. Psychological factors.
   When lactate is produced, the muscles get stiff and painful. The stronger the will of a person to perform the very best he can, the longer he can go on exercising while having pain, using energy that is formed by anaerobic energy production.

I.2 METHODS TO MEASURE THE ANAEROBIC CAPACITY

In 1985 in "Médecine du Sport" J.M.Crielaard and P.Franchimont (3) published a survey of the different methods that have been used to measure the anaerobic capacity:

1. It is measured **indirectly** by the amount of exercise that a person can perform: the time that a subject can go on cycling on an ergometer at a certain load or running on a treadmill at a certain speed. The maximal pedalling frequency on the ergometer at a certain load, carried out during one minute, can also serve as a measure of the anaerobic capacity.
   There are a few major problems in interpreting the results of such tests. First of all it is not possible to say how large the contribution of the anaerobic energy production is, compared to the aerobic energy production. Secondly, the total energy production is not exactly known, for only a part of all the energy that is produced is used for the performance of the exercise. The extent of this part is different for every subject and depends on his running/cycling economy.
2. Another method to determine the anaerobic capacity is measurement of the oxygen debt. This is based on the assumption that only when a subject exercises at a supra-maximal intensity (an intensity at which it is not possible to supply enough oxygen for aerobic production of all the energy that is needed), energy is produced by the **anaerobic breakdown of glucose**. After a period of strenuous exercise, the oxidative metabolic processes continue to operate at a high level of activity for many minutes to 1) reconvert the lactic acid into glucose and 2) reconvert ADP and creatine into ATP and creatine phosphate respectively. The extra oxygen that is needed in the oxidative energy process to rebuild these substances is called oxygen debt.(3) See fig.3.(3)

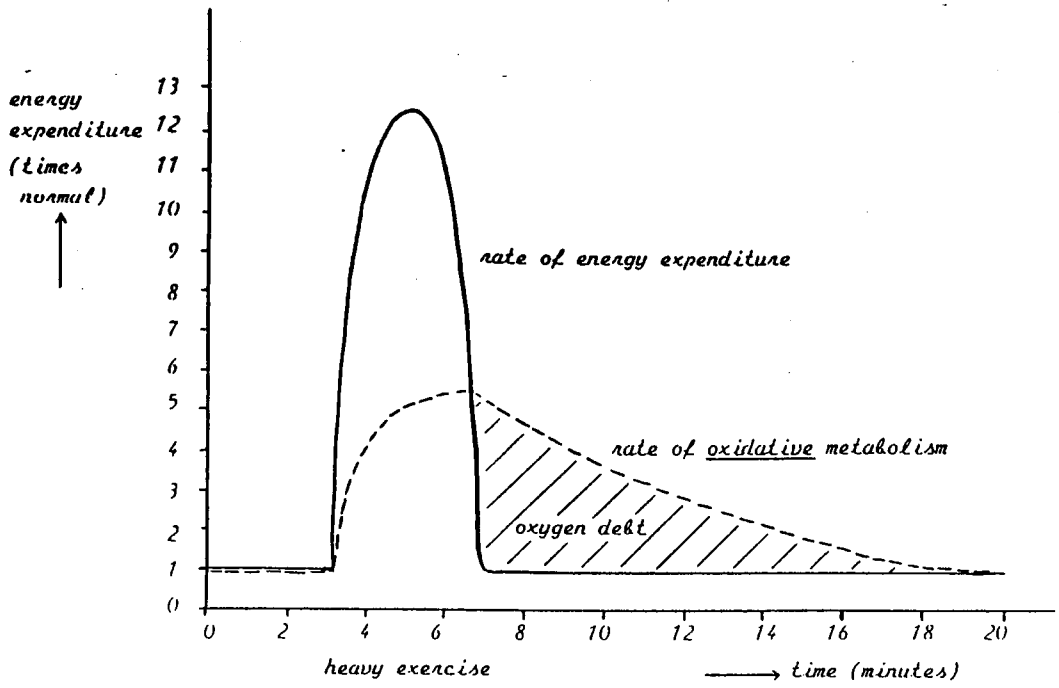

Fig. 3 : Oxygen debt occurring after a bout of strenuous exercise

This classical concept of oxygen debt has been strongly controverted : "O2 debt does not exist" (Harris, 1980 (in (1))); "The term O2 debt is archaic and should not be used" (Brooks and Gaesser, 1980 (in (1))). 3. The difference between the estimated oxygen demand integrated over time is called accumulated oxygen deficit. See fig. 4.

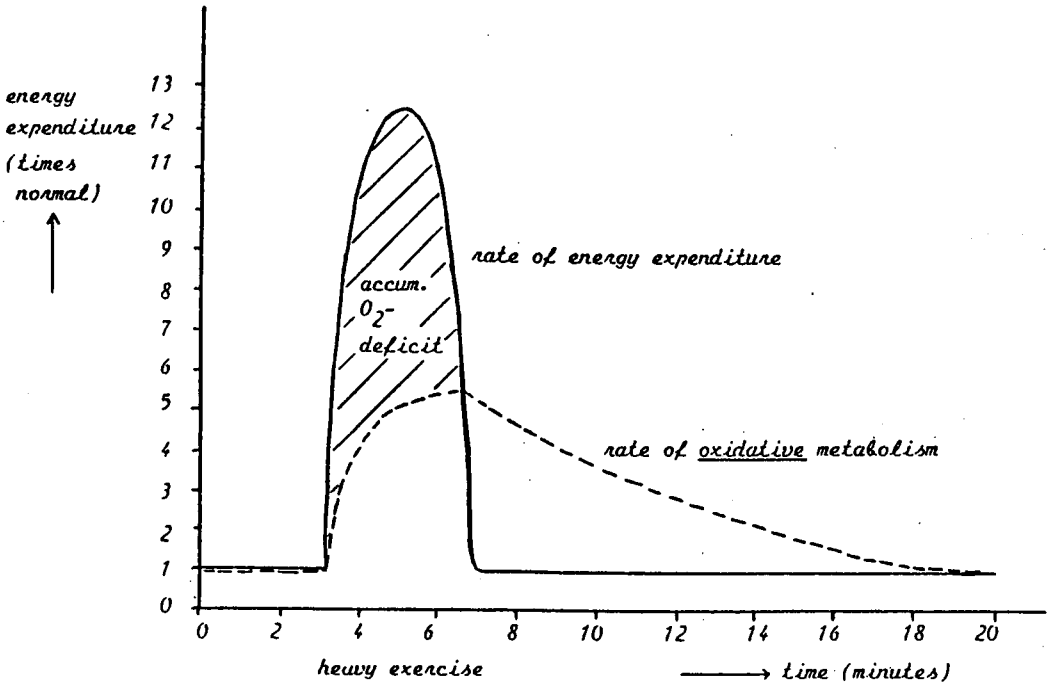

Fig. 4 : Accumulated oxygen deficit occurring during a bout of strenuous exercise

Jon I. Medbø et al. from the Muscle Physiological Institute in Oslo, Norway, have shown that maximal accumulated oxygen deficit provides an accurate estimate of the anaerobic capacity.(6) Since I carried out my research project at this institute, I used this method to measure the anaerobic capacity and will describe it more extensively.

In their experiments Medbø et al. found that

1) accumulated oxygen deficit increases with the duration of the exhausting exercise until a plateau is reached after 2 minutes. See fig. 5. This is in accordance with the assumption that there are rate limiting steps in the process of anaerobic ATP-formation;

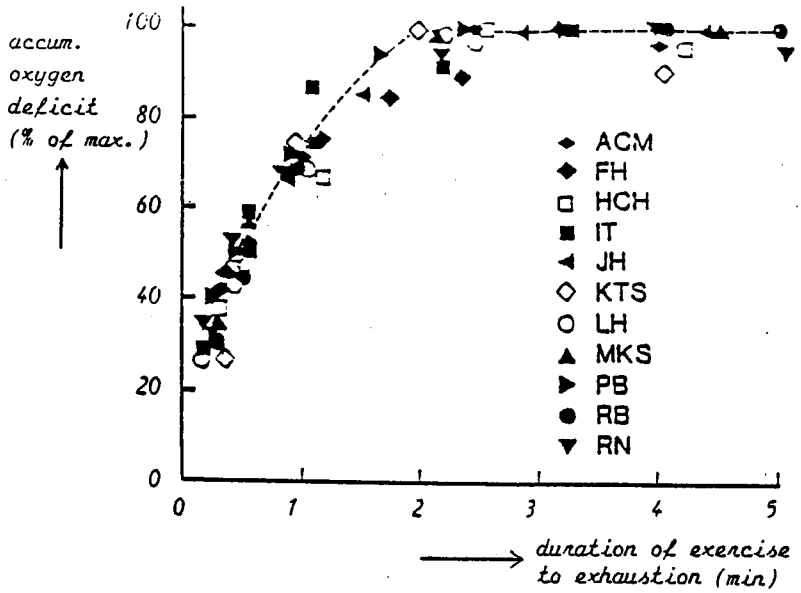

Fig. 5 : The relative accumulated oxygen deficit (in percentages of maximal accumulated oxygen deficit) versus duration of supra-maximal runs to exhaustion.

