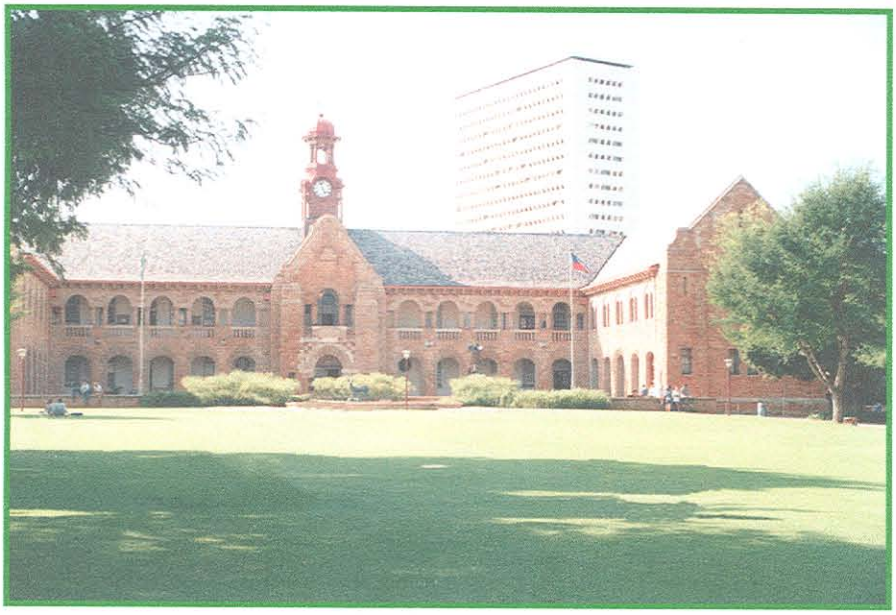
PHYSIOLOGICAL FACTORS ASSOCIATED WITH SUCCESSFUL COMRADES ATHLETES



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DEDICATION

This dissertation is dedicated to my husband Armand.

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SYNOPSIS

TITLE:	Physiological factors associated with successful Comrades Athletes
CANDIDATE:	E Heydenrych
PROMOTER:	Prof. P.E. Krüger
DEGREE:	MA(HMS)

Many scientific investigations have consistently identified the physiological variables that are positively related to successful endurance performance (Hawley, 1995). The extent to which these and other factors are "trainable" as opposed to genetically determined is a topic of considerable debate (Bouchard et al., 1992). Maximal oxygen uptake is the most common parameter used in exercise laboratories to estimate physical fitness. Costill et al. (1973) noted that subjects with similar VO_2 max values often performed quite differently in endurance events. To evaluate physical fitness more precisely, another parameter, reflection endurance capacity, should be determined in addition to VO_2 max (Vago et al., 1987).

Sport Science can play an important role in the success of ultra marathons by helping the athletes to achieve their optimal fitness levels. Science has given us a unique insight into the anatomy of one of the most difficult races in the world.

The purpose of the study was to assess experimentally the physiological status of a Comrades Marathon athlete and to examine the effect of training on the physiological parameters. The major objectives of the study were:

- to identify the physiological factors associated with successful endurance performance;
- to measure the effect of training on the VO_2 parameters on a regular basis;
- to provide training guidelines;

- to use heart rate monitors to guide training and optimise race performance; and
- to measure heart rate response during the Comrades Marathon race, thus to determine race intensity.

Five male marathon athletes volunteered to take part in the study. All the subjects were training for the 1998 Comrades Marathon and all of them had run the Comrades before. They had been following a training programme for a minimum of four years and were experienced treadmill runners. The first testing occurred eight months before the Comrades; thereafter, another five tests were undertaken. Their regular training regiments included weekly distances of at least 80 km, with workouts of moderate to high intensity. The day before each test, no intensive training was allowed.

Following an anthropometric evaluation, a maximal incremental treadmill test was undertaken to ascertain the VO_2 max and endurance fitness of the athletes. After a progressive warm-up phase, starting at a speed of 10 km/h, the speed was increased by 2 km/h every three minutes, until a speed of 16 km/h was reached. The treadmill speed was then increased by 1 km/h every two minutes until exhaustion. Blood lactate samples were collected during the test, at the end of each workload.

The physiological parameters measured were oxygen consumption (VO_2 max), maximal heart rate (HR), lactate threshold, ventilatory threshold, respiratory exchange ratio (R), and oxygen pulse. Each subject's best running times for the 10, 21, 42, 50, 56 and 90km had been recorded during the previous year. Training distances were also recorded during the testing period. Each subject ran the Comrades Marathon with a Polar Vantage NV to determine race intensity, heart rate response during the race, percentage below the lactate threshold and percentage above the lactate threshold.

The results indicated statistically that some of the VO_2 max parameters change during the eight months time period. It has been found however, that some of the maximum parameters did not change to a great extent (VE/VO_2 = 0.01%, VO_2/HR = 1.3%, VO_2 max = 3.54%, VO_2 absolute = 0.98%, RQ = 0.58%, VT = 4.31%, VE = 0.73%), speed and heart rate showed a decrease at the maximal exercise intensity (speed = -4.94%, heart rate = -4.37%). There was a

greater improvement at parameters measured at threshold level (VE/VO_2 = 1.45%, VO_2/HR = 5.43%, VO_2 max = 5.73%, VO_2 absolute = 6.62%, RQ = 1.70%, VT = 4.19%, VE = 7.78%, speed = 9.10% and heart rate = 6.34%).

Relationship between the lactate threshold and the actual heart rate response indicated that none of the athletes could complete a 90km race at the lactate threshold intensity. It has been found that the athletes could only keep their heart rate above a certain percentage of the lactate threshold during the duration of the race (30.3% above 95% of the lactate threshold, 58.3% above 90% of the lactate threshold and 77.3% above 85% of the lactate threshold).

The anthropometric data did not change much during the training months. The most substantial change could be seen in the fat percentage of the athletes (2.45% decrease). All of the athletes do however, showed a decrease in LBM (3.37%)

In conclusion it has been found that the athletes were not able to keep up with the heart rate just below the lactate threshold during the Comrades Marathon. As a result of worsened running economy, especially during the last 20km, none of the athletes could complete the race between 2-4-mmol/L lactate. It seems that more interval training and gymnasium work would be necessary to build enough of the stamina endurance that is an important parameter for Comrades athletes. Laboratory testing can help the athlete to optimise his running potential. Parameters of importance are an improvement at lactate threshold intensity and not at maximum intensity because those parameters simulate the race intensity.

LIST OF KEY WORDS:

COMRADES MARATHON, HEARTRATE RESPONSE, LACTATE THRESHOLD, VENTILATORY THRESHOLD, VO_2 max, RUNNING ECONOMY, AEROBIC EXERCISE, FATIGUE, RESPIRATORY EXCHANGE RATIO, VENTILATORY EQUIVALENT, VELOCITY.

SINOPSIS

TITEL:	Fisiologiese faktore wat met suksesvolle Comrades Marathon atlete geassosieer word
KANDIDAAT:	E Heydenrych
STUDIELEIER:	Prof. P.E. Krüger
GRAAD:	MA(MBK)

Sportwetenskaplikes bestudeer gereeld die fisiologiese veranderlikes wat geassosieer word met uithouvermoë prestasie (Hawley, 1995). Die mate waarin die veranderlikes ingeoefen kan word in teenstelling met genetiese bepaling is 'n onderwerp van debat (Bouchard et al., 1992). Maksimale suurstofverbruik is die algemeenste parameter wat in laboratoriums gebruik word om fisieke fiksheid te bepaal. Costill et al. (1973) het gemerk dat persone met dieselfde VO_2 maks waardes, verskillend presteer in uithouvermoë items. Om fisieke fiksheid meer presies te meet moet ander parameters wat aerobiese kapasiteit weerspieël, bepaal word tot aanvulling van VO_2 maks (Vago et al., 1987).

Sportwetenskap speel 'n belangrike rol in die sukses van ultra maratons om die atlete te help om hulle optimale fiksheidsvlakke te kan bereik. Wetenskap gee 'n unieke insig in die anatomie van een van die moeilikste wedlope in die wêreld naamlik die Comrades Marathon.

Die doel van die studie is om eksperimenteel die fisiologiese status van Comrades Marathon atlete te bepaal asook die effek van oefening op die fisiologiese parameters. Die hoof doelwitte was:

- om die fisiologiese faktore te identifiseer wat geassosieer word met uithouvermoë prestasie;
- die effek van oefening op die VO_2 parameters te bepaal op 'n gereelde basis;

- om oefen riglyne te voorsien;
- om hartmonitors te gebruik as 'n riglyn vir oefening asook om prestasie te optimaliseer; en
- om harttempo respons tydens die Comrades Marathon te meet, dus om wedloop intensiteit te bepaal.

Vyf marathon mans atlete het vrywilliglik aangebied om aan die studie deel te neem. Al die atlete het geoefen vir die 1998 Comrades Marathon en almal het voorheen al aan die Comrades deelgeneem. Al vyf atlete volg reeds 'n oefenprogram die afgelope vier jaar, en almal het al op 'n trapmeul gehardloop. Die eerste toetse het agt maande voor die Comrades plaasgevind; daarna is nog vyf toetse gedoen. Atlete se oefenprogram sluit afstande van ten minste 80 km per week in, teen hoë en gemiddelde intensiteite. Die dag voor die toetse was geen intensiewe oefeninge toegelaat nie.

