

# Comparing VO<sub>2</sub>max Improvement in Five Training Methods

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## Abstract

This paper presents a theoretical study comparing the improvement of the whole body maximum oxygen uptake (maximum aerobic power or VO<sub>2</sub>max) when training at different intensities and with different methods matched for total work performance and frequency of training. We compare VO<sub>2</sub>max improvement in five training methods, testing Helgerud et al.'s (2007) experimental study against Moxnes and Hausken's (2008) theoretical model for athletic performance, fitness, and fatigue. The five methods are long slow distance running (LSD), lactate threshold running (LT), 15/15 sec interval training, 4x4 min interval training, and running at 87.5% of VO<sub>2</sub>max for 22.17 min. A weight function that scales the importance of aerobic utilization and stroke volume utilization is established.

**Keywords:** VO<sub>2</sub>max, aerobic utilization, stroke volume, performance, fitness, fatigue, Trimp intensity

## 1 Introduction

### 1.1 Overview

Whole body maximal oxygen uptake (maximum aerobic power, aerobic capacity,  $VO_{2max}$ ) of human beings has interested researchers for many years and regular reviews have been published (Saltin and Strange 1992, Nielsen 2003). Its absolute magnitude and malleability with physical training has practical interest for elite soldiers, for the sports elite, and for anyone involved in physical exercise. Important for the interest in the topic is also that determination of oxygen uptake not only is a measure of aerobic energy turnover, but also offers precise measure of the capacity to transport and utilize oxygen, i.e., the functional capacities of the lungs, cardiovascular system and muscle mitochondria combined. In general aerobic power has been recognized as one of the fundamental components of physical performance and health (Åstrand and Rodahl 1986, Johnson 1991)<sup>1</sup>.

During exercise, the increase in metabolism is expressed as the whole-body oxygen uptake that increases with exercise intensity to reach a maximum ( $VO_{2max}$ ). In general the oxygen consumption ( $VO_2$ ) in humans seems to level off at around 6-7 liter oxygen /min and around 80-90 ml/min/kg for highly trained athletes (Saltin and Åstrand 1967, Rusko et al. 1978)<sup>2</sup>. Different steps in the oxygen ( $O_2$ ) chain from air to the cells are considered to contribute as a barrier for  $O_2$  utilization. The first step of the oxygen ( $O_2$ ) transport chain is the conductive transport of  $O_2$  from the expired air to the alveoli (R1). This conductive transport from inspired to alveolar gas is next followed by a diffusive flow of  $O_2$  from the alveoli to the arterial blood (R2). The next step is a conductive transport of  $O_2$  to the cells where  $VO_2 = Q (Ca - Cv)$  (R3).  $Q$  is the cardiac output and  $Ca$  and  $Cv$  is the capillary and venous  $O_2$  content respectively. We can also write that  $(Ca - Cv) = \beta b (Pa - Pv)$ , where  $\beta$  is the mean slope of the blood  $O_2$  dissociation curve<sup>3</sup>,  $b$  is the solvability of  $O_2$  in the blood<sup>4</sup>.  $Pa$  is the arterial  $O_2$  pressure while  $Pv$  is the venous  $O_2$  pressure. Thus  $VO_2 = Q \beta b (Pa - Pv)$  (R3). A next step

<sup>1</sup> Three major factors accounting for aerobic endurance is simply the  $VO_{2max}$ , the work economy, and the capacity to sustain a high fractional utilization of the aerobic power (Pate and Kriska 1984). The role of the oxidative capacity of the muscles might be most important during submaximal work of long duration and when relatively small muscle mass is activated (long distance running or skiing).  $VO_{2max}$  might be the most important determinant of performance when large muscle mass is activated during maximal work from several minutes to 1 h (typically cross-country skiing) (Rusko et al. 1978). There is a strong correlation between running performance and  $VO_{2max}$ , when the groups investigated are heterogeneously trained athletes. However, in populations with more homogeneously trained athletes, the correlation with running performance is relatively weak. World class cross-country skiers had extremely high  $VO_{2max}$  values, and  $VO_{2max}$  per unit time per unit mass raised to the power of 2/3 seems to be a fairly accurate determinant of the performance ability of elite cross-country skiers (Ingjer 1991).

<sup>2</sup> A linear increase in aerobic power induced by a strenuous program of endurance exercise has been observed without any tendency to flatten out (Mikesell and Dudley 1984, Hickson et al. 1977).

<sup>3</sup> The blood  $O_2$  dissociation curve i.e., the percentage saturation of  $O_2$  as a function of  $O_2$  pressure shifts to the right for increasing temperature, pH or carbon dioxide content.

<sup>4</sup> In mammalian blood the amount of physically dissolved oxygen is around 0.2 ml  $O_2$  per 100 ml blood, while the amount bound to hemoglobin is around 20 ml  $O_2$  pr 100 ml blood. In water the amount physically dissolved is around 0.5 ml/100 ml water. The venous  $O_2$  content is typical around 6 ml  $O_2$  per 100 ml blood. This gives an extraction of  $(20-6)/20 = 70\%$ . For elite skiers extraction up to 93% can be achieved (Calbet et al. 2005).

in the O<sub>2</sub> chain is the diffusive transport of O<sub>2</sub> from the muscle capillary to the mitochondria (R<sub>4</sub>). The final step is the utilization of O<sub>2</sub> in the mitochondria (R<sub>5</sub>). Thus all together it can be written that  $VO_{2max} = (P_i - P_m) / (R_1 + R_2 + R_3 + R_4 + R_5)$ , where  $P_i$  is the partial pressure of O<sub>2</sub> in expired air, and  $P_m$  is the partial pressure of O<sub>2</sub> in the mitochondria. During maximal exercise with large muscle groups it has been found that R<sub>3</sub> is around 50% of (R<sub>1</sub>+R<sub>2</sub>+R<sub>3</sub>+R<sub>4</sub>+R<sub>5</sub>). Let Q<sub>max</sub> be the maximal value of the cardiac output Q. Using the numbers above it follows that an increase of Q<sub>max</sub> x beta by 10% gives an increase in VO<sub>2</sub>max of 5%. It has been found that R<sub>4</sub> and R<sub>5</sub> contribute around 7% each. Thus at maximal exercise the cardiac output seems to be the major factor in determining the oxygen delivery. R<sub>1</sub> and R<sub>2</sub> sum to 36% (Wagner 2000, di Prampero 2003).

The amount of trainability of the parameters R<sub>1</sub>-R<sub>5</sub> is important. For soldiers and elite athletes, or indeed for anyone engaged in athletic activity, it is important to know how intensity, duration, frequency of training and the initial fitness level of the individual relate to VO<sub>2</sub>max enhancement through time. When comparing trained and untrained college students with Olympic athletes, the overall dominant difference is the cardiac output Q and the systemic arteriovenous O<sub>2</sub> difference (Ca-Cv). For control students and Olympic athletes the VO<sub>2</sub>max is 3.30 and 5.38 Liter O<sub>2</sub> per minute respectively (Blomqvist and Saltin 1983). This is a factor 1.63 in advance of the Olympic athletes. The cardiac output Q was 20 and 30.4 liter blood per minute respectively, which gives a factor of 1.52 in advance of the Olympic athletes. The Ca-Cv =74% and 80% respectively, giving a factor of 80/74= 1.08 in advance for the Olympic athletes. A total factor of 1.52 x 1.08=1.64 is achieved. This leaves little room for trainability of R<sub>1</sub>+R<sub>2</sub>+R<sub>4</sub>+R<sub>5</sub> which indeed is roughly the same factor 1.63/1.64=1.0. However, the maximum stroke volume<sup>5</sup> of the heart was 104 ml for the students and 167 ml for the Olympic athletes. This gives a factor of 1.6 in advance of the Olympic athletes. The Olympic athletes have a somewhat smaller maximum heart rate of 182 compared to 192 for the students: 192/182= 1/1.52 x 1.6. The volume of oxygen consumed during physical exercise is dependent upon the load on the muscles and also on the mass of muscles at work. If maximum heart rate is achieved the engaged muscle mass is usually sufficient. However, even for maximum heart rate, the VO<sub>2</sub>max often differs across activities. Usually Ca-Cv could be much smaller for instance in swimming that in running, leaving much room for improvement (Åstrand and Saltin 1961). A change in the Ca-Cv difference could be due to either a redistribution of blood flow and/or increased O<sub>2</sub> extraction from the blood by skeletal muscle as such<sup>6</sup>.