2) maximal accumulated oxygen deficit is independent of maximal oxygen uptake. It is expected that they are independent, because the rate limiting step in the aerobic ATP-generation follows the formation of pyruvate, while the maximal rate of anaerobic ATP-generation is dependent on the capacity of the glycolytic enzymes. See fig. 6.

Fig. 6 : Rate limiting steps in aerobic and anaerobic glycolysis.

<u>aerobic glycolysis</u> :

glucose ⟶ pyruvate —CoA→ acetyl CoA ⟶ citric acid cycle
rate limiting step

<u>anaerobic glycolysis</u> :

glucose ⟶ pyruvate ⟶ lactate
rate limiting : glycolytic enzymes

Maximal accumulated oxygen deficit is calculated as the difference between the estimated oxygen demand and the measured oxygen uptake during a run on a treadmill leading to exhaustion in 2-3 minutes. The oxygen demand, which is a measure for the total energy turnover rate, is estimated from the linear relation between running speed and the oxygen uptake at submaximal intensities. This is called running economy. It is necessary to define the individual running economy of each subject, because there is a large interindividual variability.(7)

4. The method to measure the anaerobic capacity that is probably most frequently used, is measurement of plasma lactate concentration in arteries, veins or capillaries. This method has many disadvantages :
   a) there is a low correlation between plasma lactate and exercise performance;
   b) plasma lactate concentration is irregular, as it is not only influenced by exercise of high intensity, but also by the alimentary regime and by catecholamines;
   c) lactate is measured in plasma, not in muscle, where it has an exercise limiting effect;
   d) a considerable amount of produced lactate is reoxydized in the muscle itself or by the myocard;
   e) there are large differences in lactate level in arteries, veins and capillaries;
   f) plasma lactate concentration is also dependent on diffusion speed from the intra- to the extramuscular compartment and on the distribution volume.
5. A more complicated method is determination of muscle enzymes in plasma, for example LDH (lactate dehydrogenase) and CK (creatine kinase). Recently, a negative correlation was found between the plasma CK concentration after a 400 meter run and the performance on that 400 m. A positive correlation was found between the iso-enzyme LDH-M concentration and the performance on 400 m. (Ohkuma et al., 1984 (in (1))).
6. The best method to measure the anaerobic capacity would be to measure pH and lactate in the muscle itself. This is possible by taking muscle biopsies, which is quite an aggressive method and has its specific disadvantages. The most important disadvantage is the fact that only a tiny little piece of the muscle is analyzed, which is probably not representative for the whole muscle.
7. Also NMR (Nuclear Magnetic Resonance) can be used to measure pH and lactate in the muscle itself, but this method is very expensive and technically difficult.

One of the general conclusions of this overview of methods to measure anaerobic capacity is that the interpretation of the results of the measurements is often difficult and that there are large differences between the results of the different tests.
Crielaard and Franchimont conclude that there is no single reliable method to measure anaerobic capacity; but when several different complementary methods are used, it is possible to discover talented people with an inborn large anaerobic capacity and to predict performance capacity.(1)

Therefore I used two different methods to determine the anaerobic capacity in my research project:

1) maximal accumulated oxygen deficit
   This method was developed at the institute where I carried out my project and seemed to provide an accurate estimate of the anaerobic capacity.
2) peak capillary blood lactate concentration
   This is a simple method, which I could use at the institute where I carried out my project.

II AIM OF THE STUDY

The main question in this research project is:

- Is it possible to improve the anaerobic capacity by means of (specific) physical exercise ?

Since I worked with the method of accumulated oxygen deficit, a second question became interesting:

- Can training change the relation between accumulated oxygen deficit and the duration of exhausting exercise (as visualized in fig. 5.) ?

One could expect that the effect of a form of training that consists of short, fast bouts of about 20 seconds results in a faster supply of anaerobic energy (accumulated oxygen deficit), while the total anaerobic capacity (maximal accumulated oxygen deficit) will not increase at all. This would change the relation between accumulated oxygen deficit and the duration of exhausting exercise as shown in fig. 7.

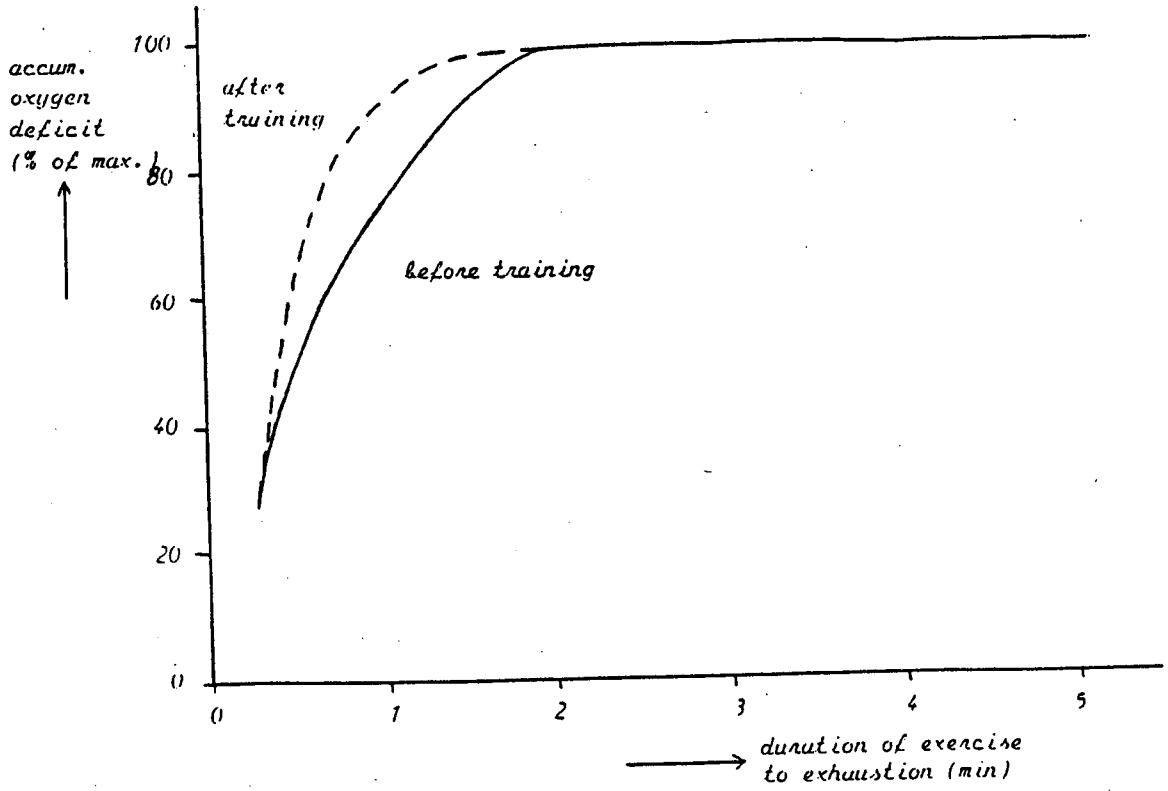

Fig. 7 : Hypothetical change in the relation between relative accumulated oxygen deficit and the duration of exhausting exercise, induced by training in short fast bouts (about 20 seconds).

III MATERIALS AND METHODS

III.1 SUBJECTS

Seventeen volunteers, 8 males and 9 females, served as subjects. Four of them, 2 males and 2 females, dropped out before or during the first training period, because of injuries and illness.
Five subjects (KN, GL, SM, SL and TN) dropped out after the measurements following the first training period.
Two subjects (DHA and TK) started the training late in the research period. I therefore did not take measurements of their anaerobic capacity three times, as I did for the others, but only twice, before and at the end of their training periods.
The physical characteristics of the subjects are shown in table 1.