Na die antropometriese evaluasie is 'n maksimale VO_2 maks toets uitgevoer om aerobiese potensiaal te bepaal. Na 'n progressiewe opwarmingsfase is die toets teen 10 km/hr begin. Elke las duur 3 minute waarna die spoed met 2 km/hr vermeerder word tot 16 km/hr bereik is. Daarna is die spoed met 1 km/hr verhoog elke 2 minute tot totale uitputting. Bloed laktaat monsters is geneem na elke inkrement en aan die einde van die toets.

Die fisiologiese parameters bepaal sluit maksimale suurstofverbruik (VO_2 maks), maksimale harttempo (HT), laktaat draaipunt, ventilatoriese draaipunt, respiratoriese kwosiënt (RK), en suurstof pols in. Beste 10, 21, 42, 50, 56 and 90km hardloop tye gedurende die laaste jaar bereik is genotuleer. Oefen afstande is ook weergegee tydens die toets periode. Al die atlete het die Comrades Marathon met 'n Polar Vantage NV voltooi om die intensiteit en harttempo respons gedurende die wedloop te bepaal asook persentasie bo en onder die laktaat draaipunt.

Die resultate het statisties aangetoon dat van die VO_2 maks parameters verander het gedurende die agt maande oefenperiode. Daar het egter in sommige van die parameters nie groot veranderinge plaasgevind nie (VE/VO_2 = 0.01%, VO_2/HT = 1.3%, VO_2 maks = 3.54%, VO_2 absoluut = 0.98%, RK = 0.58%, VT = 4.31%, VE = 0.73%), spoed en harttempo het beide 'n afname getoon tydens maksimale inspanning (spoed = -4.94%, harttempo = -4.37%). Daar was egter 'n groter verbetering by die parameters wat tydens laktaat draaipunt gemeet is

(VE/VO_2 = 1.45%, VO_2/HR = 5.43%, VO_2 maks = 5.73%, VO_2 absoluut = 6.62%, RK = 1.70%, VT = 4.19%, VE = 7.78%, spoed = 9.10% and harttempo = 6.34%).

Harttempo respons tydens the wedloop het aangedui dat nie een van die atlete die Comrades Marathon teen laktaat draaipunt intensiteit kon voltooi nie. Die atlete kon slegs 'n sekere persentasie van die laktaat draaipunt harttempo handhaaf gedurende die wedloop (30.3% bo 95%van die laktaat draaipunt, 58.3% bo 90% van die laktaat draaipunt 77.3% bo 85% van die laktaat draaipunt).

Die antropometriese metinge het nie baie verander gedurende die oefen maande nie. Grootste veranderinge is aangetoon in die vet persentasie (2.45% afname). Al die atlete het egter 'n afname in vetvrye massa aangetoon (3.37%).

Opsommend is gevind dat die atlete nie in staat was om hulle harttempo's net onder die laktaat draaipunt te handhaaf gedurende die Comrades Marathon nie. As gevolg van verswakte hardloopekonomie, veral gedurende die laaste 20 km, kon nie een van die atlete die wedloop tussen die 2-4 mmol/L intensiteit handhaaf nie. Dit blyk asof meer gimnasium werk en interval oefening nodig is om stamina en spieruithouvermoë te verhoog wat 'n belangrike parameter is vir Comrades atlete. Laboratorium toetse kan die atleet help om sy hardloop potensiaal te optimaliseer. Parameters van belang is verbetering in laktaat intensiteit en nie noodwendig teen maksimale intensiteit nie, omdat dit die wedloop intensiteit simuleer.

LYS VAN SLEUTELWOORDE:

COMRADES MARATHON, HARTTEMPO RESPONS, LAKTAAT DRAAIPUNT, VENTILATORIESE DRAAIPUNT, VO_2 maks, HARDLOOPEKONOMIE, AEROBIESE OEFENING, UITPUTTING, RESPIRATORIESE KWOSIëNT, VENTILATORIESE, SPOED.

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CHAPTER 1

BACKGROUND AND STUDY OBJECTIVES

1.1 INTRODUCTION

Many scientific investigations have consistently identified the physiological variables that are positively related to successful endurance performance (Hawley, 1995). The extent to which these and other factors are "trainable" as opposed to genetically determined is a topic of considerable debate (Bouchard et al., 1992). Maximal oxygen uptake is the most common parameter used in exercise laboratories to estimate physical fitness. Costill et al. (1973) noted that subjects with similar VO_2 max values often performed quite differently in endurance events. To evaluate physical fitness more precisely, another parameter, reflection endurance capacity, should be determined in addition to VO_2 max (Vago et al., 1987).

Endurance running performance has repeatedly been shown to be related more to submaximal effort measurements, such as the onset of blood lactate accumulation and the anaerobic threshold than to VO_2 max (Maffulli et al., 1994). Long-distance runners have somewhat lower maximal oxygen uptake values than middle-distance runners, but they run at exceptionally high percentages of VO_2 max before the onset of blood lactate accumulation occurs (Costill, 1972; Costill et al., 1973; Louanne et al., 1989;).

Judging by the frequency with which the topic is discussed amongst athletes and coaches, it would still appear that the vast majority of runners, cyclists and triathletes implicitly believe that the VO_2 max is the single best predictor of athletic potential in all endurance events (Noakes, 1988). Endurance running performance is strongly influenced by VO_2 max, running economy, fractional utilisation of VO_2 max, blood lactate accumulation during submaximal exercise, and ventilatory and lactate thresholds (Maffulli et al., 1994).

Although VO_2 max is a satisfactory predictor of endurance performance in a heterogeneous group of athletes (Costill et al., 1973; Farrel et al., 1979), individuals with similar VO_2 max

values can differ markedly in performance velocity (Costill et al., 1973; Daniels, 1985; Coyle et al., 1988; Noakes, 1988).

Recently, both sports physiologists (Hawley & Noakes, 1992) and coaches have recognised the importance of peak sustained power output as a predictor of endurance performance (Hawley, 1995). In runners, for example, the peak treadmill velocity that an athlete can achieve during a maximal test has been found to be as good a predictor of endurance performance as any physiological variable currently measured (Morgan et al., 1989; Noakes et al., 1990)

Distance runners who are the fastest over the shorter distances will also be the fastest over longer distances (Hawley, 1995). Time to exhaustion at the velocity associated with VO_2 max, together with the anaerobic threshold, should provide information about the anaerobic capacity of an individual (Hill & Rowell, 1996). The more economical a particular runner was, the faster his pace could have been before he accumulated blood lactate (Louanne et al, 1989). Fohrenbach et al. (1987) determined that high correlation's existed between marathon velocity and running velocity at lactate accumulation levels of 2,5, 3,0 and 4,0 mmol/L, with elite marathoners racing at a pace that elicited about 3,0 mmol/L (Louanne et al., 1989).

Lactate threshold merely reflects the highest exercise intensity that an athlete can sustain for an extended period without amounts of lactate accumulating that are limiting for performance (Hawley, 1995). The strong relationship between endurance performance and lactate kinetics led to the suggestion that blood lactate concentration could be used as a training tool (Keith et al., 1992).

There is a close relationship between lactate threshold and endurance performance (Coyle et al., 1988). Running endurance training increases the speed at the lactate turning point, and these changes correlate closely with the actual improvements in running performance (Hawley, 1995).

In theory, exercise at power outputs above individuals anaerobic threshold will result in a progressive metabolic acidosis (McLellan & Jacobs, 1989). Endurance training has been

shown to increase the rate of oxidation or clearance of blood lactate during exercise (Mc Lellan & Jacobs, 1989).

The intensity and duration of exercise will determine the production and accumulation of lactate. By altering the intensity of exercise and the work:rest ratio, the contribution of anaerobic and aerobic metabolism to total energy needs may be manipulated (Burke et al., 1994). The question which is the most appropriate training intensity for exercise prescription has not been resolved. Some investigators suggest that lactate threshold is a critical training intensity; others support the use of fixed blood lactate concentrations of 2,0, 2,5 and 4,0 mmol/L (Hetzler et al., 1991).

Sjodin et al. (1982) reported that middle-distance and long-distance runners who were able to train closest to the running speed that elicited a lactate concentration of 4 mmol/L demonstrated a greater training response after 14 weeks of endurance training than those runners who had higher or lower lactate concentrations during training (Keith et al., 1992). These results suggest that there may be an optimal lactate concentration to stimulate aerobic training effects during steady-state training (Keith et al., 1992). In contrast, they were able to calculate an individual anaerobic threshold that was the intensity that could be maintained for a relatively long duration with stable lactate concentrations, rather than being a fixed concentration (Keith et al., 1992). This value varied significantly among subjects.

The Comrades is a race steeped in history, controversy and tradition. The American edition of "Runners World" rated the Comrades as one of the top ten races in the world, along with events of such status as the New York, London, and Boston Marathons. South Africa is the only country in the world where ultra-running is a national sport.
Internationally, the only comparable situation is in Japan, where the popularity of ultras is also growing. Many people have discovered the challenge of the Comrades. Success in ultra-running lies in doing as much volume as one's physiology, biomechanics, immune system and mind can deal with. Although running is the world's largest participation sport, most runners have to train alone. Without the benefit of a coach, they have no one to make sure they're using the most effective training methods, no one to show them how to achieve their

maximum running potential. Sport Science can play a vital role in the success of ultra-marathons by helping the athletes to achieve their optimal fitness levels. Science has given us a unique insight into the anatomy of one of the most difficult races in the world.