The early studies did not address the cardiac output Q and the arteriovenous O<sub>2</sub> difference Ca-Cv separately for the VO<sub>2</sub>max enhancement. However, many years ago it was suggested that longer duration and higher intensity may be required to increase Ca-Cv compared to the cardiac output Q (Kilbom 1971). This hypothesis was supported by Cunningham and Hill (1975) showing that a period of 9 weeks training resulted in significant increase in VO<sub>2</sub>max

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<sup>5</sup> The volume of blood pumped per beat.

<sup>6</sup> A relationship has been observed between VO<sub>2</sub>max and muscle fiber types indicating that a subject with a high VO<sub>2</sub>max will most probably have a high percentage of slow twitch fibers in the exercising muscles (Bergh et al. 1978, Rusko et al. 1978). See also Calbet et al. (2004) for extraction studies on skiers.

which was due to a larger stroke volume with little change in Ca-Cv. However, when training for 1 year a significant increase in Ca-Cv was found. So indeed, the cardiac output Q and the arteriovenous O<sub>2</sub> difference Ca-Cv are the two key parameters for trainability of VO<sub>2</sub>max. For a specific oxygen uptake say 3 liter per minute (below VO<sub>2</sub>max), training increases the arteriovenous O<sub>2</sub> difference Ca-Cv, and reduces the cardiac output Q (Ekblom et al. 1968). However, Cunningham and Hill (1975) found that training increases the cardiac output for the same VO<sub>2</sub>. Gledhill et al. (1994) found that trained persons had a cardiac output of  $Q = 7.2 + 5.9 \times VO_2$  (liter/min) while untrained persons had  $Q = 6.6 + 5.1 \times VO_2$  (liter /min). The heart rate at the same oxygen uptake is reduced by training while the stroke volume is much the same (Ekblom et al. 1968), or higher (Cunningham and Hill 1975). The stroke volume seems to be increasing with VO<sub>2</sub> (Cunningham and Hill 1975, Gledhill et al. 1994, Warburton and Gledhill 2008). However, for untrained persons the stroke volume seems to plateau at a heart rate of around 120 beats per minute. It is hypothesized that at high heart rates the progressive diminishing time available for diastolic filling limits stroke volume, causing a plateau. However, for trained athletes, because of an enhanced myocardial contractility, less time in cardiac cycle would be required for ventricular emptying, so that more time would be available for blood filling (Gledhill et al. 1994)<sup>7</sup>.

From an evolutionary perspective it would be relevant to study the effect of different training programs when matched for total metabolic energy consumption per training session<sup>8</sup>. Although a large body of scientific work has been reported on the effect of VO<sub>2</sub>max on training, relatively few articles have compared the response of training when matched for total metabolic energy (or total work) per session. Interval (IT) and continuous (CT) training of various variants constitute a base of line of training regimes to improve physical fitness. The VO<sub>2</sub> associated with submaximal CT reflects mainly ATP production from aerobic mechanisms. Oxidative processes predominate and are associated with lipid utilization, little anaerobic glycolysis utilization or creatine phosphate depletion. Furthermore there is predominant slow-twitch fiber recruitment with this type of exercise. With IT exercise VO<sub>2</sub> represents the addition of two terms VO<sub>2</sub> during work, and VO<sub>2</sub> during recovery. For the work periods, the intensity is near 100% or higher and a large O<sub>2</sub> deficit is manifest. This is reflected by a high ATP utilization from anaerobic mechanisms, and consequently a high muscle ATP, CP and myoglobin depletion and high lactate production. There is a partially recovery of ATP, CP and myoglobin during recovery. Lactate is removed during the rest intervals via oxidation and glycogen synthesis in liver, kidney heart and skeletal muscles. The intensity work load in IT results in a high rate of fast-twitch fiber recruitment. IT may change motor unit recruitment patterns and induce specific biochemical adaptations in fast-twitch fibers. In general it is not known whether IT or CT stimulates cardiac output and systemic arteriovenous O<sub>2</sub> difference differently. Presumably a good measure for increased cardiac output is increased stroke volume, which should correlate with decreased resting heart rate. A good measure for systemic arteriovenous O<sub>2</sub> increase could be enhancement in the VO<sub>2</sub> per

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<sup>7</sup> The stroke volume and its function of the utilization of VO<sub>2</sub>max or maximum heart rate is under intense discussion. See also Vella and Roberts (2004).

<sup>8</sup> We set that match by total work or match by total energy expenditure to be much the same.

unit cardiac output. One would hypothesize that IT with oscillations in VO<sub>2</sub> throughout the exercise and pause periods might better enhance the O<sub>2</sub> kinetic response.

Roskam (1967) studied the effect of CT and IT training matched by total work. Soldiers cycled 5 times a week for 4 weeks for. The CT group was most effective in decreasing the heart rate at rest and in the low and middle intensity. However the IT group was more effective in improving the maximum work performance. Knuttgen et al. (1973) studied three groups running 3 times per week for 8 weeks. The groups were somewhat matched for work performance. One group performed strenuous exercise for 15 seconds and 15 seconds rest. A second group performed 3 minute strenuous exercise and 3 minutes rest. The first group increased the VO<sub>2</sub>max by a factor of 1.16, while the second group increased the VO<sub>2</sub>max by a factor 1.26 after 8 weeks. Fox et al. (1973) studied two groups that run for 7 weeks 3 times a week. The groups were matched for work. The first group runs 50-200 meter fast pace sprint, while the second group runs 600-1200 meters at relatively slow pace running. The VO<sub>2</sub>max increased by a factor of 1.09 for the first group and a factor of 1.05 for the second group. It was concluded that muscular hypoxia per se is an important stimulus for the improvement of maximal aerobic power. Fox et al. (1977) studied two groups that ran 3 times a week for 8 weeks. The workouts for the high power group consisted of a maximum of 19 high intensity runs at 30 seconds duration alternated with relief or recovery intervals long enough in duration to allow the heart rate to return between 120 and 140 beats/min. The workout for the low intensity group consisted of a maximum of 7 lower intensity runs of 2-min duration with rest relief intervals lasting until the heart rates returned to between 120 and 140 beats /min. The groups were matched for work. It was found that low power and high power output interval training programs elicit similar changes in maximal aerobic and anaerobic metabolism. Eddy et al. (1977) studied subjects participating in a training program upon bicycle ergometer for 7 weeks with training 4 days a week. The CT group trained at 70% VO<sub>2</sub>max and the IT group trained at 100% VO<sub>2</sub>max for 1 minute and 1 minute rest. CT and IT training produced identical changes in heart rate response, blood lactic acid concentration and VO<sub>2</sub>max when the total work load was equated per training session. The CT group and the IT increased the VO<sub>2</sub>max with a factor 1.15 and 1.14 respectively. Lesmes et al. (1978) studied groups that run IT with different intensity 4 times a week for 8 weeks. One group runs short distances at 50- 200 meters with an intensity of around 170% VO<sub>2</sub>max. The other group runs distances of 600-1200 meters at intensity of 170%. The high intensity group increased the VO<sub>2</sub>max by a factor of 1.15, while the low intensity group increased the VO<sub>2</sub>max by a factor of 1.20. Gregory (1979) studied two groups that run 5 times a week for 6 weeks. The training was matched for total work performance. The CT group runs at the heart rate of 162, while the IT group runs 4 minutes and rests 4 minutes with heart rate 174. The CT group increased the VO<sub>2</sub> at a given pulse by a factor of 1.20 while the IT group increased the VO<sub>2</sub> with a factor 1.24. Cunningham et al. (1979) designed a study on cycling to determine the effect of CT and IT programs on the cardiac (Q) and the peripheral (Ca-Cv) response to training. The total work was matched between the groups. Cunningham et al. (1979) studied two groups. The CT group cycled for an intensity of 70% of VO<sub>2</sub>max for 20 minutes 4 times per week for 12 weeks. The IT group cycled with an intensity of 90-100% of VO<sub>2</sub>max for 2 minutes with a 1 minute rest period. Both groups demonstrated similar and significant increase in Ca-Cv (leading to an VO<sub>2</sub>max enhancement of around 20%), but only a marginal