Table 1 : Physical characteristics of the subjects.

subject	sex	age	height cm	weight kg	state of training before the project
group 1 (trained 3 times 2 minutes)					
1. EK	F	40	171	83.5	little trained
2. KE	F	26	168	57	well trained
3. KG	F	23	175	62	moderately trained
4. DHA	M	40	176	82.5	long dist. trained
5. TK	M	42	175	65	little trained
6. KN	F	30	180	66	little trained
7. GL	M	27	188	84.5	moderately trained
average	M:F=3:4	33	176	72	
average 1-3	M:F=0:3	30	171	68	
group 2 (trained 8 times 20 seconds)					
8. ILH	F	24	171	69	little trained
9. KF	F	31	175	63	little trained
10. SG	F	30	180	65	moderately trained
11. SM	M	40	184	87	moderately trained
12. SL	M	21	186	85.5	little trained
13. TN	M	30	183	74	moderately trained
average	M:F=3:3	29	180	74	
average 1-3	M:F=0:3	28	175	65.5	

The averages of the first 3 subjects of group 1 and the first 3 of group 2 are of importance, because only these subjects kept on training until the last series of measurements.

III.2 METHODS

For every subject running economy, maximal accumulated oxygen deficit (anaerobic capacity), maximal peak blood lactate concentration in capillary blood (anaerobic capacity), maximal oxygen uptake (aerobic capacity) and accumulated oxygen deficit and peak blood lactate concentration in an exhausting run lasting about 30 seconds, were determined three times: before the subjects started training, half way through and right after the training period.
All experiments were done on the treadmill at 3 degrees inclination.
The subjects were trained in treadmill running before testing started.

Running economy:
Over a period of 2-3 weeks prior to the exhausting runs, steady state oxygen uptake was measured during the last 2 minutes of several runs lasting 10 minutes, at different submaximal intensities. For each subject all results relating submaximal treadmill speed to steady state oxygen uptake were plotted and visually checked for linearity; deviating values were excluded. A linear relation was determined for each subject by calculating the regression of steady state oxygen uptake on exercise intensity, thus expressing the oxygen demands for all intensities.(7) See fig. 8.

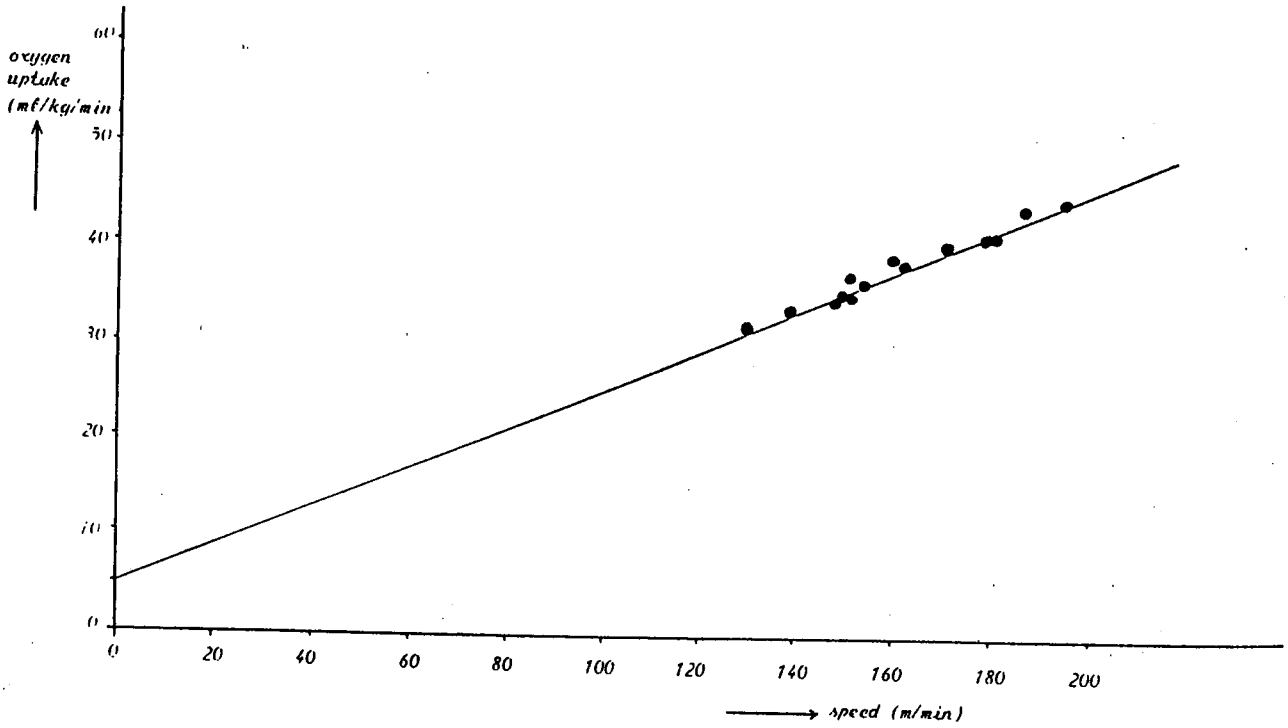

Fig. 8 : Example of a relation between oxygen uptake (VO2) and running speed for subject KG, which is in formula : y = 5 + 0.203 x .
(5 (ml/kg/min) is a common Y-intercept; 0.203 is her individual running economy characteristic)

Maximal accumulated oxygen deficit:
The maximal accumulated oxygen deficit is defined as the difference between the estimated accumulated oxygen demand in an exhausting exercise lasting longer than 2 minutes, and the accumulated oxygen uptake during that exercise.
To determine the maximal accumulated oxygen deficit, the subjects ran on a treadmill at a supramaximal intensity that would lead to exhaustion in 2-3 minutes.
Expired air was continuously collected in Douglas bags during the exhausting run. The expired volume was measured in a wet spirometer. Fractions of oxygen and carbondioxide were determined on an Ametek oxygen analyzer and a Simrad Optronics CO2 analyzer. All gas volumes were expressed as STPD (Standard Temperature Pressure Dry). Thus the accumulated oxygen uptake could be calculated.
The oxygen demand at that supramaximal intensity was determined by extrapolating the linear relation between oxygen uptake and running speed at submaximal intensities (running economy).

Maximal peak blood lactate concentration:
To determine the maximal peak blood lactate concentration in capillary blood, blood samples were taken from a prewarmed finger at 4 and 7 minutes after the exhausting exercise of 2-3 minutes. For each blood sample 20 µl of blood was put into 500 µl of 0.4 mol/l perchloric acid and kept on ice for later measurement of lactate concentration, as described in O.H. Lowry, "A flexible system of enzymatic analysis", 1972 (5): 10 µl plasma is added to a reagent that consists of hydrazine, 2-amino-2-methylpropanol buffer pH 9.9, NAD+ and LDH. Thus an enzymatic reaction takes place, which results in the formation of pyruvate hydrazone. By spectrophotometry the pyruvate hydrazone concentration can be determined. Then the plasma lactate concentration can be calculated.

Maximal oxygen uptake:
During the exhausting runs expired air was continuously collected in Douglas bags.
The oxygen uptake during the second minute of the exhausting run seemed to be almost equal to the maximal oxygen uptake that was determined in several runs at low supramaximal intensities. Therefore the oxygen uptake during the second minute of the exhausting run could serve as the maximal oxygen uptake of a subject.

Exhausting run lasting about 30 seconds:
Accumulated oxygen deficit and peak blood lactate concentration in capillary blood were determined during / after an exhausting run lasting about 30 seconds.

III.3 STATISTICS

Data are presented as individual values or averages ± SE . Statistical tests were done using Students T-test, with 0.05 as the level of statistical significance.

III.4 TRAINING OF ANAEROBIC CAPACITY

The subjects in this research project were divided into two groups, which followed different training programmes. All subjects trained three times a week for 6-11 weeks, some on the treadmill, others outside after having experienced the intensity on the treadmill. All subjects wrote down every physical exercise they did during the test period.
Group 1 ran , after 10 minutes of warming up, 3 times 2 minutes at an intensity that corresponds to about 122 % (115 - 127 %) of their maximal oxygen uptake, 8 minutes calmly walking between the bouts.
Group 2 ran, after 10 minutes of warming up, 8 times 20 seconds at an intensity that corresponds to about 177 % (171 - 185 %) of their maximal oxygen uptake, 5 minutes calmly walking between the bouts.
The common factor in these two types of training is that both cause a high oxygen debt (gasping for breath after the exercise) and high blood lactate concentrations. During an average training of every subject accumulated oxygen deficit and blood lactate concentrations were determined.

IV RESULTS

IV.1 TRAINING

The accumulated oxygen deficit and the blood lactate concentrations in capillary blood during an average training session of every subject, are shown in fig. 9 - 12. They are expressed in percentages of the maximal values. These were determined in the exhausting run of 2-3 minutes, performed within one week before or after the determination of the training values.

Group 1:
The average accumulated oxygen deficit during the bouts was 70 % of maximal (52 - 93 %). See fig. 9.
The capillary blood lactate level increased from 5 - 17 % of maximal (0.6 - 2.5 mmol/l) after the warming up, to 45 - 80 % of maximal (6.4 - 12.7 mmol/l) after the last bout. See fig. 10.