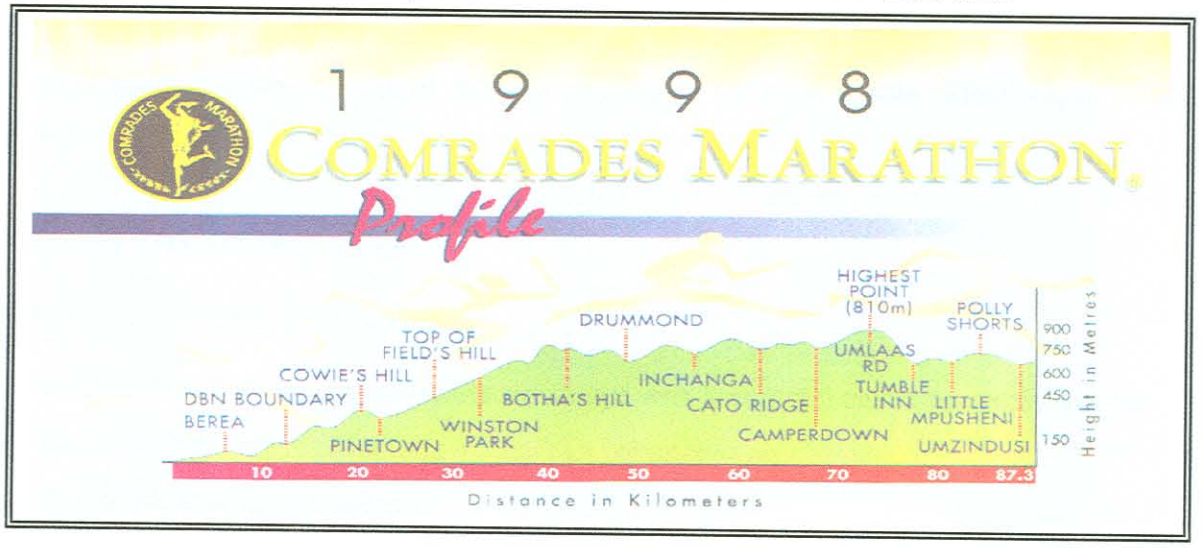


Figure 1: The profile of the 1998 Comrades race (up run)

1.2 FORMULATION OF THE PROBLEM

The purpose of the study was to assess experimentally the physiological status of a Comrades Marathon athlete and to examine the effect of training on the physiological parameters.

The testing programme indicates the athlete's strengths and weaknesses in relation to marathon running and provides baseline data for an individual training programme prescription. However, marathon running involves several physiological components. Although in the field setting it may be relatively easy to evaluate the sum results, it usually is difficult to assess the athlete on each of the components. In the laboratory, the biokineticist or sport scientist is often able to isolate a given component and to assess objectively the athlete's performance on that variable. The results of the assessment then become the basis for prescribing an optimal training programme that concentrates on identified areas of weakness. A testing programme provides feedback. Comparing the athlete's results on a given test item with those of his previous tests provides a basis for assessing the effectiveness of the intervening programme.

Laboratory testing does have severe limitations for identifying potential talent. The complete performance of any athlete is a composite of many different factors of which physiological function is only one. Therefore performance cannot be predict by a single physiological test or battery of physiological tests, especially in sports in which technical, tactical, and psychological components might relegate physiology to a lesser role (MacDougall et al., 1991).

1.3 STUDY OBJECTIVES

The major objectives of the study were:

- to identify the physiological factors associated with successful endurance performance;
- to measure the effect of training on the VO_2 parameters on a regular basis;
- to provide training guidelines;
- to use heartrate monitors to guide training and optimise race performance; and
- to measure heartrate response during the Comrades Marathon race, thus to determine race intensity.

A comprehensive exposition of a literature review appears in Chapter 2. The various procedures that have been used are shown in Chapter 3. Chapter 4 contains the results and discussion and a conclusion and recommendations follow in Chapter 5.

CHAPTER 2

LITERATURE REVIEW

2.1 INTRODUCTION

Today's top coaches recognise that the most effective methods of preparing their athletes for competition are those based on proven scientific principles rather than on trial and error. It has become commonplace for sport to seek the input of sport scientists so that they can reach their full potential.

Many physiological factors are related to successful endurance performance. **Maximal oxygen uptake** is generally considered to be a useful indicator of successful performance in endurance activities when the subjects are heterogeneous in terms of VO_2 max (Costill et al., 1973; Farrel et al., 1979; Daniels, 1985; Vago et al., 1987; Noakes, 1988; Schneider & Pollack, 1991; Hawley, 1995).

It would appear, however, that the **fraction of VO_2 max** that an athlete can sustain for prolonged periods is an even better indicator than VO_2 max alone (Costill et al 1973; Sjodin et al., 1982; Hawley, 1995). Recently, both sports physiologists (Noakes, 1988; Hawley, 1995) and coaches have recognised the importance of **peak sustained power output** as a predictor of endurance performance.

Other variables include **fatigue resistance** (Noakes, 1988; Hawley, 1995; Holtzhausen et al., 1994), **anaerobic threshold** (Vago et al., 1987; McLellan & Jacobs, 1989; Louanne et al., 1989; Keith et al., 1992; Hirokoba et al., 1992; Urhausen et al., 1993; Burke et al., 1994; Hawley, 1995), **economy of motion** (Hawley, 1995; Brisswalter et al., 1996; McArdle et al., 1996) and **fuel utilisation** (Vago et al., 1987; Hawley, 1995).

2.2 MAXIMAL OXYGEN UPTAKE

2.2.1 Definition of VO_2 max

Maximal oxygen consumption (VO_2 max) can be defined as the maximal rate at which oxygen can be consumed per minute during large-muscle-group activity of progressively increasing

intensity that is continued until exhaustion (MacDougall et al., 1991). The region where oxygen uptake plateaus and shows no further increase, or increases only slightly with an additional workload, is called the maximal oxygen uptake, maximal oxygen consumption, maximal aerobic power, or simply VO_2max (McArdle et al., 1996). VO_2 max is usually reported as an absolute volume per minute (L/min) for sports such as rowing, in which total work output is important, and as a volume per minute relative to body weight (ml/kg/min) in activities such as running, in which the body weight is supported during the performance (McDougal et al., 1991).

The measurement of oxygen consumption during exercise is the most valid means of determining a person's maximal aerobic power. It is generally accepted as the best measure of the functional ability of the cardiorespiratory system – thus, of cardiorespiratory fitness (Foss & Keteyian, 1998).

Athletes who excel in endurance sports generally have a large capacity for aerobic energy transfer (McArdle et al., 1996). The maximal oxygen uptakes recorded for competitors in distance running is almost double those of sedentary men and women (McArdle et al., 1996). This is not to say that the VO_2 max is the only determinant of endurance performance. Other factors, principally those at the local tissue level such as capillary density, enzymes, mitochondrial size and number, and muscle fibre type, exert a strong influence on a muscle's capacity to sustain a high level of aerobic exercise (McArdle et al., 1996).

The VO_2 max does, however, provide important information on the capacity of the long-term energy system. In addition, this measure has significant physiologic meaning in that attaining a high VO_2 max requires the integration of a high level of ventilatory, cardiovascular, and neuromuscular functions (McArdle et al., 1996). This makes VO_2 max a fundamental measure in exercise physiology. Martin & Coe (1997) find a high statistical correlation between aerobic power and competitive performance.

While VO_2 max in elite male middle-distance and long-distance runners typically ranges from 75-85 ml/kg/min, such high values are probably not as critical for athletes participating in prolonged endurance events which last 60 min and longer (Hawley, 1995). Top marathon runners (i.e. sub 2 h 20 min) can sustain 86% of VO_2 max for the duration of a race (Hawley

1995), whereas slower runners (i.e. 2 h 45 min up to 3 h) can sustain only 75% of their VO_2 max for the same distance (Farrel et al 1979; Hawley, 1995). High VO_2 max values (between 83–85 ml/kg/min) have been measured in men and (73–77 ml/kg/min) in women (Noakes et al., 1990). VO_2 max values measured in healthy young athletes with mean average ability are much lower, usually between 45–55 ml/kg/min, i.e. about 60% lower than in elite athletes.

Research has shown that considerable variance may exist in race performance times within a group of highly trained runners with similar VO_2 max values (Schneider & Pollack., 1991). Powers et al. (1983) and his colleagues demonstrated that the oxygen uptake measured at the ventilatory threshold was a better predictor of distance running success than either VO_2 max or running economy (Louanne et al., 1989; Schneider & Pollack., 1991). The failure of VO_2 max to accurately and consistently predict the racing times of all athletes has led to the belief that other factors must play a role. In fact, some recent studies question whether the "plateau phenomenon" exists at all (Noakes et al., 1990).

2.2.2 Direct determination of maximal oxygen uptake

Based on research, the measure of VO_2 max became the "benchmark" to quantify cardiovascular functional capacity and aerobic "fitness". Maximal oxygen uptake can be determined using a variety of exercises that activate large muscle groups as long as the intensity and duration of the effort are sufficient to engage maximal aerobic energy transfer (McArdle et al., 1996). Test protocols tend to vary widely, having been developed to suit available equipment, subjects studied, and investigator preferences.