stroke volume enhancement. It was suggested that the cycling utilized a too small muscle mass compared to jogging, and may therefore place a lesser demand on the heart as a pump to emphasize extraction of oxygen by the working muscles. Fourier et al. (1982) compared skeletal muscle adaption in sprint and endurance training. It was found that CI increased VO<sub>2</sub>max, glycolytic phosphofructokinase (PFK), oxidative succinate dehydrogenase (SDH), ST and FT muscle fiber area. Sprint training only results in significant enhanced PFK. Thomas et al. (1984) found that interval training may benefit aerobic capacity more than continuous running in young adults who have moderately high initial fitness level. They in particular studied continuous 4 miles running at 75% of maximum heart rate, and 8 sets of 1 minute running and 3 minutes walking with 90% of maximum heart rate in the running period. The energy consumption was around 500 kcal per session. The enhancement in VO<sub>2</sub>max after three sessions per week in 12 weeks was 6.9% for continuous running and 10.3% for the interval training. Poole and Gaesser (1985) found that for the group training continuously for 55 minutes at 50% of VO<sub>2</sub>max 3 days a week, the VO<sub>2</sub>max increased by 15% after 8 weeks. The group training continuously for 35 minutes at 75% increased the VO<sub>2</sub>max by 20%. The group training interval 2 minutes at 105% VO<sub>2</sub>max with 2 minutes rest walking, 10 repetitions, increased the VO<sub>2</sub>max by 15% (MXX:have number for 4 weeks also 1.17, 1.12, 1.16). The groups were matched for work performance. Gorostiaga et al. (1991) studied two groups. One group cycled continuously for 50% of VO<sub>2</sub>max 20 minutes 3 days a week for 8 weeks. The group that trained interval trained 30 seconds at 100% VO<sub>2</sub>max, and 30 seconds rest, for 20 minutes. The increase in VO<sub>2</sub>max was 7 and 11% respectively. Overend et al. (1992) investigated the change in VO<sub>2</sub>max during 10 weeks 4 times a week of training on cycle ergometer. The group performing continuous cycling for 40 minutes at 80% of VO<sub>2</sub>max increased the VO<sub>2</sub>max by 8%. The group performing interval training in 3 minutes at 100% VO<sub>2</sub>max and 2 minutes at 50% VO<sub>2</sub> max increased the VO<sub>2</sub>max by 9%. The other group performing interval training for 30 seconds at 120% VO<sub>2</sub>max and 30 seconds at 40% VO<sub>2</sub>max increased the VO<sub>2</sub>max by 17%. All the groups were matched for work. Edge et al. (2005) compared CI and IT training. The CI performed 20-30 minutes at VO<sub>2</sub>max of 80-95%. The IT performed sprints at 120-140% VO<sub>2</sub>max for 2 minutes. The groups trained 3 times a week for 5 weeks. The VO<sub>2</sub>max increased with around 11% for the IT group and around 9% for the CI group.

All together, the results in the literature are somewhat conflicting with regards to the effect of IT or CT training on VO<sub>2</sub>max. Intensity and quantity of work performed during the training program appeared to be more important than any differences in training-type protocol. VO<sub>2</sub>max increases with intensity when holding work constant (Burke and Franks 1975), and with time holding intensity constant (Miles et al. 1976). For a review see Wenger and Bell (1986). It becomes appropriate to quantify training load (TL) for work and intensity. To quantify TL for work and intensity, different methods have been used. For endurance the concepts for quantifying the training load has been the training impulse (Trimp) (Banister et al. 1975, Banister et al. 1984, Morton et al. 1990, Taha and Thomas 2003). Trimp is the cumulated exercise duration multiplied with a weight function that depends on the heart rate, VO<sub>2</sub> or the lactate level (see section 3 for further discussion). The model has been used in running (Morton et al. 1990), cycling (Busso et al. 1991, Busso et al. 1997), swimming (Banister et al. 1975), weight lifting (Busso et al. 1994) and triathlon (Millet et al. 2002).

### 1.2 This paper's contribution

This paper uses a fitness function that synthesizes all positive effects causing increased performance, and a fatigue function that synthesizes all negative effects causing fatigue (Moxnes and Hausken 2008). Banister et al. (1975) define performance as fitness minus fatigue, whereas this paper defines performance as fitness multiplied with an exponential decay function that accounts for fatigue. During exercise, fatigue increases more than fitness and performance decrease. After exercise, during the recovery, fatigue decreases faster than fitness and performance increase, and reaches a higher level than before exercise. If a new exercise occurs before complete recovery from the preceding one, the negative effect due to fatigue would be amplified, and the recovery time needed would be longer than if there was a longer gap between exercise sessions. This paper considers exercise induced muscle damage as one possible realistic variable descriptive of fatigue. Analogously, we consider muscle fitness to account for fitness. But, this includes the heart muscle which provides a link so that the paper models maximum stroke volume of the heart as performance. We compare Helgerud et al.'s (2007) experimental study of four training methods, together with a fifth training method presented in this paper, against Moxnes and Hausken's (2008) theoretical model for athletic performance, fitness, and fatigue. An athlete's training impulse (Trimp) is developed and used in the analysis. The Trimp intensity which is defined as the Trimp per unit time, equals the aerobic utilization, multiplied with a weight function which scales the importance of stroke volume utilization. The analysis implies developing a weight function for stroke volume enhancement which is compared with Banister et al.'s (1985/1986) exponential weight function.

Section 2 presents Helgerud et al.'s (2007) four training methods and a fifth training method. Section 3 determines energy consumption and training impulse. Section 4 conducts an empirical study of Helgerud et al.'s (2007) four training methods using heart rate monitors. Section 5 develops a mathematical model of performance (maximum stroke volume). Section 6 simulates the model. Section 7 simulates alternative training methods. Section 8 concludes.

## **2 The five training methods**

Helgerud et al. (2007) analyzed the effect of four different training methods on VO<sub>2</sub>max and stroke volume. The four groups ran three sessions per week for eight weeks on a treadmill with 5.3% upward incline. Each session started with 10 min warmup at 60% of VO<sub>2</sub>max, and ended with three minutes of cooldown. Each session comprised warmup, exercise, and cooldown.

a) "Long slow distance running" (LSD) at 60% of VO<sub>2</sub>max for 45 min. Hence warmup and cooldown have the same VO<sub>2</sub>max percentage as the exercise. b) "Lactate threshold running" (LT) at 80% of VO<sub>2</sub>max for 24.25 min. c) "15/15 interval training" (15/15) at 87.5% of VO<sub>2</sub>max, 47 times, each period lasting 15 sec. Exercise proceeded at 60% of VO<sub>2</sub>max during the 46 rest periods, each lasting 15 sec. d) "4x4 min interval training" (4x4) at 87.5% of

VO<sub>2</sub>max, four times, each period lasting 4 min. Exercise proceeded at 60% of VO<sub>2</sub>max during the rest periods, each lasting three min.<sup>9</sup>

55 non-smoking and well trained male university students took part in the study. Their mean weight was 82 kg, with VO<sub>2</sub>max 54 ml/min/kg before the study. This gives a maximum aerobic power  $e_M = 1500\text{W}$ <sup>10</sup>. Before the study the participants trained 3 times per week. The exercise regime for the groups was matched such that their total energy consumption including warmup, exercise, rest periods, and cooldown was equivalent in all sessions. This gives 5.9 km running distance per session around for around 130 L oxygen. This gives around  $130 \times 1000 \times 20 = 2.6 \times 10^6 \text{ J} = 622 \text{ kCal}$ .

Helgerud et al. (2007) found that the stroke volume decreased with 1% for LSD, increased with a factor 1.0093 for LT, increased with a factor of 1.094 for 15/15, and increased with a factor 1.104 for 4x4 (Table 2 in Helgerud et al. (2007)). They found that VO<sub>2</sub>max decreased with 0.6% for LSD, and increased with 2.0% for LT, 5.5% for 15/15, and 7.2% for 4x4 (Table 1 in Helgerud et al. (2007)). All groups had improvement in the running economy (ml/meter kg<sup>0.75</sup>), that is 8% for LSD, 12% for LT, 8% for 15/15, and 10% for 4x4. Somewhat surprisingly, no changes were found in the lactate threshold for any of the groups.

This implies that 4x4 and 15/15 increase VO<sub>2</sub>max, but not that 4x4 and 15/15 are optimal training method in the sense maximum improvement in VO<sub>2</sub>max per session for a given energy consumption per session.

### 3 Determining energy consumption and training impulse

The energy consumption during a training session is defined by

$$\text{Energy}(t) \stackrel{\text{def}}{=} \int_{t_0}^t e(u) du = e_M \int_{t_0}^t \frac{e(u)}{e_M} du \quad (3.1)$$

Where “def” means definition,  $t_0$  is the start time,  $t$  is the stop time, and  $e(t)$  and  $e_M$  are aerobic power (rate of energy) and maximum aerobic power, respectively, relating proportionally to VO<sub>2</sub> and VO<sub>2</sub>max. We define the ratio  $e(t)/e_M$  as the utilization of maximum aerobic power, referred to as aerobic utilization. This gives

<sup>9</sup> The five training methods are described in terms of the percentage of VO<sub>2</sub>max, and not in terms of the percentage of maximal heart rate.