Group 2:
The average accumulated oxygen deficit during the bouts was 42 % of maximal (31 - 62 %). See fig. 11.
The capillary blood lactate level increased from 4 - 41 % of maximal (0.6 - 5.9 mmol/l) after the warming up, to 60 - 98 % of maximal (8.2 - 14.0 mmol/l) after the last bout. See fig. 12.

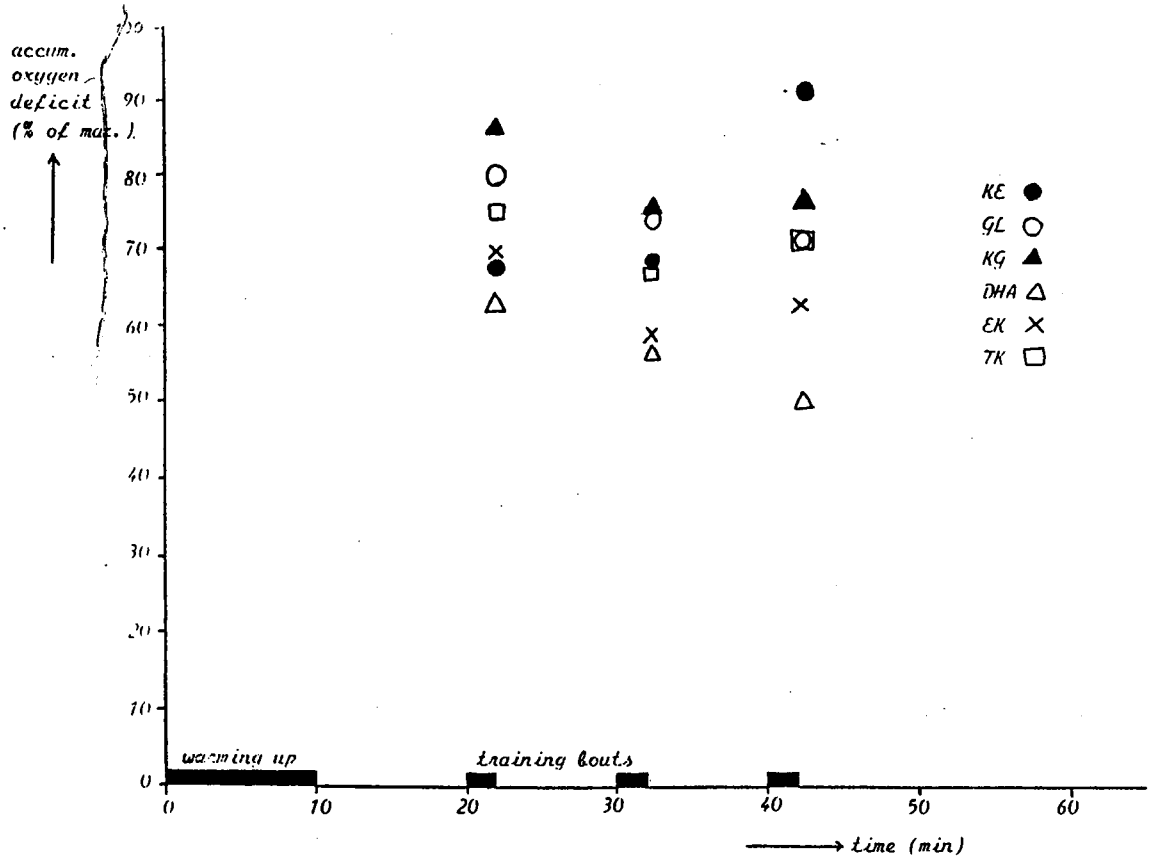

Fig. 9 : The accumulated oxygen deficit during an average training of the subjects in group 1 in percentages of maximal. The warming up and the training bouts (3 times 2 minutes) are shown on the time-axis.

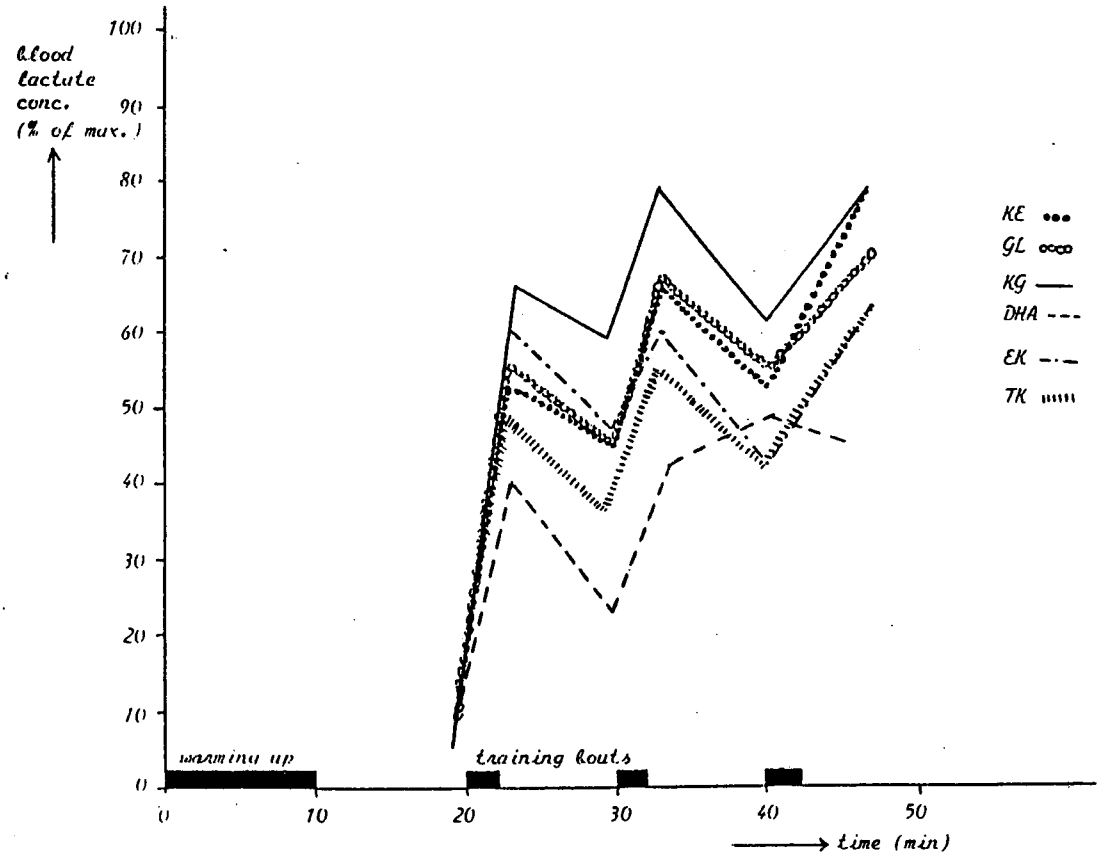

Fig. 10 : The blood lactate concentrations during an average training of the subjects of group 1 in percentages of maximal.