Concerns and practical considerations that must be addressed when measuring VO_2 max include:

- exercise modality;
- protocol selection;
- laboratory environment;
- subject preparation and motivation; and
- accuracy of the measure.

2.2.2.1 Exercise modality

The elite level, highly fit athletes prefer a single treadmill test that essentially mimics a competition: a continuous test that will measure all necessary fitness-related variables with no stopping along the way for such things as intermittent blood sampling (Martin & Coe, 1997). Evaluation of untrained people who have had very little experience in either running or bicycling shows them to have higher VO_2 max value when a treadmill is used than when a bicycle ergometer is used. Treadmill testing can lead to higher values by as much as 4% to 23% (Martin & Coe, 1997). Because a larger total muscle mass is active, venous blood return to the heart is greater.

2.2.2.2 Protocol selection

McConnel (1988) investigated the difference in treadmill running grade. They demonstrated that when using a motorised treadmill, running on a grade elicit greater and more reliable VO_2 max values when compared with that obtained with flat treadmill running and speed increments. Zhang et al. (1991) compared different treadmill protocols and noted that VO_2 max values for all protocols correlated highly. However, Froelicher et al. (1974) found that there was a statistical difference in VO_2 max between the Bruce (3min) and Balke (1min) protocols. According to Zhang et al. (1991), the parameters of aerobic function and the physiologic responses to progressive exercise tests are independent of the work rate protocol if the overall work rate increases at a constant level.

It is important, however, to measure the improvements in VO_2 max using a test protocol that challenges the body in the manner most similar to the mode of training (Martin & Coe, 1997). VO_2 max usually consists of progressive increments in effort to the point at which the subject will no longer continue to exercise. McConnel (1988) findings suggest that a continuous protocol with workstage durations of 1 minute or less may be most efficient, in terms of testing time for obtaining VO_2 max in runners and may be perceived as being less difficult by the runner.

A number of studies have examined the problem of day-to-day variability in oxygen consumption during submaximal running. Variability across subjects ranges from 0.30 to 4.40% in Morgan et al. (1991) and 1.20 to 5.80% in Williams et al. (1991). Pereira et al.

(1991) demonstrated that intraindividual variation in VO_2 during steady-rate graded treadmill running is small. In the light of the biological variation in submaximal exercise, repeated tests should be employed under the same conditions when submaximal exercise is used to measure training effects or treatments. This may be especially important if subjects are no already accustomed to treadmill running and/or the laboratory equipment, in which case a habituation period for accommodation to the treadmill and protocols is recommended.

Although knowledge of VO_2 parameters may be useful for exercise prescription, laboratory determination is limited by the fact that lactate measurement is invasive and ventilatory technique required both high subject motivation and a sophisticated data acquisition system. Such measurements are therefore not practical for coaches and do not lend themselves to testing large groups. An alternative prescription is the use of field-tests i.e. the percentages of maximal parameters obtained during the course of aerobic tests (Ahmaidi et al., 1992). Coaches prefer the use of maximal velocity, as it is an objective measure easily applied to numerous sports disciplines.

2.2.2.3 Laboratory environment

It is essential for laboratories to simulate as closely as is practically possible the training and competitive environments of the athletes during the test. In today's era of excellent technology, most laboratories take great care to perform proper calibrations, maintain constant room temperature and humidity for repeat test procedures, and ensure consistency in technician competence. Even so, VO_2 max values obtained on different days from the same athlete will not be identical.

2.2.2.4 Subject preparation and motivation

A period of familiarisation with testing procedures is important for allowing the runner to become accustomed to the equipment understanding the objectives of the test, reducing anxiety and allowing consistency of efficiency between tests. The test should be preceded by a warm-up at a relative intensity of at least 50% of VO_2 max (Mc Connel et al. 1988).

2.2.2.5 Accuracy of the measurement

Extrinsic factors should be controlled as beast as possible. Intraindividual variability in VO_2 max has been investigated for 50 years (Pereira et al., 1991)

Investigators must address the following considerations to help minimise the variability of VO_2 max (Mc Connel, 1988):

- since the runner subjectively determines the test termination points, the reasons for test termination must be consistent for all tests;
- it is advisable to perform tests under consistent conditions with regards to time of day, ambient conditions, and length of time since previous exercise; and
- calibration gasses must be accurate.

Martin & Coe (1997) suggested that the coefficient of variation of VO_2 max could be held within ± 3%, and the most important variables to be measured are:

- O_2 consumption at several submaximal training paces (running economy);
- the threshold at which steady-state work can no longer be maintained (lactate and ventilatory thresholds); and
- the absolute limits of aerobic performance (VO_2 max) and anaerobic performance.

However, modern studies suggest that this 'plateau phenomenon' – which occurs in less than 50 percent of tested subjects – does not mean that a true oxygen deficiency has developed in the muscles. They clearly become exhausted and stop exercising, but the absence of the plateau indicates that they had plenty of oxygen going to the muscles at that time. Noakes (1992) stated that the rate of oxygen transport is not the critical factor determining exercise performance. Rather he suggests that the best athletes have muscles with superior contractility. To make this point clearer, it could be said that one's muscle strength allows us to run at high speeds; once one reaches those high speeds, one needs a high rate of oxygen consumption, i.e. a high VO_2 max. But the high VO_2 max does not create the ability to run fast – rather, the high VO_2 max is a result of the ability to run fast (Noakes, 1992).

To be sure that a person has reached the maximum capacity for aerobic metabolism during exercise, a levelling-off or peaking-over in oxygen uptake should be demonstrated. Often the highest (Peak VO_2) oxygen uptake is recorded in the last minute of exercise. Peak VO_2 refers to the highest value of oxygen uptake measured during the test.

2.2.3 Specificity of VO_2 max

The specificity principle implies that for example fitness for swimming, bicycling, running or rowing is most effectively improved by training the specific muscles involved in the desired performance. Based on available research, it is reasonable to advise that during training for specific aerobic activities, the overload must both engage the appropriate muscles required by the activity and induce an exercise stress on the central cardiovascular system (Franklin, 1989). Little improvement is observed when aerobic capacity is measured during a dissimilar exercise, yet improvements are significant when the test exercise is the same exercise used in training (McArdle et al., 1996). It should be noted, however, that changing exercise mode rarely changes oxygen uptake more than 15% (Bergh et al., 2000). Hence there is an upper limit for the attainable oxygen uptake. Therefore, we need to specify the exercise mode for the sake of comparison, especially when using maximal oxygen uptake to characterise athletes.

2.2.4 Plateau in VO_2

Not all subjects showed a plateau in VO_2 at the end of a graded exercise test, when graphed against work intensity. It has repeatedly been shown that about 50% of subjects do not demonstrate a plateau when stressed to maximal effort (Howley et al., 1995). Failure to achieve a plateau does not mean that these subjects have failed to attain their "true" VO_2 max. With a continuous graded exercise test protocol a subject may fatigue just as VO_2 max is reached. Even with a discontinuous graded exercise test most researchers require that a subject complete 3 – 5 min at each stage. Thus if a subject reaches VO_2 max in 2 min at a supramaximal intensity and then becomes too fatigued to continue, this data point would not be graphed. For these reasons, the plateau in VO_2 cannot be used as the sole criterion for achievement of VO_2 max (David et al., 2000).

2.2.5 Limitations of VO_2 max

It has become clear that the VO_2 max, as an indicator of endurance performance is liable to some restrictions. These restrictions are based on the observation that several individuals, especially endurance trained athletes may still increase their performance, although VO_2 max does not further increase despite continued training. In addition, athletes with comparable performance can have considerable differences in VO_2 max (Noakes, 1988; Morgan et al.,

1990; Kuipers & Arts., 1994). Another restriction of the use of VO_2 max as an indicator of endurance performance is that the VO_2 max shows day-to day variation.

Wagner (2000) named four new ideas on limitation to VO_2 max after Noakes, in his 1996 Wolfe Lecture, considered VO_2 max, defined as reaching a plateau of VO_2 at high power outputs a myth.

- The existence of a plateau in VO_2 at some maximal value can indeed be noted at very high power outputs in human and animal subjects with high pain and fatigue tolerance. From basic laws of chemical mass action, this phenomenon must also potentially exist, but may not be observable, in subjects whose pain or fatigue tolerance is low.
- VO_2 may be set by metabolic limits. As seen in unfit humans and nonathletic animals with low peak VO_2 values, the VO_2 does not improve by providing extra O_2 and reducing $F10_2$ moderately does not diminish VO_2 max.
- VO_2 max may be alternatively be sit by O_2 transport limits. This seems to be the case for fit subjects and athletic animal species, because providing extra O_2 does improve VO_2. Under such conditions all elements of the O_2 transport pathway act to limit VO_2 max in an integrated way, such that changes in any one component have the potential to change VO_2 max, but in a manner that depends on the values of all the other components.
- When VO_2 max is determined by O_2 availability, the quantitative importance of each O_2 transport pathway component can be estimated, and will vary with the experimental conditions. Cardiac function, is by no means the only factor determining VO_2 max. Other factors such as muscle O_2 conductance are equally important in fit young subjects. Such conclusions seem to differ at higher altitudes, where convective O_2 transport, reflecting cardiac function, is no longer as important. Rather, diffusive processes in the lungs and muscle become more influential because of the reduction in P_{O2} gradients for diffusion in these locations.

However Berg et al. (1998) questioned some of the aspects from Noakes (1998). Bergh et al. (1998) briefly comment on some of the more fundamental points to demonstrate the consequence of his reasoning.