<sup>10</sup> We set that 1ml O<sub>2</sub> corresponds to 20 J.



$$\begin{aligned}
 Energy_{LSD} &= 60 e_M \left( \underbrace{45 \times 0.6}_{Run} \right) s = e_M 1620s = 2.58 \cdot 10^6 J, e_M = 1590W \\
 Energy_{LT} &= 60 e_M \left( \underbrace{10 \times 0.6}_{Warmup} + \underbrace{24.25 \times 0.8}_{Run} + \underbrace{3 \times 0.6}_{Cooldown} \right) s = e_M 1632s = 2.49 \cdot 10^6 J, e_M = 1527W \\
 Energy_{15/15} &= 60 e_M \left( \underbrace{10 \times 0.6}_{Warmup} + \underbrace{47 \times 0.25 \times 0.875}_{47 Run} + \underbrace{46 \times 0.25 \times 0.6}_{46 Cooldown} + \underbrace{3 \times 0.6}_{Cooldown} \right) s = e_M 1499s \\
 &= 2.45 \cdot 10^6 J, e_M = 1637W \\
 Energy_{4 \times 4} &= 60 e_M \left( \underbrace{10 \times 0.6}_{Warmup} + \underbrace{4 \times 4 \times 0.875}_{4 Run} + \underbrace{3 \times 3 \times 0.6}_{3 Cooldown} + \underbrace{3 \times 0.6}_{Cooldown} \right) s = e_M 1632s \\
 &= 2.48 \cdot 10^6 J, e_M = 1520W \\
 Energy_{LQD} &= 60 e_M \left( \underbrace{10 \times 0.6}_{Warmup} + \underbrace{22.27 \times 0.875}_{Run} + \underbrace{3 \times 0.6}_{Cooldown} \right) s = e_M 1632s \\
 &= 2.48 \cdot 10^6 J, e_M = 1520W
 \end{aligned} \tag{3.2}$$

Multiplication with 60 is to convert to time in seconds. Equation (3.2) also determines values for a fifth hypothetical training method (subscript LQD) referred to as “long quick distance running” where the participant runs at the same VO2max percentage as under 15/15 and 4/4, that is 87.5% of VO2max, but without rest periods. There is a warm up of 10 minutes and a cool down of 3 minutes. Such a training method is common for ambitious participants, and is easily performed by athletes during competition. The runner runs for 22.17 min, chosen such that the runner consumes the same energy as under 4x4. Equation (3.2) implies

$$Energy_{LT} / Energy_{LSD} = 0.97, Energy_{15/15} / Energy_{LSD} = 0.95, Energy_{4 \times 4} / Energy_{LSD} = 0.96 \tag{3.3}$$

The four groups trained with different training stimuli per session, defined with the common Trimp (Training impulse, see Moxnes and Hausken (2008) and references)

$$\underbrace{Trimp(t)}_{\text{Training impulse}} \stackrel{def}{=} \int_{t_0}^t \underbrace{w \left( \frac{e(u)}{e_M} \right)}_{\text{Weight function}} \underbrace{\frac{e(u)}{e_M}}_{\text{Utilization of max aerobic power}} du \tag{3.4}$$

A weight function is given by Banister et al. (1985/1986) and Morton et al. (1990), that is  $w(x) = w_0 \text{Exp}(bx) = \text{Exp}(b(x-1))$  when  $w_0 = \text{Exp}(-b)$ ,  $0 \leq x \leq 1$  (3.5)

The weight function is exponential to ensure large training impulse (Trimp) when the utilization is large. We define the Trimp intensity as the derivative of Trimp with respect to time. The parameter b is estimated by Morton et al. (1990) to be b=1.92. Without loss of generality we set  $w_0 = \text{Exp}(-b)$  so that the weight function is 1 for x=1, and less than 1 when  $0 \leq x < 1$ .

The Trimp values then become

$$w(0.6) = 0.464, w(0.8) = 0.681, w(0.875) = 0.787, b = 1.92$$

$$Trimp_{LSD} = 60 \left( \underbrace{45 \times 0.6 \times 0.464}_{Run} \right) s = 752s$$

$$Trimp_{LT} = 60 \left( \underbrace{10 \times 0.6 \times 0.464}_{Warmup} + \underbrace{24.25 \times 0.8 \times 0.681}_{Run} + \underbrace{3 \times 0.6 \times 0.464}_{Cooldown} \right) s = 1010s$$

$$Trimp_{15/15} = 60 \left( \underbrace{10 \times 0.6 \times 0.464}_{Warmup} + \underbrace{47 \times 0.25 \times 0.875 \times 0.787}_{47 Run} + \underbrace{46 \times 0.25 \times 0.6 \times 0.464}_{47 Cooldown} + \underbrace{3 \times 0.6 \times 0.464}_{Cooldown} \right) s \quad (3.6)$$

$$= 895s$$

$$Trimp_{4 \times 4} = 60 \left( \underbrace{10 \times 0.6 \times 0.464}_{Warmup} + \underbrace{4 \times 4 \times 0.875 \times 0.787}_{4 Run} + \underbrace{3 \times 3 \times 0.6 \times 0.464}_{3 Cooldown} + \underbrace{3 \times 0.6 \times 0.464}_{Cooldown} \right) s$$

$$= 1028s$$

$$Trimp_{LQD} = 60 \left( \underbrace{10 \times 0.6 \times 0.464}_{Warmup} + \underbrace{22.17 \times 0.875 \times 0.787}_{Run} + \underbrace{3 \times 0.6 \times 0.464}_{Cooldown} \right) s = 1133s$$

Obviously LT and 4x4 give higher Trimp than LSD and 15/15. But the hypothetical training method LQD gives the highest Trimp, and thus the highest Trimp per consumed energy unit.

Presumably the parameter b can be substantially higher than 1.92 for trained athletes. For instance when b=10, (3.6) becomes

$$w(0.6) = 0.0183, w(0.8) = 0.135, w(0.875) = 0.286$$

$$Trimp_{LSD} = 29.7s, Trimp_{LT} = 166s, Trimp_{15/15} = 193s, Trimp_{4 \times 4} = 255s, Trimp_{LQD} = 342s \quad (3.7)$$

LT gives lower Trimp than 15/15 which gives lower Trimp than 4x4. The weight function is especially important for the relative comparison of the Trimp values. However, we will show that the exponential function will not apply for our dataset.

#### 4 Empirical study of four training methods (excluding LQD) using heart rate monitors

In addition Helgerud et al. (2007) measured the heart rates as functions of time for four participants for the four training methods (one participant per method). We have digitized these data. We scale the curves to obtain aerobic power as functions of time. The relationship between utilization of maximum heart rate  $HR/HR_M$  and the utilization of the maximum aerobic power  $e/e_M$  is given by

$$HR / HR_M = 0.25 + 0.75(e / e_M) \Rightarrow e / e_M = (-0.25 + HR / HR_M) / 0.75$$

Figure 2 shows the results for the four training methods.

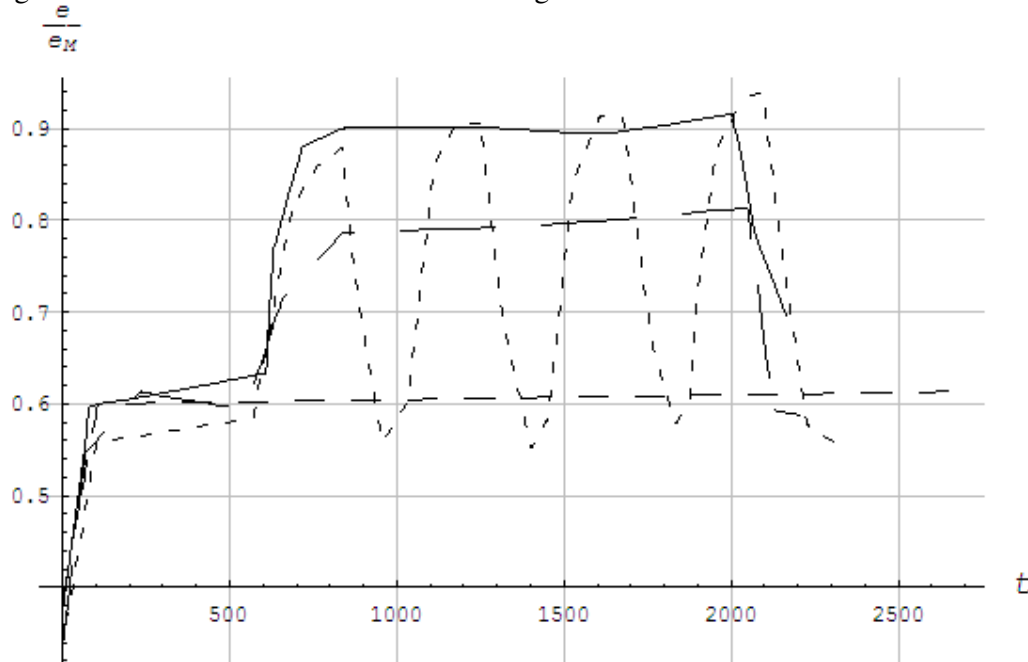


Figure 2: The utilization of maximum aerobic power as functions of time in seconds for the four training methods.