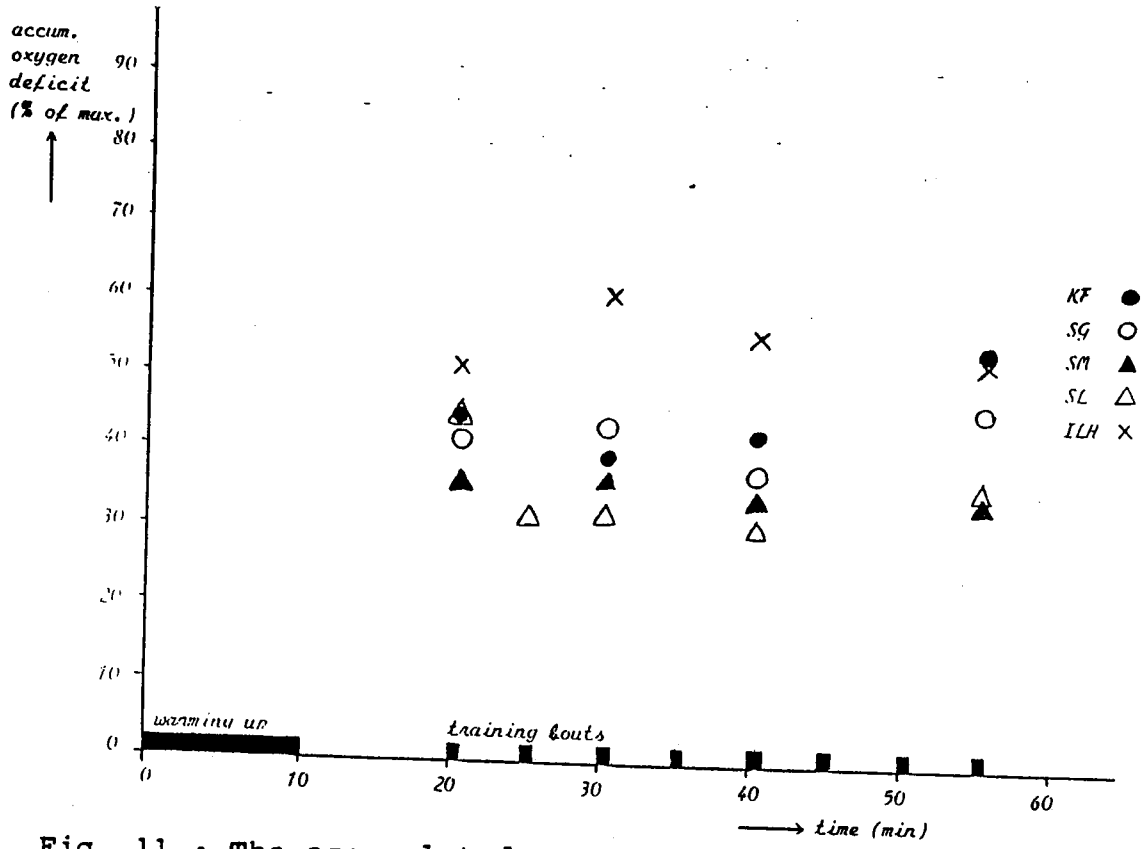

Fig. 11 : The accumulated oxygen deficit during an average training of the subjects of group 2 in percentages of maximal. The warming up and the training bouts (8 times 20 seconds) are shown on the time-axis.

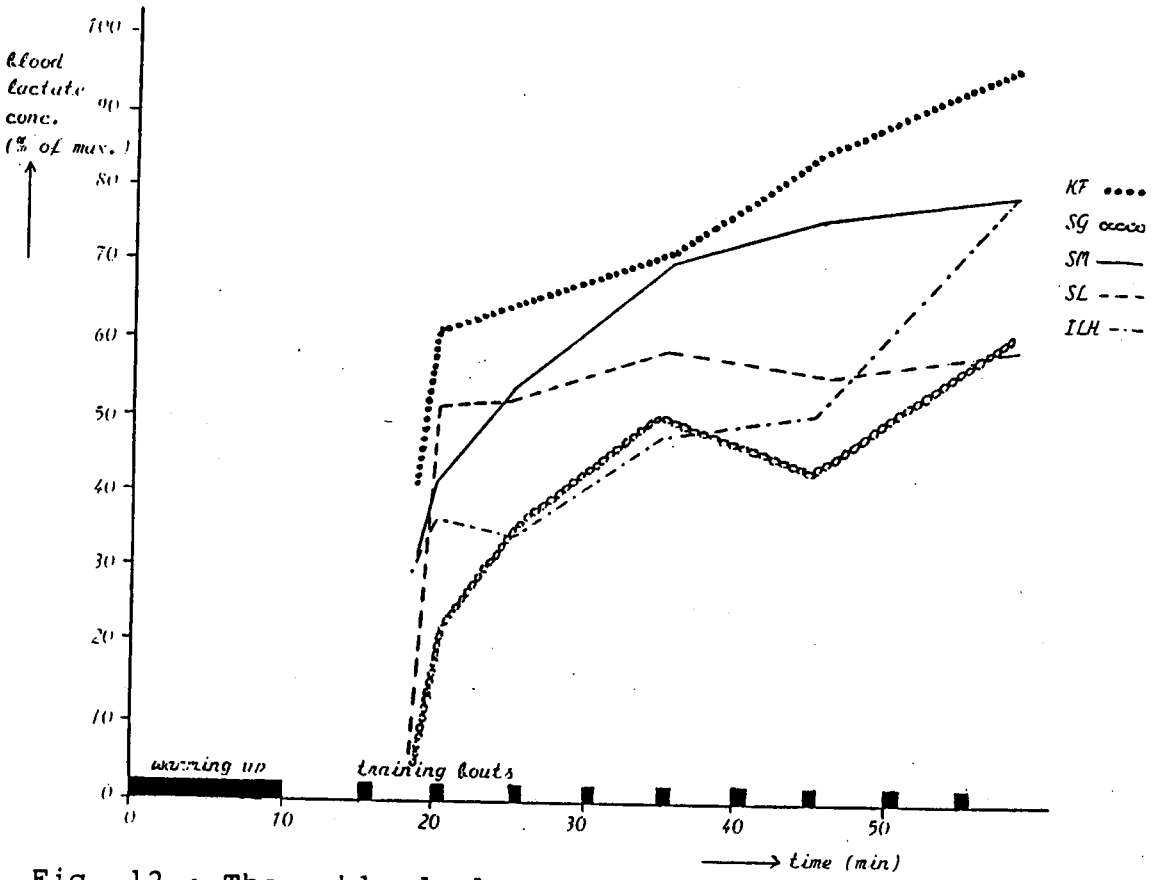

Fig. 12 : The blood lactate concentrations during an average training of the subjects of group 2 in percentages of maximal.

IV.2 EXHAUSTING RUNS LASTING 2-3 MINUTES

Table 2 shows the individual values for the running economy, the maximal oxygen uptake (aerobic capacity), the maximal accumulated oxygen deficit (anaerobic capacity) and the maximal peak capillary blood lactate level before the subjects started training, after having trained for 4-6 weeks and after the whole training period (9-11 weeks).
Before the subjects started their training programmes, there were no significant differences in running economy, in maximal oxygen uptake, maximal accumulated oxygen deficit and maximal peak capillary blood lactate level between group 1 and group 2.
Neither were there any significant differences between the subjects that dropped out after 4-6 weeks and the ones that kept on till the end, except for the fact that the latter group consisted of only females.
Table 3 shows the increase in running economy characteristic, in maximal oxygen uptake, maximal accumulated oxygen deficit and maximal peak capillary blood lactate level for every subject.
Some subjects improved in running economy (lower running economy characteristic), from 1 to 4 %, others worsened, from 1 to 7 %.
Most subjects improved their maximal oxygen uptake, but not enough to reach statistical significance.
All subjects but one (GL) improved their maximal accumulated oxygen deficit, but there were great differences in the grade of improvement, which does not depend on the length of the period they had trained, as shown in fig. 13.
In group 2 the maximal accumulated oxygen deficit increased more than in group 1, but this difference is not statistically significant ($p = 0.1$).
The peak capillary blood lactate level did not change at all in group 1. In group 2 there was an increase, but not enough to reach statistical significance.

IV.3 EXHAUSTING RUNS LASTING ABOUT 30 SECONDS

There was no change in the relation between the duration of the exhausting exercise and the accumulated oxygen deficit after the training period, neither in group 1 nor in group 2. See fig. 14

Table 2 : The individual values for running economy, maximal oxygen uptake, maximal accumulated oxygen deficit and maximal peak blood lactate concentration (the latter three determined in exhausting runs lasting 2-3 minutes), as measured before the subjects started training, half way through and after the training period.

subject	running economy (y = 5 + .. x)	VO2-max. (ml/kg/min)	max.acc.O2-def. (ml/kg)	max.bl.lactate (mmol/l)
BEFORE TRAINING				
group 1				
1. EK	0.208	39.5	39.2	14.6
2. KE	0.234	61	47.0	17.0
3. KG	0.203	47.5	32.4	12.0
4. DHA	0.223	47.5	42.6	14.8
5. TK	0.250	49	52.0	12.7
6. KN	0.228	45	42.6	15.2
7. GL	0.206	50	57.2	19.4
average ± SE				
1-3	0.215 ± 0.010	49.5 ± 6	39.5 ± 4.2	14.5 ± 1.4
all	0.221 ± 0.007	48.5 ± 2.5	44.7 ± 3.1	15.1 ± 0.9
GL excl.	0.224 ± 0.007	48 ± 3	42.6 ± 2.7	14.4 ± 0.7
group 2				
8. ILH	0.224	45	33.6	13.3
9. KF	0.235	43	45.7	15.7
10. SG	0.214	47.5	39.1	13.5
11. SM	0.218	46	51.5	18.6
12. SL	0.220	52	51.9	13.3
13. TN	0.247	49.5	51.9	17.8
average ± SE				
8-10	0.224 ± 0.006	45 ± 1.5	39.5 ± 3.5	14.2 ± 0.8
all	0.226 ± 0.005	47 ± 1.5	45.6 ± 3.2	15.4 ± 1.0