- His line of reasoning seems to be based on the assumption that the cardiovascular model postulates that maximal oxygen uptake is the sole determinant of endurance performance.

However fatigue should be explain under all conditions. Thus, it is not fair to claim that this is an established model. So the logic of bringing it up seems to be if a factor fails to be the only determinant, it is without importance. In fact, it might even be the most important one.

- Noakes report that about 50% of all studies, mainly those using incremental procedure, fail to demonstrate a plateau. But there is no discussion about the influence of incremental procedures on the probability to find a plateau. Thus it is absolutely necessary to use the same standards both qualitatively as well as quantitatively. The effect different test protocols on the plateau were also studied by David et al. (2000).
- Noakes ignores many studies supporting the belief that central circulation limits the peak oxygen uptake during exercise engaging a large muscle mass.
- Noakes has excluded vital sources of information and included data

David et al. (2000) conclude that each and every step in the O_2 pathway contributes in an integrated way to determining VO_2 max, and a reduction the transport capacity of any of the steps will predictably reduce VO_2 max. For instance, a reduction in the inspired PO_2 at altitude will result in a decreased VO_2 max. A reduced hemoglobin level in anemia will result in a decreased VO_2 max. Also a reduction in cardiac output with cardioselective beta-blockade will result in a decreased VO_2 max.

2.3 OXYGEN CONSUMPTION PARAMETERS

In the scientific literature, and increase in VO_2 max is the most common method of demonstrating a training effect. In addition, VO_2 max is frequently used in the development of an exercise prescription. Given these applications of VO_2 max, there has been great interest in identifying the physiological factors that limit VO_2 max and determining the role of this variable in endurance performance (David et al., 2000).

2.3.1 Cardiac output

Given the level of technology in 1923, it was the great scientist Hill who deduced that endurance athletes have heart with superior pumping capacities (David et al., 2000). Trained individuals have a much higher maximal cardiac output than untrained individuals - 40 vs 25 L/min. Maximal cardiac output is limited by the maximal rate of depolarisation of the sino-

artrial (SA) node and the structural limits of the ventricle. It is estimated that 70 – 85% of the limitation in VO_2 max is linked to maximal cardiac output (David et al., 2000).

Tesch (1985) has written an authoritative review of 24 studies detailing the cardiovascular responses to beta blockade. According to Tesch (1985) the beta blockade can decrease maximal heart rate by 25 – 30%. In these studies cardiac output decreases by 15 – 20%, while stroke volume increases slightly. As a result the VO_2 max declines by 5 – 15%. Tesch (1985) conclude that the decline in VO_2 max seen with cardio-selective beta-blockade is caused by diminished blood flow and oxygen delivery.

2.3.2 Oxygen deficit

During the beginning of exercise, the oxygen uptake is considerably below the steady-rate level even though the energy requirement presumably remains unchanged. The oxygen deficit can be viewed quantitatively as the difference between the total oxygen actually consumed during exercise and the total that would have been consumed had a steady rate of aerobic metabolism been reached at the start (McArdle et al., 1996).

Maximal accumulated oxygen deficit has been proposed as a valid and reliable measurement of anaerobic capacity (Buck & McNaughton, 1999). Maximal oxygen deficit is determined as the difference between the calculated oxygen demand and the actual oxygen uptake. Once the steady rate is attained, oxygen uptake during light and moderate exercise is similar in trained and untrained persons. For the endurance trained person, however, the steady rate is reached more rapidly and with a smaller oxygen deficit compared with someone who is untrained (Hagberg, 1980). Consequently, the total oxygen consumed during exercise is greater for the trained person, and presumably, the anaerobic component of energy transfer is proportionately smaller (McArdle et al., 1996).

Rieu et al. (1990) noted that time-dependent changes in lactate during submaximal exercise consist of a first phase corresponding to the oxygen deficit, with considerable accumulation of lactate, and a second phase during which VO_2 is stable, with minor changes in lactate. These two phases are modified by previous supramaximal exercise inducing hyperlaticaemia. Lactate decreases during the VO_2 transient phase. The explanation of this phenomenon is probably largely linked to the reduction of oxygen deficit, i.e. to the decreased energy contribution of

anaerobic glycolysis (Rieu et al., 1990). Wasserman et al. (1986) describes the following two phases: at the onset of moderate work, oxygen uptake from the lungs normally increases abruptly (50%-100% resting VO_2) and remains relatively unchanged for the first 15 seconds (Phase I). Then the VO_2 increases as a single exponential with a time constant of approximately 30 seconds (Phase II). The difference between the total oxygen uptake and the product of the steady-state oxygen uptake and the exercise duration is referred to as the oxygen deficit. Weyand et al. (1994) concluded that peak oxygen deficit is the best measure of anaerobic capacity available, but relatively little research has been conducted using this measure.

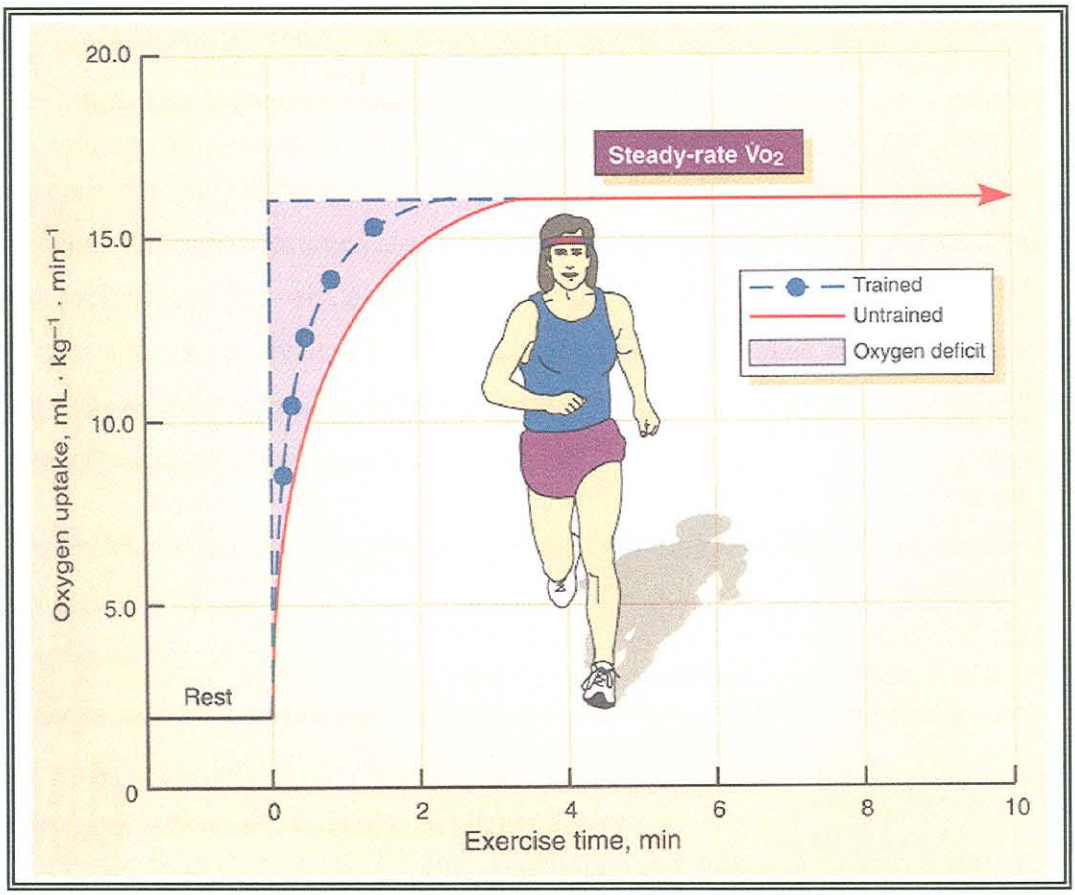


Figure 2: Time course of oxygen uptake during a continuous jog at a relatively slow pace for endurance-trained and untrained individuals who exercise at the same steady-rate VO_2. The shaded area indicates the oxygen deficit or the quantity of oxygen that would have been consumed had the oxygen uptake reached a steady rate immediately (McArdle et al., 1996)

2.3.3 Oxygen dept (excess postexercise oxygen consumption - EPOC)

After exercise, bodily processes do not immediately return to resting levels. If the activity is particularly stressful or the duration is extended during high-intensity aerobic exercise, considerable time may be required for metabolism to return to the resting level. Regardless of the intensity of the exercise, oxygen uptake during recovery always exceeds the resting value. This excess has commonly been termed the oxygen debt.

The oxygen debt or, more accurately, the recovery oxygen uptake or excess postexercise oxygen consumption (EPOC), reflects both the anaerobic metabolism of previous exercise and the respiratory, circulatory, hormonal, ionic, and thermal adjustments that occur during recovery (McArdle et al., 1996). Once the steady-state is reached, the oxygen debt no longer increases, regardless the exercise duration (Wasserman et al., 1986).

Oxygen consumption following exhaustive exercises decreases exponentially with time; the rate at which oxygen is consumed is not constant throughout the recovery period. During the first 2 or 3 minutes of recovery, oxygen consumption declines very rapidly, then more slowly until a constant rate, equivalent to resting levels, is reached. The initial rapid portion of recovery is now identified as the fast component, whereas the slower phase is now referred to as the slow component (Kochan et al., 1979).