— : LSD — — — :LT .....:15/15 - . - :4x4

The curves have been smoothed somewhat compared with the experimental results. Integrating the curves gives the energy consumption per session, that is

$$Energy_{LSD} = e_M 1621s, Energy_{LT} = e_M 1601s, Energy_{15/15} = e_M 1737s, Energy_{4x4} = e_M 1623s \quad (4.1)$$

Increased time for 15/15 can be due to the Polar heart rate monitors ([www.polarusa.com](http://www.polarusa.com)), programmed to measure the mean heart rate every five seconds, not registering the actual heart rate decrease during the 15 sec of rest. Applying the heart rate data for  $b=1.92$ , the Trimp values become

$$Trimp_{LSD} = 757s, Trimp_{LT} = 974s, Trimp_{15/15} = 1273s, Trimp_{4x4} = 1007s \quad (4.2)$$

15/15 gives the highest Trimp, which is doubtful since the heart rate monitors possibly do not register the actual heart rate decrease during the 15 sec of rest. Helgerud et al. (2007) additionally conducted a pilot study where they measured the O2 consumption of eight

participants for the four training methods (two participants per method), where  $e_M = 1600W$ . They found

$$\begin{aligned}
 Energy_{LSD} &= 131.0L O_2 = 2.62 \cdot 10^6 J = e_M 1638s, \\
 Energy_{LT} &= 128.1L O_2 = 2.56 \cdot 10^6 J = e_M 1601s, \\
 Energy_{15/15} &= 133.6L O_2 = 2.67 \cdot 10^6 J = e_M 1670s, \\
 Energy_{4x4} &= 127.3L O_2 = 2.55 \cdot 10^6 J = e_M 1591s
 \end{aligned} \tag{4.3}$$

$Energy_{15/15}$  in (4.3) matches (4.1) better than (3.2). The above results give Table 1.

Table 1: Energy/ $e_M$  for the four training methods using different calculation methods.

	From (3.2)	From (4.1)	From pilot study (4.3)
LSD	1620s	1621s	1638s
LT	1632s	1601s	1601s
15/15	1499s	1737s	1670s
4x4	1632s	1623s	1591s

The results in Table 1 are consistent across the three methods aside from 15/15. For 15/15 we use columns 1 and 2 to illustrate the difference. For the other training methods we do not use the heart rate measurements in column 2 and only use column 1.

## 5 Mathematical model of performance (maximum aerobic power)

We now proceed to determine performance defined as muscle strength, expressed as stroke volume in this paper. We use Moxnes and Hausken's (2008) equations for muscle growth, which are based on the two fundamental principles for muscle growth: "train to failure" and "use it or lose it". We set that performance in the model is maximum stroke volume  $V_M(t)$ , i.e.  $p(t) = V_M(t)$ ,  $p_0 = V_M(t_0)$ . Differentiating (3.4) and inserting performance as stroke volume gives  $\dot{T}(t) = w(V(t)/V_M(t))V(t)/V_M(t)$ , where  $V(t)$  is the stroke volume during exercise. Helgerud et al.'s (2007) study does not report the stroke volume during a training session, only the percentage of maximum aerobic power. However, a one to one relationship most likely exists between the stroke volume and the aerobic power since the stroke volume most likely increases monotonically with the aerobic power (Gledhill et al. 1994). Assume that the utilization of the maximum heart rate can be described as a function of the utilization of maximum stroke volume as  $HR/HR_M = F(V/V_M)$ ,  $F(1) = 1$ , for instance as in Figure 2 of Gledhill et al. (1994). The utilization of the maximum aerobic power as a function of the utilization of stroke volume is then

$0.25 + 0.75 e/e_M = F(V/V_M) \Rightarrow e/e_M = (F(V/V_M) - 0.25)/0.75$ . Since the  $F()$  function is not known for the athletes in Helgerud et al.'s (2007) study we describe the Trimp intensity

(and the weight function) as a function of the utilization of maximum aerobic power in this paper, although we suggest a curve showing the weight function as a function of the utilization of the maximum stroke volume. From Moxnes and Hausken (2008) we achieve

$$\begin{aligned}
 \underbrace{\dot{h}(t)}_{\text{Derivative failure}} &= \alpha \underbrace{\dot{T}(t)}_{\text{Trimp intensity}} - \beta \underbrace{h(t)}_{\text{Failure}}, (a) & \underbrace{\dot{g}(t)}_{\text{Derivative fitness}} &= \underbrace{\mu h(t)(1-\dot{T}(t))}_{\text{Anabolic process due to failure}} - \underbrace{\nu g(t)}_{\text{Catabolic process}}, (b) \\
 \underbrace{p(t)}_{\text{Performance}} &= g(t) \text{Exp}(-\eta h(t)), (c), & \dot{T}(t) &= w(e(t)/e_M(t))e(t)/e_M(t), (d)
 \end{aligned}
 \tag{5.1}$$

where  $\alpha, \beta, \mu$  and  $\nu$  are positive parameters with dimension 1/time. In each training session  $e(t)/e_M(t)$  changes in a complicated way due to variation in  $e(t)$  and variation in the  $e_M(t)$  due to muscle failure. However, for  $\dot{T}(t)$  we can neglect the variation in  $e_M(t)$  in each training session. Equation (5.1) assumes anabolic and catabolic processes. High Trimp intensity causes microscopic ruptures (failure) in the form of anabolic substances in the muscle, which decrease asymptotically over time when training ceases, as expressed in (5.1a). The muscle growth is limited during training if the Trimp intensity is too high. The main part of the muscle growth occurs over time outside the training sessions when the body recuperates. Without muscle ruptures, the muscle volume decreases asymptotically over time, as expressed in (5.1b). Muscle strength is a function of muscle volume and the amount of muscle ruptures, as expressed in (5.1c). That is, performance is a function of fitness and failure. The steady state solution of (5.1) is

$$h_{ss} = \frac{\alpha \dot{T}}{\beta}, g_{ss} = \left( \frac{\mu \alpha}{\nu \beta} \right) (1 - \dot{T}) \dot{T}, p_{ss} = h_{st} \text{Exp}(-\eta g_{ss})
 \tag{5.2}$$

## 6 Simulating the model

The unit of failure is arbitrary and we assume  $\alpha = 1/24$  without loss of generality, using time in hours as a unit. We determine  $\beta$  by assuming that muscles usually heal over 1-2 days after usage. We thus assume  $\beta = 0.5/24$  and use time in hours as a unit. The unit of performance is also arbitrary and we assume  $\mu = 1/24$  without loss of generality, using time in hours as a unit. We consider the muscle volume reduction to be a result of catabolic processes in the body. Around 0.3% protein is released from the body per day. We thus assume  $\nu = 0.003/24$ . Moxnes and Hausken (2008) assume  $\eta = 0.2$ , also assumed in this paper.

We use the Trimp values in Table 1 for the various training methods and insert the Trimp intensity into (5.1). The amount of Trimp is calculated according to the weight function. The training occurs over a short time period (ca. 30-40 min) compared with the time it takes for muscles to grow or degrade (several days). Due to numerical necessity we set that the Trimp

intensity for input into (5.1) is a Gaussian function spread over a fixed time period of standard deviation 2 hours. The Gaussian function is thus such that the integral over time equals the number of Trimps. Two uncertainties are the participants' physical fitness levels at the start of the test and the weight function. If the participants are well trained, and for example trained at the same level as the various training methods prior to testing, VO<sub>2</sub>max or stroke volume improvement cannot be expected. The participants trained three times per week in various forms of endurance training. There is reason to believe that the LSD training at 60% of VO<sub>2</sub>max for 45 min three times per week did not exceed the training level they were already used to. Improvement of stroke volume could thus not be expected, as also confirmed by the data. We let the weight function be variable, and sought to match the experiment visually. The participants are assumed to be in a "steady-state" situation at the start of the test. The participants' start condition is described with a "steady-state" form factor  $\lambda$  which we adjust to match the initial conditions. The initial conditions are

$$h(0) = \frac{\alpha \dot{T}(0)}{\beta}, g(0) = \left( \frac{\mu \alpha}{\nu \beta} \right) (1 - \dot{T}(0)) \dot{T}(0), \dot{T}(0) = \lambda \frac{75W}{e_M} w(75W / e_M) \tag{6.1}$$

where 75W is the basic metabolic power. The credibility of the solution depends on the credibility of the weight function. We define  $p_0 = p(0)$  as the initial performance (stroke volume) before the test, and  $p(t)/p_0$  as the performance ratio.