subject	running economy (y = 5 + .. x)	VO2-max. (ml/kg/min)	max.acc.O2-def. (ml/kg)	max.bl.lactate (mmol/l)
AFTER 4 - 6 WEEKS TRAINING				
group 1				
1. EK	0.213	39.5	42.0	15.7
2. KE	0.235	62.5	47.4	15.5
3. KG	0.204	49	32.1	12.7
4. DHA	0.226	48.5	44.2	14.9
5. TK	0.253	50.5	60.6	13.4
6. KN	0.230	45.5	53.8	14.8
7. GL	0.197	52.5	42.9	17.7
average ± SE				
1-3	0.217 ± 0.009	50.5 ± 6.5	40.5 ± 4.5	14.6 ± 1.0
all	0.223 ± 0.007	49.5 ± 2.5	46.1 ± 3.4	15.0 ± 0.6
GL excl.	0.227 ± 0.007	49 ± 3	46.7 ± 4.0	14.5 ± 0.5
group 2				
8. ILH	0.220	49	30.5	11.5
9. KF	0.231	43	44.8	15.9
10. SG	0.222	48.5	49.3	14.3
11. SM	0.220	47	53.1	16.1
12. SL	0.215	50	60.0	14.8
13. TN	0.254	50	62.3	15.7
average ± SE				
8-10	0.224 ± 0.003	47 ± 2	41.5 ± 5.7	13.9 ± 1.3
all	0.227 ± 0.006	48 ± 1	50.0 ± 4.7	14.7 ± 0.7
AFTER 9 - 10 WEEKS TRAINING				
group 1				
1. EK	0.217	41	39.2	15.4
2. KE	0.236	62	48.4	14.6
3. KG	0.201	48	34.8	13.6
average ± SE				
1-3	0.218 ± 0.010	50.5 ± 6	40.8 ± 4.0	14.5 ± 0.5
group 2				
8. ILH	0.217	46	42.1	15.2
9. KF	0.236	43	48.5	16.4
10. SG	0.229	49.5	58.5	16.4
average ± SE				
8-10	0.227 ± 0.005	46 ± 2	49.7 ± 4.8	16.0 ± 0.4

Table 3 : The increase in the individual values for running economy, maximal oxygen uptake, maximal accumulated oxygen deficit and maximal peak blood lactate concentration (the latter three determined in exhausting runs lasting 2-3 minutes) in percentages of the former values and in absolute figures.

subject	running economy (y = 5 + .. x)	VO2-max. (ml/kg/min)	max.acc.O2-def. (ml/kg)	max.bl.lactate (mmol/l)
INCREASE IN FIRST PERIOD				
group 1				
1. EK	2 % (0.005)	0 % (0)	7 % (2.8)	8 % (1.1)
2. KE	0 % (0.001)	2 % (1.5)	1 % (0.4)	- 9 % (1.5)
3. KG	0 % (0.001)	2 % (1)	- 1 % (0.3)	6 % (0.7)
4. DHA	1 % (0.003)	2 % (1)	4 % (1.6)	1 % (0.1)
5. TK	1 % (0.003)	3 % (1.5)	17 % (8.6)	6 % (0.7)
6. KN	1 % (0.002)	1 % (0.5)	26 % (11.2)	- 3 % (0.4)
7. GL	- 4 % (0.009)	5 % (2.5)	- 25 % (14.3)	- 9 % (1.7)
average ± SE				
1-3	1 % (0.002)	2 % (1)	2 % (1.0)	1 % (0.1)
all	0 % (0.001)	2 % (1)	3 % (1.4)	- 1 % (0.1)
GL excl.	1 % (0.003)	2 % (1)	10 % (4.1)	1 % (0.1)
group 2				
8. ILH	- 2 % (0.004)	9 % (4)	- 9 % (3.1)	- 14 % (1.8)
9. KF	- 2 % (0.004)	0 % (0)	- 2 % (1.1)	1 % (0.2)
10. SG	4 % (0.008)	2 % (1)	26 % (10.2)	4 % (0.8)
11. SM	1 % (0.002)	2 % (1)	3 % (1.6)	- 13 % (2.5)
12. SL	- 2 % (0.005)	- 4 % (2)	16 % (8.1)	11 % (1.5)
13. TN	3 % (0.007)	1 % (0.5)	20 % (10.4)	- 12 % (2.1)
average ± SE				
1-3	0 % (0.000)	4 % (1.5)	5 % (2.0)	- 2 % (0.3)
all	0 % (0.001)	2 % (1)	10 % (4.4)	- 4 % (0.7)
INCREASE IN SECOND PERIOD				
group 1				
1. EK	4 % (0.009)	3 % (1.5)	- 7 % (2.8)	- 2 % (0.3)
2. KE	0 % (0.001)	- 1 % (0.5)	2 % (1.0)	- 6 % (0.9)
3. KG	- 1 % (0.003)	- 2 % (1)	8 % (2.7)	7 % (0.9)
average ± SE				
1-3	1 % (0.002)	0 % (0)	1 % (0.3)	- 1 % (0.1)
group 2				
8. ILH	- 1 % (0.003)	- 6 % (3)	38 % (11.6)	32 % (3.7)
9. KF	2 % (0.005)	0 % (0)	8 % (3.7)	3 % (0.5)
10. SG	3 % (0.007)	2 % (1)	19 % (9.2)	15 % (2.1)
average ± SE				
1-3	1 % (0.003)	- 1 % (1)	20 % (8.2)	15 % (2.1)

subject	running economy (y = 5 + .. x)	VO2-max. (ml/kg/min)	max.acc.O2-def. (ml/kg)	max.bl.lactate (mmol/l)
TOTAL INCREASE				
group 1				
1. EK	7 % (0.014)	3 % (1.5)	0 % (0.0)	5 % (0.8)
2. KE	1 % (0.002)	2 % (1)	3 % (1.4)	- 14 % (2.4)
3. KG	- 1 % (0.002)	1 % (0.5)	7 % (2.4)	13 % (1.6)
4. DHA	1 % (0.003)	2 % (1)	4 % (1.6)	1 % (0.1)
5. TK	1 % (0.003)	3 % (1.5)	17 % (8.6)	6 % (0.7)
6. KN	1 % (0.002)	1 % (0.5)	26 % (11.2)	- 3 % (0.4)
7. GL	- 4 % (0.009)	5 % (2.5)	- 25 % (14.3)	- 9 % (1.7)
average ± SE				
1-3	2 % (0.005)	2 % (1)	3 % (1.3)	0 % (0.0)
all	2 % (0.004)	2 % (1)	3 % (1.6)	- 1 % (0.2)
GL excl.	1 % (0.002)	2 % (1)	10 % (4.2)	- 0 % (0.1)
group 2				
8. ILH	- 3 % (0.007)	2 % (1)	25 % (8.5)	14 % (1.9)
9. KF	0 % (0.001)	0 % (0)	6 % (2.8)	4 % (0.7)
10. SG	7 % (0.015)	4 % (2)	50 % (19.4)	21 % (2.9)
11. SM	1 % (0.002)	2 % (1)	3 % (1.6)	- 13 % (2.5)
12. SL	- 2 % (0.005)	- 4 % (2)	16 % (8.1)	11 % (1.5)
13. TN	3 % (0.007)	1 % (0.5)	20 % (10.4)	- 12 % (2.1)
average ± SE				
1-3	1 % (0.003)	2 % (1)	26 % (10.2)	13 % (1.8)
all	1 % (0.002)	1 % (0.5)	19 % (8.5)	3 % (0.4)