Causes of excess postexercise oxygen consumption resulting from heavy exercise are:

- resynthesise ATP and CP;
- resynthesise lactate to glycogen;
- oxidise lactate in energy metabolism;
- restore oxygen to blood;
- thermogenic effects of elevated core temperature;
- thermogenic effects of hormones, particularly the catecholamines adrenaline and noradrenaline; and
- effects of elevated heart rate, ventilation, and other elevated levels of physiologic function.

Gore & Withers (1990) reported that exercise intensity is approximately five times more important than duration in determining the magnitude of EPOC. Most studies that have

reported a prolonged EPOC (60 minutes or greater) have used exercise intensities of 70% of VO_2 max (Bahr et al., 1987; Bahr & Sejersted., 1991). Sedlock et al. (1989) held exercise energy cost constant while varying intensity and duration, also found higher (73% of VO_2 max) rather than lower (50% of VO_2 max) intensity exercise to produce a greater magnitude of EPOC. However, the study of Maresh et al. (1992) did not support an exercise-duration-mediated increase in EPOC. They found no appreciable differences in recovery oxygen uptake following exercise sessions performed at either 60% or 70% VO_2 max for 20 min or 40 min, and no relationship between recovery oxygen uptake and exercise duration.

In contrast Gore & Withers (1990) and Chad & Quigley (1991) found at higher exercise intensities (80% of VO_2 max or more), both intensity and duration of exercise, and also factors such as lactate metabolism, increased tissue temperature and training status can affect the magnitude of the slow recovery phase. If the work intensity is very heavy for a normal subject, or if the subject is so impaired that his cardiorespiratory system cannot supply the total oxygen need, a steady-state is not achieved and lactate continues to increase until the subject is forced to stop exercise because of fatigue or breathlessness (Wasserman et al., 1986). Bahr et al. (1987) reported a significantly elevated recovery oxygen uptake, above control values, during the first 2h following cycle-ergometer exercise performed at 70% VO_2 max over similar lengths of time. They concluded that EPOC increase linearly with exercise duration. As long as the oxygen uptake fails to reach a steady state for constant work rate exercise, the oxygen deficit and debt continue to enlarge.

EPOC is often cited as playing an important role in significantly increasing energy expenditure in weight loss programmes that employ exercise. Exercise duration and intensity have been identified as the principal factors influencing EPOC. Brehm & Curtin (1986) reported that exercise intensity is curvilinearly related to EPOC with a disproportionate increase in the magnitude of EPOC associated with exercise intensities greater than 75 percent of VO_2 max. Kaminsky et al. (1990) conclude that split exercise sessions, respectively can significantly increase post-exercise caloric expenditure. However, the overall magnitude of the increase is small.

2.3.4 Ventilatory equivalent (V_E/VO_2)

The ratio of minute ventilation to oxygen uptake is termed the ventilatory equivalent and is symbolised V_E/VO_2. In healthy young adults, this ratio is usually maintained at approximately 25 L during submaximal exercise up to approximately 55% of the oxygen uptake (McArdle et al., 1996). In non steady-state exercise, ventilation increases disproportionately with increases in oxygen uptake, and the ventilatory equivalent may reach 35–40 L. The V_E/VO_2 max ratio increases without an accompanying increase in V_E/VCO_2. An increase in muscle mitochondria may allow a slightly greater extraction of O_2 from the blood by the working muscles, thus contributing in a minor way to an increased VO_2 max (David et al., 2000). Dempsey (1986) stated that the ability of the skeletal muscle to adapt to training is far greater than what is observed in the lung. Thus the main significance of the training induced increase in capillary density is not to accommodate blood flow but rather to maintain mean transit. This enhances oxygen delivery by maintaining oxygen extraction even at high rates of muscle blood flow.

2.3.5 Respiratory quotient (RQ) and Respiratory exchange ratio (R)

The ratio of metabolic gas exchange in the combustion of food is termed the respiratory quotient, and is defined as follows:

$RQ = CO_2$ produced / O_2 consumed

The application of the RQ is based on the assumption that the exchange of oxygen and carbon dioxide measured at the lungs reflects the actual gas exchange from nutrient catabolism in the cell (McArdle et al., 1996). This assumption is only valid during steady state or resting conditions. However when other factors such as high intensity exercise or hyperventilation affect the RQ so that it no longer reflects only the substrate mixture in energy metabolism, then it is termed as Respiratory exchange ratio (R).

During exhaustive exercise, R can rise significantly above 1,00. The lactic acid generated during anaerobic metabolism is buffered by sodium bicarbonate in the blood to maintain the acid-base balance. Because of the buffering effect, the CO_2 values rise very high, above the quantity normally released during energy metabolism. Carbon dioxide elimination increases during hyperventilation, and as a result of that, the normal level of carbon dioxide in the blood

is reduced. This elimination is not accompanied by a rise in the oxygen uptake; thus, the rise in the RQ does not represent the oxidation of food.

Hirokoba et al. (1992) found that endurance trained men generate more CO_2 excess at the same blood lactate concentration as compared with non-endurance trained and untrained men. There are two possible explanations for this:

- the increase in CO_2 excess per unit of body mass per lactate accumulation may be due to the decrease of buffering in the non-bicarbonate system; or
- the increase of buffering in the bicarbonate system.

McArthur et al. (1983) found higher muscle glycogen levels after, and higher RQ values during marathon races in the better runners.

2.3.6 Minute ventilation (V_E)

During quiet breathing at rest, the normal breathing rate is approximately 12 breaths per minute and the average tidal volume is approximately 0,5 L of air per breath. The volume of air breathed each minute is thus 6 L. During strenuous exercise, the breathing rate increase between 35–45 breaths per minute, although rates as high as 60–70 have been measured in elite athletes. In male endurance athletes, minute ventilation can increase to 160 L/min. Ventilation volumes of 200 L have been reported in research studies (McArdle et al., 1996). Even with such large V_E, tidal volumes for both trained and untrained individuals rarely exceed 60% of vital capacity. Endurance trained athletes demand a lower V_E than do untrained athletes (Bailey & Pate, 1991).

2.3.7 Breathing dynamics

The expired ventilation is the product of breathing rate and tidal volume. Excessively deep breaths, few in number, would be too energy costly. Very many breaths, each small in volume would not provide effective alveolar gas exchange. Breathing can be optimised with tidal volume never more than 60% to 65 % of the vital capacity, defined as the maximum amount of air that can be exhaled after a maximal inspiration (Martin & Coe, 1997). Breathing rate values recorded in highly trained athletes were no greater than 55 per minute (Martin & Coe, 1997). Wasserman et al. 1986 report in very fit individuals V_E values of 15 liter per minute or 20 to 40% of the maximal voluntary ventilation. A low breathing reserve is characteristic of

patients with lung disease who are ventilatory limited. The breathing reserve is high in patients with cardiovascular diseases that limit exercise performance. Quite often, runners synchronise their breathing rate to their stride frequency. One practical implication of this breathing pattern is the usefulness of shortening stride and quickening cadence when climbing hills. The resulting increased breathing rate with increased stride frequency helps increase O_2 intake.

During long duration exercise at relatively low work intensities, such as between 50% to 60% of VO_2 max for about 2 h, a gradual but measurable rise in breathing rate (15% to 40%) does occur. This is accompanied by a reduction in tidal volume of about 10% to 15%. The decrease in tidal volume does not exactly compensate for the increased frequency, because V_E increases as well. This drift is not observed during the short-duration runs (Martin & Coe, 1997).

CO_2 is a powerful ventilatory stimulant, and a small rise in the P_a CO_2 probable increases the V_E by 10% to 30%. The level of V_E, with its removal of CO_2, thereby serves as the major determinant of arterial H^+ ion concentration during this submaximal long-term work (i.e. at workloads ranging from a long training run to marathon or ultradistance racing). These changes in volume and rate dynamics are controlled automatically to optimise mechanical efficiency while maintaining normal blood O_2 and CO_2 concentrations. Thus, it is unwise for coaches or athletes to attempt voluntary regulation of breathing patterns (Martin & Coe, 1997).

Trained endurance runners tend to exhibit a reduced ventilatory response to very intense exercise. One could suggest that, because dyspnea is a limiting symptom for exercise tolerance, removing it might permit greater exercise tolerance. Particularly in view of the reservoir of O_2 bound to haemoglobin, it might be possible for trained runners to optimise for slightly reduced ventilation at the expense of greater arterial haemoglobin desaturation, thereby permitting increased high-level work tolerance. Indeed, such arterial haemoglobin desaturation does occur, as described in the literature (Dempsey & Henderson, 1984) and seen in our own experience with trained runners.

Powers et al. (1989) had highly trained subjects and normal subjects perform two VO_2 max tests. In one test the subjects breathed room air and in the other they breathed a 26% O_2 gas mixture. On hyperoxic gas, the highly trained group had an increase from 70.1 to 74.7 ml/kg/min as well as an increase in arterial O_2 saturation from 90.6% to 95.9% during maximal work. None of these changes were observed in normal subjects.