Figure 3 shows the five theoretically and experimental performance ratios  $p(t)/p_0$  for the training methods.

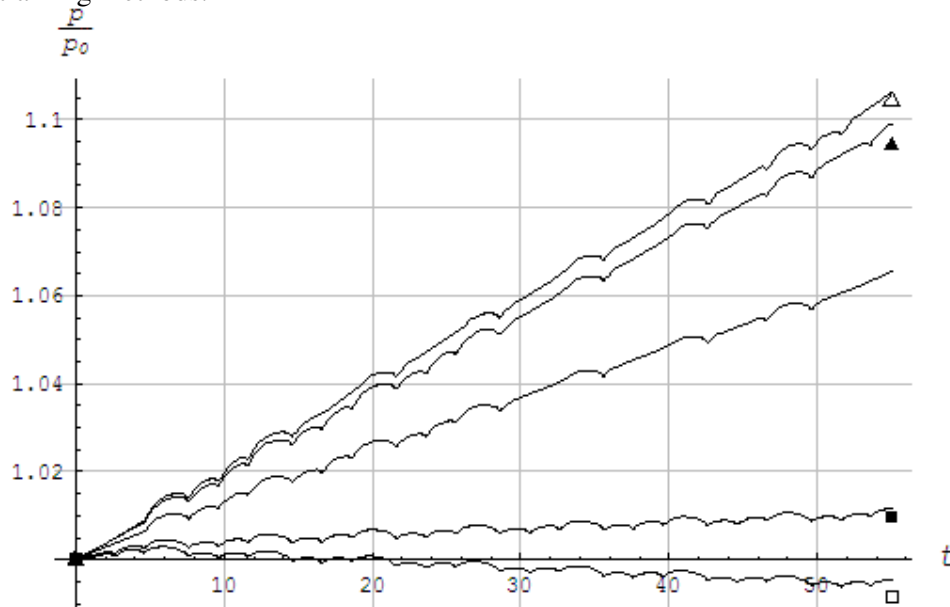


Figure 3: The performance ratios  $p(t)/p_0$  as functions of time in days over eight weeks assuming training on Monday, Wednesday, and Friday.

■: Exp. LT, □: Exp. LSD, ▲: Exp. 15/15, Δ: Exp. 4x4

*Theoretical curves from above: a) 4x4. b) 15/15 by use of heart rate data as given in (4.1). c) 15/1. d) LT. e) LSD.  $\lambda = 2.42$  in all cases.*

Helgerud et al.'s (2007) initial values are assumed to be valid when measurements start Monday morning at 00.00 o'clock referred to as the start point  $t=0$ , where  $t$  is measured in days. Helgerud et al. (2007) do not specify which days training occurs, but we assume training Monday, Wednesday, and Friday at 12 o'clock noon. Helgerud et al. (2007) do not specify when measurements cease in week eight, but we assume the last VO<sub>2</sub>max measurement occurs when  $t=55$ , which is at 24.00 o'clock Saturday in week eight.

The theoretical curves change approximately linearly over the eight weeks. For LSD, LT and 4x4 the weight function is adjusted to ensure match with the experimental data at day 55. This locks the weight function to three values and three corresponding utilizations. LSD gives  $w=0.20$  at  $e/e_M=0.6$ , LT gives  $w=0.23$  at  $e/e_M=0.8$ , and 4x4 gives  $w=0.50$  at  $e/e_M=0.875$ . The third largest theoretical increase occurs for 15/15 which gives lower increase than the experimental point (filled triangle). The likely reason is that the theoretical Trimp value is too low. We have earlier discussed uncertainties about the Trimp values for 15/15. The Trimp values depend on whether or not heart rate data are used in (4.1). The reason is that heart rate data for 15/15 gives the highest Trimp values. It is well known that heart rate data measured during interval training can give too high estimated energy consumption (Ballor and Volosek 1992). The upper curve for 15/15 in Figure 3 is found by adjusting the Trimp data for 15/15 with a factor of 1737/1499 according to Table 1 for heart rate data. Since the 15/15 data were not consistent on VO<sub>2</sub>max measurements and heart rate, they were not used in the fitting procedure.

The theoretical curves are linear in the long term, which matches J. Hoff's statement to the newspaper VG ([www.vg.no](http://www.vg.no)) July 11, 2008: "Experiments have been conducted upwards towards 18 interval sessions per week. We have never seen worse response than 0.25% VO<sub>2</sub>max increase per session."

Figure 4 shows with solid line the estimated weight function based on stepwise linear adjustment to the three data points generated by the experiments LSD, LT and 4x4. The three stapled lines show the Banister-Morton weight functions with  $b=1.9$  (upper curve),  $b=5$  (middle curve), and  $b=10$  (lower curve). The estimated weight function shows some resemblance with the Banister-Morton value  $b=5$ , but we found no solution by using the Banister-Morton exponential form of the weight function in (3.5).

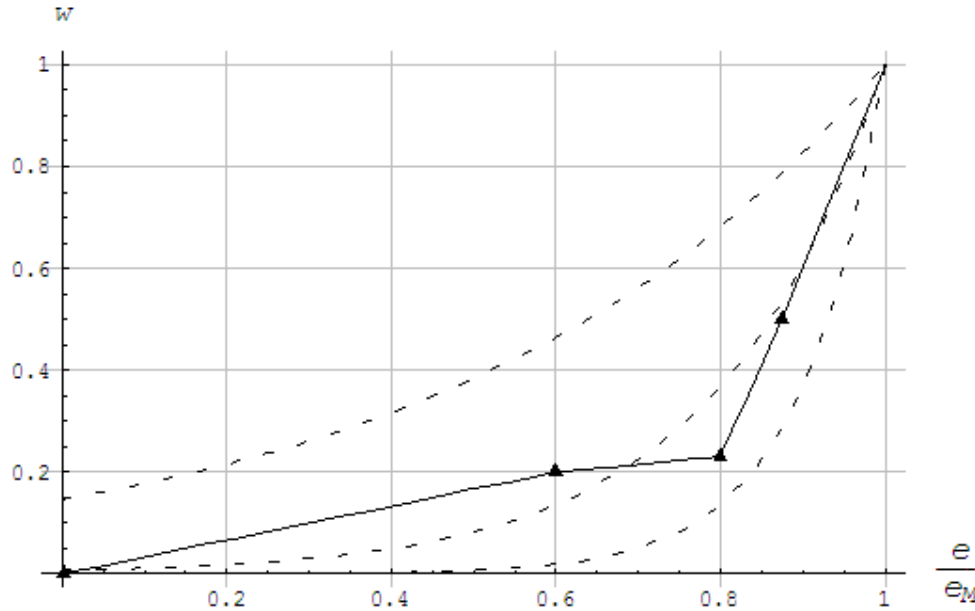


Figure 4: Four weight functions  $w$  as functions of the utilization of the maximum aerobic power. From above: a)  $b=1.9$  for Banister-Morton. b) Stepwise linearly adjusted to the three data points in the experiments LSD, LT and 4x4, c)  $b=5$  for Banister-Morton and d)  $b=10$  for Banister-Morton

In Figure 4 we have for the middle solid line specified with filled triangles the three experimental values of the weight function for the four intensities: rest ( $e/e_M=0.05, w=0.0$ ), LSD ( $e/e_M=0.6, w=0.20$ ), LT ( $e/e_M=0.8, w=0.23$ ) and 4x4 ( $e/e_M=0.875, w=0.50$ ). We can recalculate (3.2) to achieve

$$w(0.6) = 0.20, w(0.8) = 0.23, w(0.875) = 0.55$$

$$Trimp_{LSD} = 324s, Trimp_{LT} = 362s, Trimp_{4x4} = 562s, Trimp_{15/15} = 485s, Trimp_{LQD} = 676s \quad (6.2)$$

We find that the enhancement in stroke volume (or  $VO_{2max}$ ) follows the trend in Trimp.