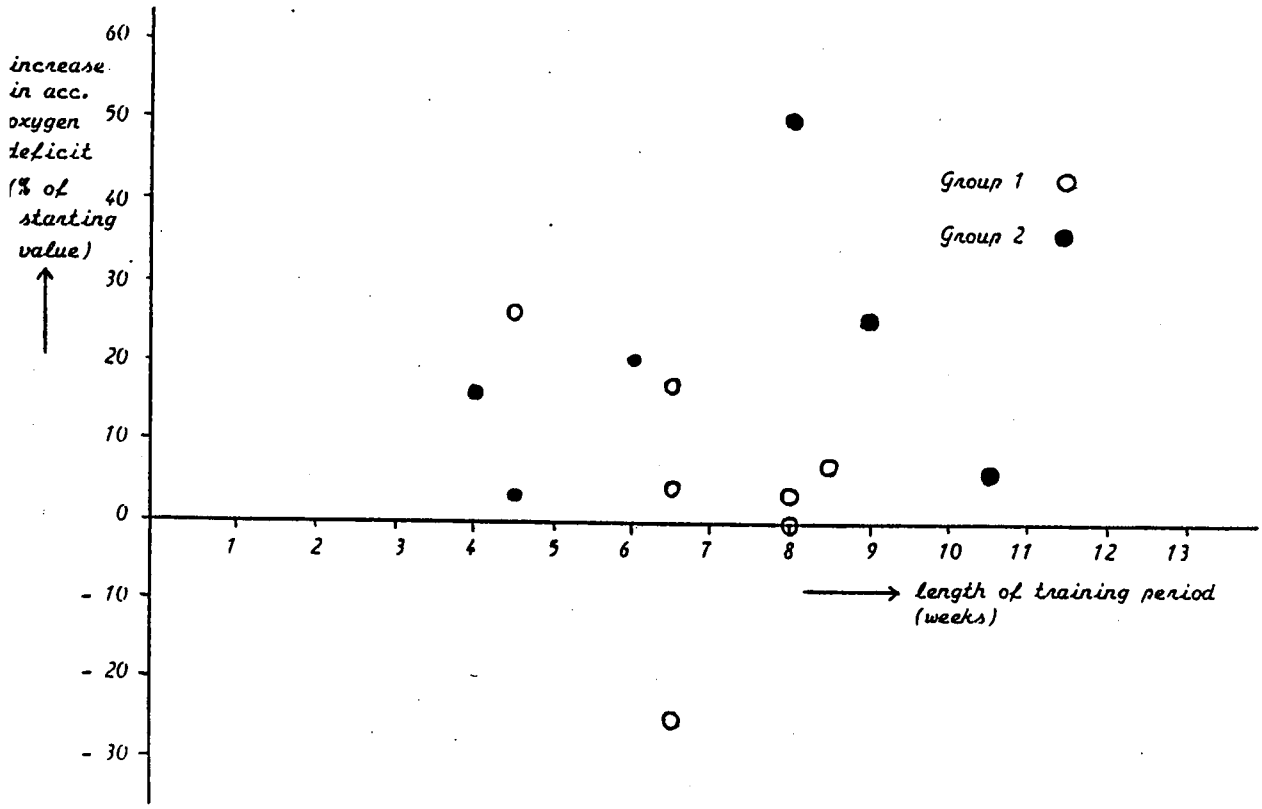

Fig. 13 : Increase in maximal accumulated oxygen deficit in percentages of starting value versus length of training period.

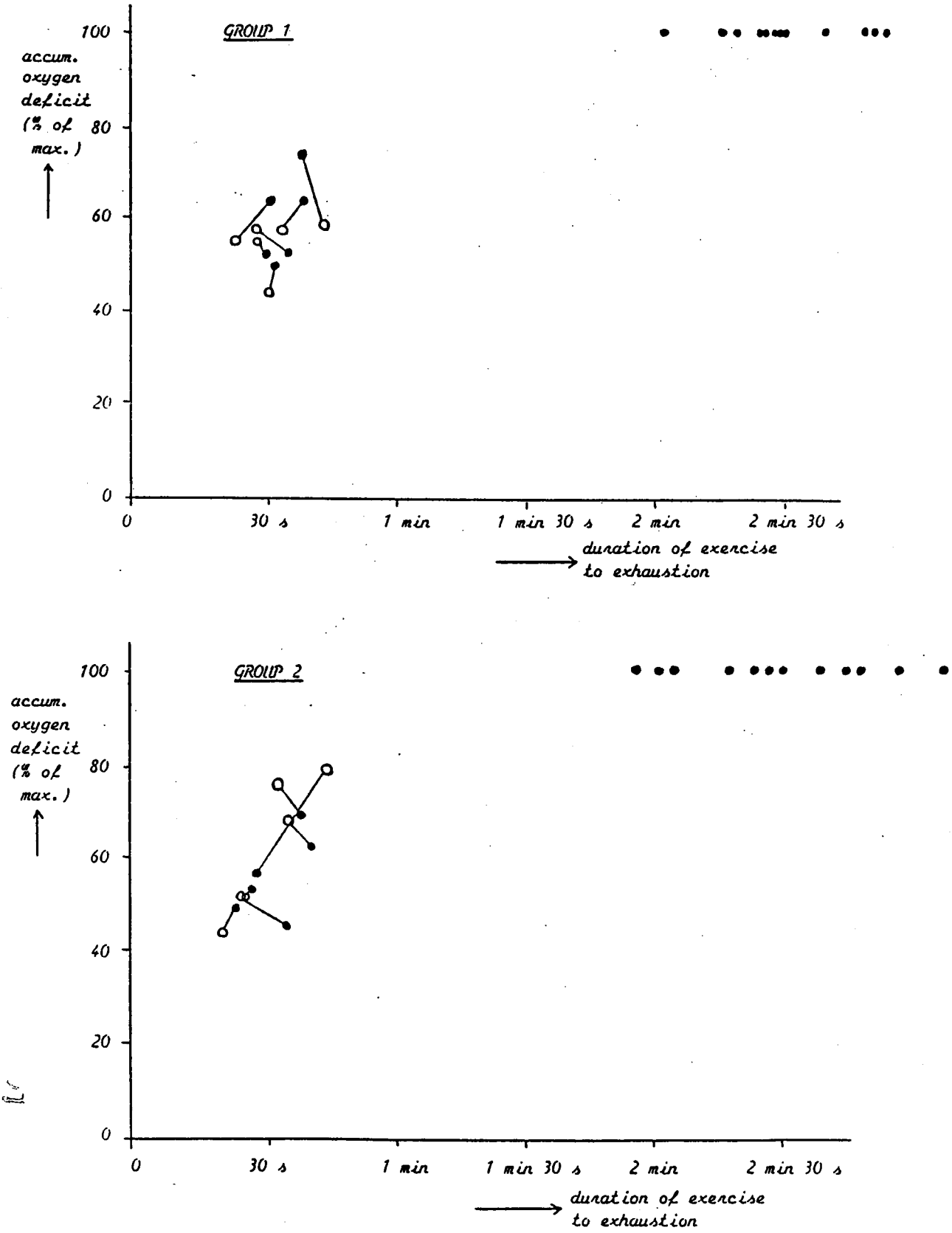

Fig. 14 : Relation between accumulated oxygen deficit (in percentages of maximal) and duration of exhausting exercise before (o) and after the whole training period (•), plotted on the curve of ref.(6)(fig. 5).

V DISCUSSION

The improvement in maximal accumulated oxygen deficit after the training period is statistically significant for both groups: group 1 (GL eliminated) : $p = 0.035$ and group 2 : $p = 0.01$. (In the addendum I shall explain why I eliminated GL)
Can we therefore conclude that it is possible to improve the anaerobic capacity by means of physical exercise ?
I recall the definition of anaerobic capacity that I used in the introduction : anaerobic capacity is the maximal amount of ATP (energy) formed by anaerobic processes.
The performances of the subjects in the exhausting runs improved, that is they ran faster and/or for a longer time. Thus they were able to supply more energy, since their running economy did not change markedly. Their oxygen uptake did not increase very much, in contrast to their accumulated oxygen deficit. So the amount of energy formed by processes in which oxygen is not used (= the anaerobic capacity), increased.
If we assume that the subjects in every anaerobic capacity test (2-3 minutes to exhaustion) really ran <u>to exhaustion</u>, then we can conclude that it is indeed possible to improve the anaerobic capacity.

What kind of changes in the subjects' bodies (or minds) might have caused this improvement ?
In chapter II (Biochemical backgrounds) I have already mentioned possible limiting factors on the anaerobic capacity :

1. It is possible that the energy store in the muscles becomes larger because of :
   a) thickening of muscle fibers
   b) just a larger store of ATP, creatine phosphate or glycogen in the muscle fibers.

   Although it has been found that it is not the total amount of glycogen stored in the cells that limits the anaerobic energy production (because there is still about 70 % left after exhausting exercise), a larger glycogen store could provide for a larger anaerobic energy production, assuming that still about 30 % is used.
2. It can also be postulated that, through training, the muscles mobilize more than 30 % of their glycogen stores, because of an increase in the amount of enzymes necessary for anaerobic energy production.
3. It has often been hypothesized that the buffer capacity of muscle and blood is the main limiting factor in the anaerobic energy production. That is why lactate levels and pH in blood and muscle are supposed to be a measure of anaerobic capacity.

   In my experiments I did not find an increase in maximal capillary peak blood lactate concentration at the end of the training period, which could be an argument against this theory. But since it is only a small percentage of muscle lactate that comes into the blood, and since there is an inaccuracy because of inter- and intra-individual differences in distribution volume of

the lactate, it is better not to reject this theory, but to reject the method of measuring the anaerobic capacity by determining the capillary blood lactate level.

4. The increased breathing during strenuous exercise does not only provide the body with more oxygen, but also results in an increased removal of carbondioxide from the blood, which results in a reduced decrease in blood pH.
5. Finally, I mention psychological factors, which are very important when it comes to running to exhaustion.