2.3.8 Energy cost of breathing

At rest and during light exercise, the oxygen requirement of breathing is small, approximately 4% of the total energy expenditure. Exercise ventilation has been shown to constitute 7% to 8% of the total oxygen cost of exercise (Bailey & Pate., 1991). As the breathing rate and tidal volume increase, the energy cost rises to between 2,1 ml and 4,5 ml of oxygen per litre of ventilation (Coast, 1993). The energy cost of breathing accounts for approximately 19% of the oxygen deficit and 11% of the recovery oxygen uptake (McArdle et al., 1996). The reason is that ventilation increases at a greater rate than the oxygen uptake at the beginning of exercise. During exercise that elicits VO_2 max, as much as 8% – 11% of the total oxygen uptake is required for respiratory muscle work. The respiratory muscles use approximately 40% – 60% of their maximum capacity to generate pressure at this exercise level (Aaron, 1992). Lower ventilation, particularly over a prolonged effort (e.g. the Marathon), would mean, on a ratio basis, less oxygen to the respiratory muscles and more to the working skeletal muscles (Fox et al., 1993). Thus manipulation of the amount of ventilatory work necessary at a given running velocity could alter overall running economy (Bailey & Pate., 1991).

Maximal voluntary ventilation (MVV) is elevated among both trained male and trained female runners. This is determined by a 12s to 15s test of maximal airflow generation. Such increased performance is predictable, since distance running requires the muscles of breathing to be moderately active during long runs and highly active during fast-paced sessions. Although MVV may be an indicator of short-term endurance, it may not be a good indicator of maximum sustainable ventilation (MSV). MSV can be measured during the final moments of treadmill testing as athlete's approach their performance limits. MSV is also elevated among trained runners when compared with matched, untrained controls. MVV is typically larger than MSV by about 35% (Martin & Coe, 1997).

2.3.9 Ventilatory threshold

2.3.9.1 Definition

The point at which ventilation departs from a linear increase with workload and CO_2 production has been identified as the ventilatory threshold. Recent research from Schneider & Pollack (1991) refers to the marked rise in lactate concentration during incremental exercise as the lactate threshold and the non-linear increase in V_E as the ventilatory threshold. Hill & Rowell (1996) defined ventilatory threshold as the treadmill velocity and corresponding VO_2 at which there was a breakaway in V_E, an increase in V_E/VO_2 and an initial increase in the percentage of O_2 in the expired air, plotted against time. Martin & Coe (1997) refers to ventilatory threshold when the VCO_2 produced exceeds the VO_2 utilised.

2.3.9.2 Correlation between ventilatory and lactate threshold

The concept of ventilatory and lactate threshold has been widely used to evaluate endurance capacity or to assess the effect of training. Strong correlation between lactate threshold and ventilatory threshold has been explained on the basis of the increased carbon dioxide produced as a result of lactate buffering by the bicarbonate system (Burke et al., 1994). However, Schneider & Pollack (1991) have shown that lactate threshold and ventilatory thresholds may not occur together and thus the measurement of ventilatory threshold may not always reflect lactate threshold. Mocellin et al. (1990) who have also demonstrated distinct differences between lactate concentrations at the anaerobic threshold and at maximal steady state blood lactate in children aged 9-15 years, with no correlation between the parameters.

Although the physiological mechanisms that cause the ventilatory threshold remain controversial, many physiologists believe that the ventilatory threshold is important to endurance performance success. In fact, endurance training has been shown to delay the onset of both the lactate and the ventilatory thresholds (Schneider & Pollack., 1991; Hoffmann et al., 1993). Thus, ventilatory threshold is a useful measurement that provides valuable information concerning the relative level of lactate threshold.

Hoffmann et al. (1993) found that changes in ventilatory threshold for the runners were dependent upon the specific mode of training, i.e. running or cycling. Runners demonstrated a significantly greater ventilatory threshold on the treadmill than the cyclists did. Hoffmann et

al. (1993) attributed this difference to specific local muscle adaptations caused by cycling and running, speculating that these adaptations reduced the rate of lactate production in the working muscle and thereby increased ventilatory threshold. These findings are supported by others who indicate that lactate threshold is highly correlated with ventilatory threshold (Walsh & Davis., 1990; Hoffmann et al., 1993).

2.3.9.3 Determination of ventilatory thresholds

The buffering of increased lactic acid mainly by HCO_3 during exercise is accompanied by the increase in VCO_2. The transition point on the curve of VCO_2 can be used as a powerful variable to detect the ventilatory threshold (Cheng et al., 1992). Carbon dioxide production was also shown to be an effective threshold indicator (Cheng et al., 1992). In the study of Cheng et al. (1992) VCO_2 was the respiratory variable, which had the greatest relationship with lactate and the other variables at the threshold point and possessed the best reproducibility among the variables. Breathing frequency can also be used to determine the threshold (James et al., 1989). Because breathing frequency can be easily monitored without the use of invasive techniques and expensive analysers, it can be determined in field tests.

2.3.10 Oxygen pulse (VO_2/HT)

The O_2 pulse is calculated by dividing the oxygen uptake by the heart rate. It is the volume of O_2 extracted by the peripheral tissues or the volume of O_2 added to the pulmonary blood per heart beat and can be shown to be equal to the product of stroke volume and the arterial-mixed venous O_2 difference. As the work rate is increased, the O_2 pulse rises, primarily because of an increasing arterialmixed venous O_2 difference. If, however, the stroke volume is reduced, the arterial-mixed venous oxygen difference and, therefore, the O_2 pulse reach maximal values at a relatively low work rate, and the O_2 pulse approaches an asymptote at a low value. The O_2 pulse will also be low with anemia, hypoxemia, all because of a reduced arterial O_2 content (Wasserman et al., 1986).

The predicted maximum oxygen pulse is the quotient of predicted maximum VO_2 and predicted maximum HR. In a given individual there is a close relationship between VO_2 and HR during exercise. The quotient of the VO_2 and HR is the oxygen pulse. The normal relationship of VO_2 to HR is linear over a wide range with a positive intercept on the HR axis.

The maximum VO_2 is five to fifteen times resting VO_2 while the maximum HR is two the three times resting HR. Occasionally just before the end of the incremental exercise test, VO_2 reaches a maximum before HR, in which case the maximum O_2 pulse may be slightly higher than the O_2 pulse at the end of exercise. The predicted O_2 pulse at any given VO_2, including maximum VO_2, is strongly dependent on the normal individuals body size, sex, age, degree of fitness, and hemoglobin concentration. However, the O_2 pulse can be considerably higher than predicted in the cardiovascularly fit person

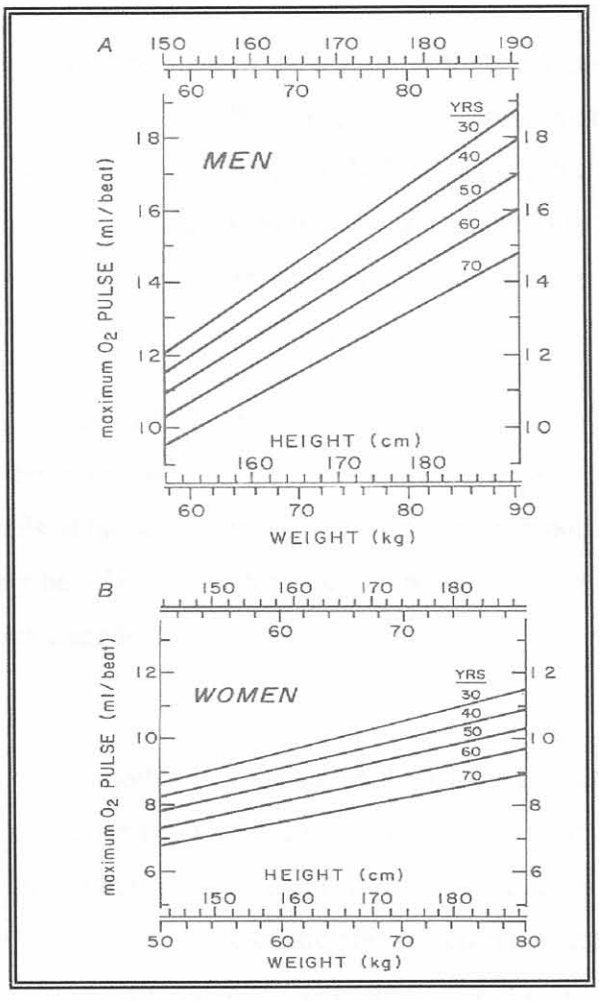


Figure 3: Mean maximum O_2 pulse for sedentary (A) men and (B) women. To use locate on the horizontal axis both the patient's weight and height. From the more leftward point draw a line vertically to the patient's age on the diagonal lines. From this point draw a horizontal line to the vertical axis to read off the maximum O_2 pulse in ml/beat (Wasserman et al., 1986).

The VO_2 max is limited primarily by the rate of oxygen delivery, not the ability of the muscles to take up oxygen from the blood. Therefore the following factors could play a role in the limiting of VO_2 max: the pulmonary diffusing capacity, maximal cardiac output, oxygen carrying capacity of the blood, and skeletal muscle characteristics (David et al., 2000).

2.4 FACTORS THAT INFLUENCE MAXIMAL OXYGEN UPTAKE

2.4.1 Mode of exercise

It is generally accepted that variations in VO_2 max during different forms of exercise reflect the quantity of muscle mass activated (McArdle et al., 1996). The highest values are generally obtained during treadmill running (McArdle et al., 1996). In the non-athletic population, VO_2 max determined on a treadmill is usually 5% - 15% higher than that achieved during cycle ergometry (Foss & Ketejian., 1998). In the laboratory, the treadmill is the apparatus of choice for determining VO_2 max in healthy subjects. Exercise intensity is easily determined and regulated.