Future research should assess how the weight function possibly could be linked to the heart's stroke volume. However, by using the curve for trained athletes in Figure 2 of Gledhill et al. (1994), Figure 4 can be plotted as function of the fraction of the stroke volume. The result is shown in Figure 5. We find that the weight function increases strongly around 0.8-0.9 of the maximum stroke volume. The somewhat conflicting results in the literature regarding how Trimp intensity causes or implies  $VO_{2max}$  enhancement can be clarified by, as shown in this



paper, by developing a relationship between utilization of maximum stroke volume and utilization of VO2max. If the stroke volume for untrained athletes only marginally increases with aerobic power, increased utilization of VO2max will probably not increase the load on the heart. It could be that the weight function, as a function utilization of aerobic power, actually mimics the utilization of the maximum stroke volume as a function of the utilization of maximum aerobic power.

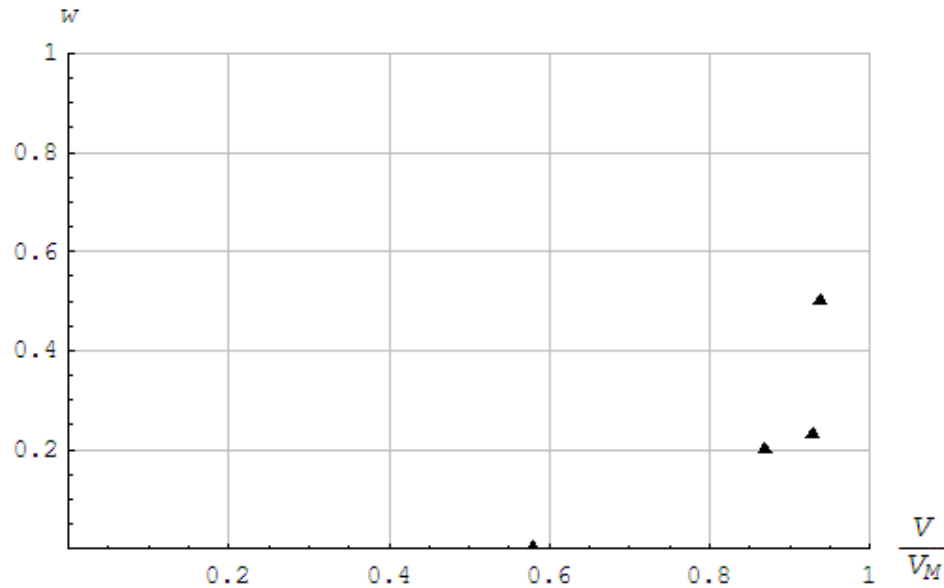


Figure 5: The weight functions  $w$  as functions of the utilization of the maximum stroke volume.

## 7 Simulating alternative training methods

Having estimated the form factor  $\lambda$  and established the weight function expressed with the middle solid line in Figure 4, our next step is to simulate alternative training methods. The participants are hypothetically assumed to double the length of each training session, and/or train seven days per week rather than three days per week.

Figure 6 shows the performance ratios  $p(t)/p_0$  as functions of time in days over 14 days when training at LSD Trimp intensity at a doubled duration or training every day. We find that the performance increases with around 12% when training with double duration and every day. We also see that training with double duration three days a week is much like training with ordinary duration every day.

Figure 7 shows the performance ratios  $p(t)/p_0$  as functions of time in days over 14 days when training at the LT Trimp intensity. We find pretty much the same qualitative and quantitative results as when training at LSD.

Figure 8 shows the performance ratios  $p(t)/p_0$  as functions of time in days over 14 days when training at the 4x4 Trimp intensity. Again we qualitatively find much the same as in Figures 5 and 6.

Figure 9 presents the performance ratio  $p(t)/p_0$  for LQD. For LQD we assume 10 min warmup and 3 min cooldown for a total of 35.17 min. We find that LQD gives the largest increase in stroke volume.

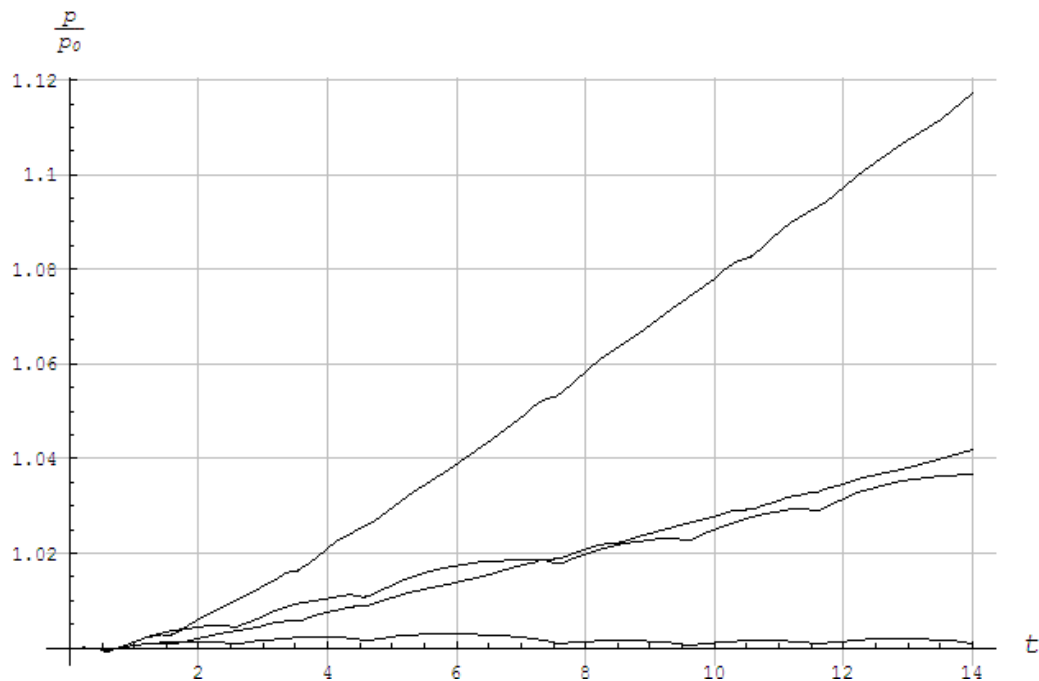


Figure 6: The performance ratios  $p(t)/p_0$ . Training at LSD VO<sub>2</sub>max percentage. From above: a) Training 2x45 min all days. b) Training 1x45 min all days. c) Training 2x45 min Monday, Wednesday, and Friday. d) Training 1x45 min Monday, Wednesday, and Friday.

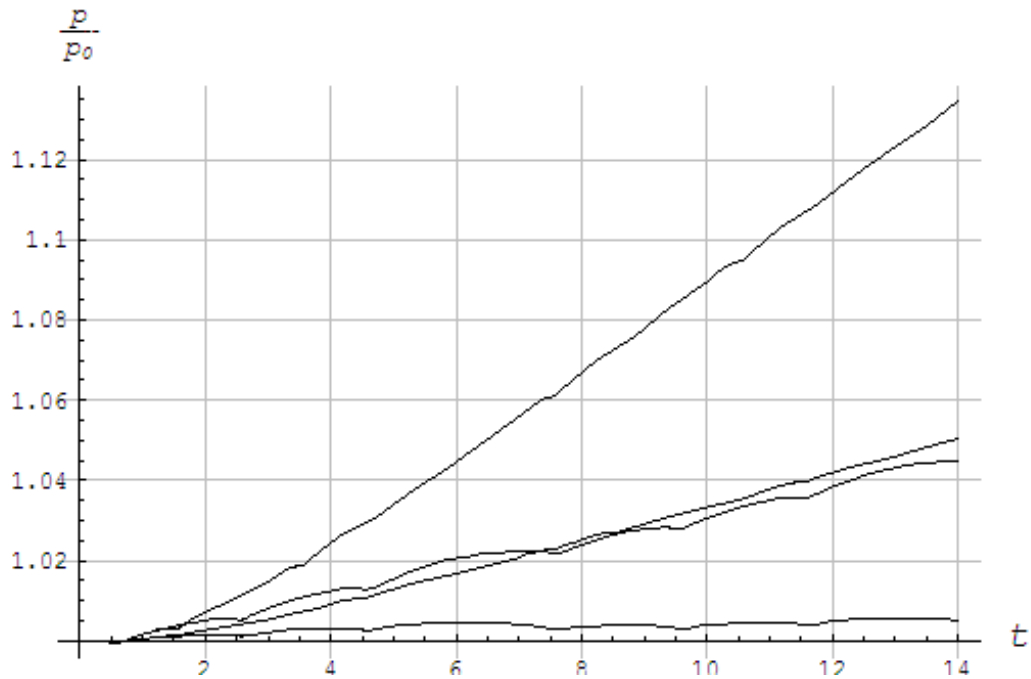


Figure 7: The performance ratios  $p(t)/p_0$  as functions of time in days over 14 days. Training at LT VO<sub>2</sub>max percentage.  
 From above: a) Training 2x37.25 min all days. b) Training 1x37.25 min all days. c) Training at 2x37.25 min Monday, Wednesday, and Friday. d) Training 1x 37.25 min Monday, Wednesday, and Friday. (LT training lasts 24.25 min. Adding 10 min warmup and 3 min cooldown gives 37.25 min.)