In the ADDENDUM I will discuss the probable causes of improvement in anaerobic capacity (maximal accumulated oxygen deficit) for every subject individually.

Training specificity

By giving the subjects two different types of training I hoped to find out whether there was a difference in the effect on the anaerobic capacity.
One would expect that the form of training that corresponds best to the test would have the greatest effect on the test performance, because of training specificity. Thus the subjects of group 1, who trained in bouts of 2 minutes , should have improved their anaerobic capacity more than the subjects of group 2, who trained in bouts of 20 seconds.
However in this research the subjects of group 2 achieved a greater improvement in anaerobic capacity than the subjects of group 1, but the difference between the two groups was not large enough to reach statistical significance ($p = 0.1$).
In the last decade middle distance runners show a tendency away from training methods based on endurance ("from endurance to speed") to training methods based on development of speed ("from speed to prolonged speed").(2)(4) Middle distances are 800 and 1500 meters. The world´s best performance on 800 meters in 1983 was 1.43.87; the world record on 1500 meters was 3.33.13.(4)
The better performance in the 2-3 minutes to exhaustion run of group 2 (that trained in short, fast bouts) compared to group 1, is in accordance with the new training concepts of middle distance runners.

The relation between accumulated oxygen deficit (in percentages of maximal) and the duration of exhausting exercise did not change, neither in group 2 nor in group 1. Since the total anaerobic capacity (maximal accumulated oxygen deficit) in both groups increased, we can conclude that the accumulated oxygen deficit in the 30 seconds run also increased. Thus training of short, fast runs does not only improve the accumulated oxygen deficit in short runs (lasting about 30 seconds), but also in runs where the

total anaerobic capacity is needed (2-3 minutes runs). Likewise training of longer runs (about 2 minutes) does not only improve the maximal accumulated oxygen deficit, but also the accumulated oxygen deficit in short runs (about 30 seconds).
Thus the theory of training specificity (a certain form of training will have the best effect on performance that corresponds best to that form of training) is contradicted by these results as well.

Research on trainability of anaerobic capacity

To improve further research results on trainability of anaerobic capacity one should first of all work with a greater number of subjects. They should all train on the treadmill, so that their training intensity is accurately known. This intensity must be the same for all subjects and can be determined by using a certain percentage of their maximal oxygen uptake.
I suggest a training of 6 times 30 seconds on 165 % of maximal oxygen uptake, carried out 3 times a week.
Because the subjects of group 2 improved their anaerobic capacity most, the best training concept is probably much like the training of this group. I would advise slightly slower and longer bouts, because many subjects of group 2 complained of pain in their legs after a few weeks training. They said that the speed was too high for their untrained legs. Some of them dropped out because of this.
I would suggest to diminish the number of bouts from 8 to 6, because it became quite boring to run so many times.
When it comes to determination of anaerobic capacity, the maximal accumulated oxygen uptake of at least 2 exhausting runs carried out in the same week should be measured.
If possible, muscle biopsies should be taken, in which lactate, buffering capacity, pH, enzymes and glycogen can be determined. Only then it is possible to find out something about the physiological changes that cause the increase in anaerobic capacity.

VI EVALUATION

The aim of this research project was to get acquainted with research methods. This should contribute to the understanding of the scientific background of medicine and it should aid in planning a future career.
By doing this research project all these aims have been achieved. I have worked with subjects, collected and analyzed air, performed biochemical analyses (blood lactate analyses) and also gained experience with calculations of data obtained in measurements and with some statistical methods to evaluate the results.
Apart from doing the project about the trainability of the anaerobic capacity, I got the opportunity to do some measurements and experiments concerning the anaerobic threshold, of which I learned very much.
I would like to compliment my supervisor at the Institute of Muscle Physiology in Oslo, Jon Ingulf Medbø, on the way he guided me, giving me much independence and making me carry out my project on my own responsibility. He was always interested in my work and prepared to help (by explaining complicated literature, helping with the measurements and analyses, or just by bringing me a cup of tea when I was too busy to get some tea myself).

I hope that this little research project may be the start of a career in exercise physiology or sports medicine.

VII ACKNOWLEDGEMENTS

I would like to thank the Institute of Muscle Physiology in Oslo, Norway, for having given me the opportunitiy to do this project, especially Ole Sejersted and Jon Ingulf Medbø. I appreciate their assistance and support, as well as that of all other "colleagues" at the Institute of Muscle Physiology.
Last but not least I want to thank the subjects who participated in the experiments.

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IX ADDENDUM

ANALYSIS OF THE RESULTS OF EVERY SUBJECT INDIVIDUALLY

Let us have a look at every subject in the research project separately to see if we can say something about the causes of their improvement in anaerobic capacity.

Group 1:

1. The maximal accumulated oxygen deficit of EK decreased to the starting value after showing an increase of 7 % after 4 weeks training. I do not have an explanation for this. Perhaps there was an inaccuracy in the calculation of the accumulated oxygen deficit, because of a relatively large worsening in running economy during the training period.
2. Subject KE was well trained when she started in the experiments. This may be the cause of a relatively small increase in anaerobic capacity, because of the general principle of levelling off of improvement of performance during a period of training; when an untrained person starts to train in for example high jumping, he will jump progressively higher during the first months. But after a while it becomes more and more difficult and it takes more and more training to jump still higher.
3. KG had an average improvement in anaerobic capacity. She started at a very low value, but did not show any better or worse training results than the other subjects who had a higher starting value of their anaerobic capacity.
4. DHA had much day to day variability in running economy, probably because of the many long distance runs he did in the first month of testing. But on the test days (the exhausting runs) he had about the same running economy, measured during the warming ups prior to the exhausting runs, so that I assume that I used the right figures in the calculation of his accumulated oxygen deficit.

5 + 6. The anaerobic capacity of TK and KN increased enormously, I think partly because of psychological factors. Especially TK seemed to have difficulties exhausting himself. Also this can be learned.

7. GL is the problem case in this research. I am absolutely sure that he did not really run to exhaustion in the test after the first training period, but I cannot prove it. He himself told me that he was not in a very good shape on the day of this test. However I think that the most important reason for this bad result is that the oxygen uptake (aerobic capacity) had increased so much (2.5 ml/kg/min, which was 5 % of his starting value). In my experiments the subject can see on a stopwatch how long they have been running and they can also see the speed of the treadmill. GL wanted, like all the subjects, to improve his first performance on the exhaustion test. Indeed he succeeded to run much longer at the same speed. Unfortunately, the improvement of his

performance was entirely due to an increase of the aerobic capacity. I was hoping that my assumption that GL did not run to exhaustion during the second test, would be confirmed in the last test after the second training period. But he got injured and dropped out.

Group 2:

8. ILH had a worse performance after the first training period than before she started training. In the 30 seconds run she showed an extremely high accumulated oxygen deficit compared to the performance in the 2-3 minutes test. This means that for some reason she did not run to complete exhaustion of her body capacity in the 2-3 minutes test after the first training period. This explanation of the apparent worsening of her anaerobic capacity is confirmed by her performance after the second training period, which showed an extremely high increase in accumulated oxygen deficit of 38 %.
9. Although KF only trained twice a week, she still improved her anaerobic capacity (a little). So it does not seem necessary to train at least three times a week to achieve an improvement in anaerobic capacity. Whether the cause of this improvement is mainly physiological or psychological cannot be said.
10. SG was the subject that showed the steepest grade of improvement in accumulated oxygen deficit. We can conclude that this is partly caused by psychological factors, because of her performances in the 30 seconds run. It is far easier for the subjects to recognize when they are exhausted in the 30 seconds run than in the 2-3 minutes run. In the 30 seconds run the legs suddenly cannot move quickly enough anymore. In the 2-3 minutes run it is mainly the breathing that hurts and it is hard to say when exactly the feeling of heavy breathing and tired legs becomes unbearable.
11. The increase in accumulated oxygen deficit of SM was only small, although he trained intensively on the treadmill.
12. SL had a roughly average improvement in anaerobic capacity. He trained outside on his own.
13. TN showed about an average improvement in maximal accumulated oxygen deficit. This subject had a very bad running economy (measured at sub-maximal intensities). At all these intensities his running economy was roughly the same. However I think that he ran more economically at higher speeds, because of an expected better running technique, when running much faster. So if we estimate his oxygen demand at an exhaustion test (at a supra-maximal intensity) by extrapolating the linear relation between running speed and oxygen uptake, the oxygen demand is overestimated if the running economy of this subject at this high intensity is better than at lower intensities. Thus the absolute values of the accumulated oxygen deficit that I calculated (using the usual extrapolation of the running economy line) are probably too high. Still I think that it is

possible to use these values if we compare them to other values of this individual subject.