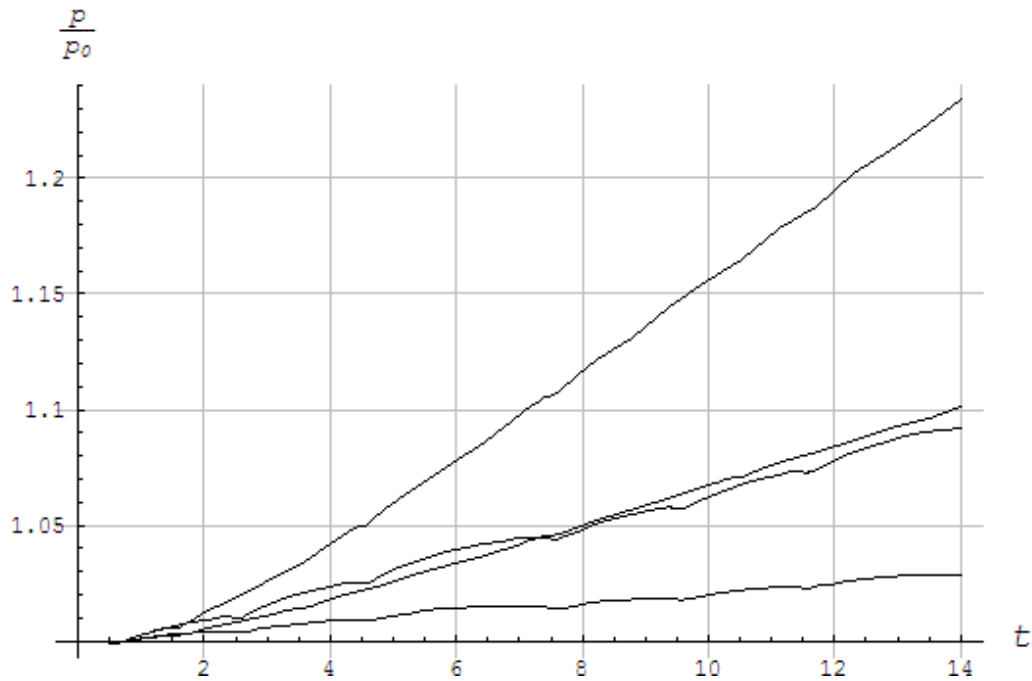


Figure 8: The performance ratios  $p(t)/p_0$  as functions of time in days over 14 days. Training at 4x4 VO<sub>2</sub>max percentage. From above: a) Training 2x38 min all days. b) Training 1x38 min all days. c) Training at 2x38 min Monday, Wednesday, and Friday. d) Training 1x38 min Monday, Wednesday, and Friday.

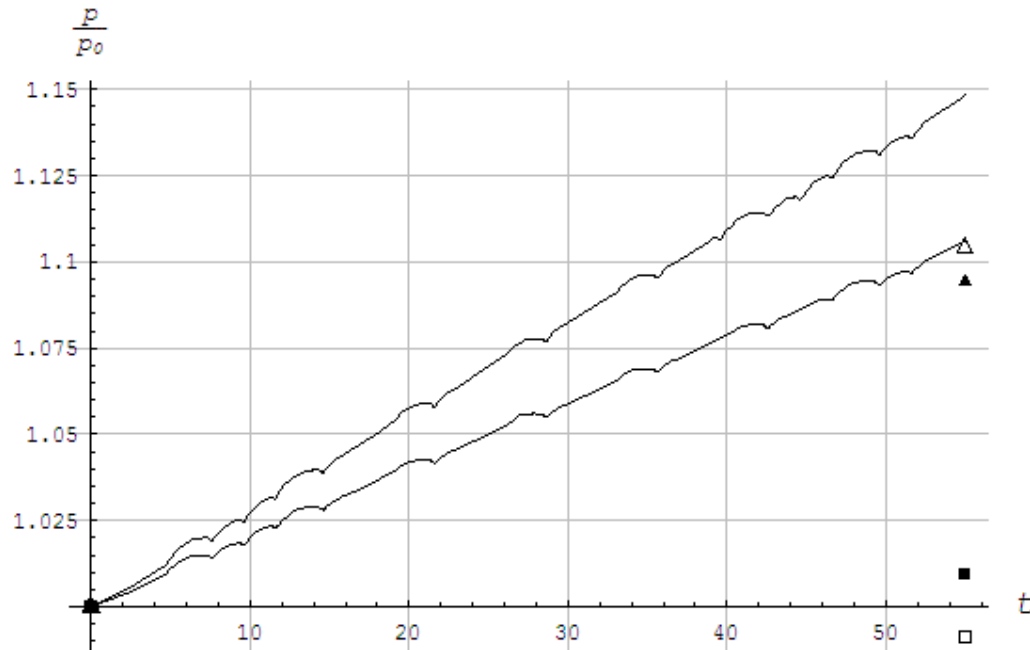


Figure 9 : The performance ratios  $p(t)/p_0$  as functions of time in days over eight weeks.

■: Exp. LT, □: Exp. LSD, ▲: Exp. 15/15, △: Exp. 4x4

Theory from above: a) Training at long quick distance running LQD 22.17 min Monday, Wednesday, and Friday. b) 4x4 Monday, Wednesday, and Friday.

## 8 Conclusion

Helgerud et al.'s (2007) experimental study has been tested against Moxnes and Hausken's (2008) theoretical model for athletic performance, fitness, and fatigue. The data in the experimental study are used to calibrate the model's parameters. Helgerud et al. (2007) tested four training methods, i.e. long slow distance running (LSD), lactate threshold running (LT), 15/15 sec interval training, and 4x4 min interval training. 4x4 min interval training gives higher training impulse per consumed energy unit than the other three methods.

This paper also presents a fifth training method which is to run at 87.5% of VO<sub>2</sub>max (which means 15/15 or 4x4 Trimp intensity without rest) for 22.17 min together with 10 warmup and 3 minutes cooldown, which is easily accomplished by well trained athletes or during competition. This fifth method gives better stroke volume improvement than Helgerud et al.'s (2007) four methods.

An athlete's training impulse defined as his Trimp is developed and used in the analysis. The time derivative of Trimp is the Trimp intensity which equals the aerobic utilization, multiplied with a weight function which scales the importance of both aerobic utilization and stroke volume utilization, integrated over the training period. Large Trimp intensity over time expresses large VO<sub>2</sub>max improvement.

We show that by training at LSD Trimp intensity seven days per week, rather than only three times per week, or training at LSD Trimp intensity for 90 min rather than 45 min three times per week, the stroke volume improves significantly over two weeks. In addition we find that training at double duration three days a week for stroke volume enhancement is much like training with ordinary duration every day.

We find that Banister et al.'s (1985/1986) exponential weight function is not descriptive, regardless which parameter is used for exponential increase. Instead we find the crucial result that the weight function increases slowly from 0 to 0.2 as the aerobic utilization increases substantially from 0 to 0.8. Thereafter the weight function increases sharply from 0.2 to 0.5 as the aerobic utilization increases moderately from 0.8 to 0.875. We think this is quite descriptive of an athlete's condition. When the objective is to increase VO<sub>2</sub>max, a weight function with low weight should be given to aerobic utilization below 80%, and a weight function with sharply increasing weight should be given to aerobic utilization within the range 80%-100%.

Due to the inherent uncertainties in the model, model parameters and experimental data, care must be taken when interpreting the results. Further studies are indeed needed to corroborate the result and especially determine the functional form of the weight function within the range 80%-100%. The kind of weight function descriptive of the arteriovenous O<sub>2</sub> difference Ca-Cv should also be tested.

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