Jones & McConnell (1999) found that a VO_2 slow component does exist for high-intensity treadmill running, and the magnitude of the slow component is less for running than for cycling at equivalent levels of lactacidaemia. The lower slow component observed in cycling compared to running may be related to differences in the muscle contraction regimen that is required for the two exercise modes.

2.4.2 Heredity

Most runners, at some time or another, have wondered how fast and how far they might run if trained to the maximum. Questions concerning the relative contribution of natural endowment (genotype) to physiologic function and exercise performance (phenotype) have been frequently raised (McArdle et al., 1996). Genetic effect is currently estimated at about 10% – 30% for VO_2 max, 50% for maximum heart rate, and 70% for physical working capacity (McArdle et al., 1996). Although a vigorous programme of physical training will enhance a person's level of fitness regardless of genetic background, it is clear that the limits for developing fitness capacity are linked to natural endowment (McArdle et al., 1996). Genetic makeup plays such a predominant role in determining the training response that it is almost impossible to predict a particular individual's response to a given training stimulus (McArdle

et al., 1996). VO_2 max is 93.4% genetically determined in males and 95.9% in both males and females together (Fox et al., 1993).

2.4.3 State of training

The effects of training on the amount of oxygen that can be consumed per minute during maximal exercise have been studied extensively; there is little doubt that it is increased with training (Frick et al., 1970). However, according to McArdle et al. (1996), maximal oxygen uptake will vary between 5% – 20% depending on whether a person is "in shape" or "out of shape" at the time of measurement. Douglas et al. (1981) reported that distance runner Jim Rhyn's maximal aerobic capacity varied from 65-81 ml O_2/kg/min depending on his state of conditioning. Bouchard et al. (1992) stated that there are high and low responders to training, and this is hereditary. This again demonstrates that VO_2 max is in fact a poor indicator of fitness, as since one's ability to run both longer and faster will increase by more than 15% with training. For example, former mile world record holder, Jim Ryan, increased his VO_2 max from 65 ml O_2/kg/min in the partially trained state, to 82 ml O_2 /kg/min in the trained state, a whopping 26% increase (Noakes et al., 1992). This once again emphasises the ability of the elite athlete to show a greater adaptation to training response. Most of the increase in VO_2 max is due to an increase in muscle contractility, which increases the capacity of the muscles to produce power.

Olympic gold medal prospects thus are most likely to be those who have:

- an interest in training;
- an inherited endowment of physiological attributes related to high-level aerobic and anaerobic performance;
- a high sensitivity of response to training;
- resistance to injury owing to excellent musculoskeletal symmetry; and
- a well designed training programme (Martin & Coe, 1997).

2.4.4 Body size and composition

An estimated 69% of the differences in VO_2 max scores among individuals can be explained simply by variations in body mass (McArdle et al., 1996). If aerobic capacity is expressed in relation to fat-free body mass, however, the difference between the two subjects is reduced

even more. Findings of Washburn & Seals. (1984) suggest that the difference in aerobic capacity between men and women is largely a function of the size of the contracting muscle mass.

Berg et al. (1998) found a strong linear relationship between VO_2 max (L/min) and gross body mass for ectomorphs and mesomorphs, while these two variables are unrelated to endomorphs. VO_2 max (ml/kg/min) is dependent on gross body mass and increases with increasing body mass for ectomorphs and mesomorphs. The opposite is true of endomorphs in which group a strong linear decrease in VO_2 max expressed as ml/kg/min is observed with an increase in body mass.

2.4.5 Age

Both females and males reach their maximal aerobic power around 15–20 years of age (Foss & Keteyian., 1998). For the majority of the population, there is a gradual decline of VO_2 max with age (about 10% per decade), which begins around age 30 (Foss & Keteyian., 1998). This decline, however, more reflects increased inactivity with age, since many studies show that the rate of decline can be markedly reduced if one maintains a regular exercise regimen (McArdle et al., 1996; Foss & Keteyian., 1998). In older individuals, it has been shown that the rate of decline of VO_2 max is much greater than that of the anaerobic threshold (Foss & Keteyian., 1998).

Cross-sectional studies have shown that endurance trained athletes show a significant slower rate of decline of VO_2 max, while maintaining or increasing the percentage of VO_2 max at which anaerobic threshold takes place (Hawley, 1995). Research of Astrand & Rodahl. (1986) showed a decline after the age of 25 of about 1% so that, by the age of 55 it is about 27% below values reported for 20 year olds. Maffulli et al. (1991) show that the rate of decline of VO_2 max in sedentary older individuals is much greater than that of the anaerobic threshold. Studies indicate, that the greater the distance one races, the higher the peak performance age (Noakes et al., 1992). However, this factor could be outweighed by the apparently greater decrease in speed in endurance running than in sprinting. As a result, very long distances can only be run at much slower speeds that do not generate the same levels of shock that the runner could comfortably sustain for many hours when younger.

Studies involving children have detected a wide range of results. Maffulli et al. (1991) found a greater running economy in older pre-pubertal boys that are associated with greater endurance and stride frequency that may influence VO_2 max. Younger runners have larger heart volumes and higher VO_2 max relative to body weight and respiratory capacity. Rowland et al. (1987) also concluded that while prepubertal children demonstrate greater weight-relative maximal aerobic power compared to young adults, endurance times during treadmill running are presumably limited by their lower submaximal running economy. Metabolic cost of respiration is greater in children than in adults. Ventilatory equivalent for oxygen is higher in child runners, who breathe more rapidly with greater ventilation per kilogram at a given work rate. This decreased efficiency of breathing in young subjects has been attributed to both a larger dead space ventilation and alveolar hyperventilation (Rowland et al., 1987). The extra costs resulting from these differences may contribute to lower submaximal running economy in children. Krahenbuhl et al. (1989) stated that run training is not required to improve running economy in active but non-run trained boys. It should be noted, however that growth and run training contributed to better running economy.

2.4.6 Sex

The VO_2 max for women is typically 15% to 30% below that of men (Vogel et al., 1986; McArdle et al., 1996). These differences, however, are considerably larger if the VO_2 max is expressed as an absolute value rather than relative to body mass. Results of Helgerud et al. (1990) showed that performance-matched male and female marathon runners had approximately the same VO_2 max. For both sexes the anaerobic threshold was reached at about the same percentage of the VO_2 max and maximum heart rate. The female's running economy was poorer, i.e. their oxygen uptake during running at a standard submaximal speed was higher.

The apparent difference in VO_2 max between the sexes has generally been ascribed to differences in body composition and haemoglobin concentration. Young adult women, for example, generally possess about 26% body fat whereas the corresponding value for men averages 15% (McArdle et al., 1996). Although trained athletes have lower percentages of fat, trained women still possess significantly more body fat than their male counterparts. Thus, the

average male can generate more total aerobic energy simply because he possesses more muscle mass and less fat than the average female.

Probably because of their higher level of testosterone, men also have a 10% to 14% greater concentration of haemoglobin than women do (McArdle et al., 1996). This difference in the oxygen carrying capacity of the blood potentially enables men to circulate more oxygen during exercise, increasing their aerobic capacities compared with women (McArdle et al., 1996). Apart from the well-known variation in height and differences in the percentage of fat, the difference between performance-matched male and female marathon runners seemed primarily to be found in running economy and amount of training (Helgerud et al., 1990).

There are indications that women's muscles may be more resistant to fatigue than those of men, so that women may in fact have greater endurance than men. To investigate this question, Jenefer Bam, who has represented South Africa in international road-running competitions, compared the performances of men and women, at a range of racing distances, who completed the 1993 Two Oceans Marathon in comparable times (Noakes et al., 1990). Her analysis showed that the men were significantly faster at the distances below 42 km, whereas the women performed better at distances greater than 56 km. In other words, the rate of decline in peak racing speed over increasing distance was greater in the male runners than in the female runners. Whereas the men were faster in the shorter distance races, the less-powerful women were more resistant to fatigue over increasing distance.

These findings raise the perennial question in the gender debate: will women one day surpass men in any running event? At present, it would appear that women are at least 6% slower than men over the popular race distances (up to 42 km) at which very large numbers of men and women compete internationally (Noakes et al., 1990). A comparison between men's and women's 1991 world records indicates a difference of 9%-12% for most Olympic distances. It is interesting to note that Frith van der Merwe's Comrades Marathon record of 5:54:43 for the 'down run' is within 9,2% of Bruce Fordyce's 5:24:07 down run record.

If you take men and women of equal experience and equal 10km times, and then compare their performances over longer distances, a very interesting pattern emerges. While the men ran about a minute faster in the half marathon, this advantage was soon lost, with women

outrunning the men by three minutes in the standard marathon and a good 53 minutes in the 90km Comrades Marathon (Weight, 1998). Women have a natural performance-enhancing substance called oestrogen. Oestrogen, apart from being the most important reproductive hormone, is a unique anti-oxidant that protects the body from the ravage of free radicals. However, if oestrogen protects muscles from this free radical-induced damage, then women athletes would be more able to sustain a consistent pace throughout an endurance event. There is also a possibility that oestrogen promotes fat burning in muscle, thereby conserving glycogen stores (Weight, 1998).

2.5 BLOOD LACTATE

The strong relationship between endurance performance and lactate kinetics led to the suggestion that blood lactate concentration could be used as a training tool (Keith et al. 1992). Lactic acid is produced in any kind of muscular exercise (Gupta et al. 1996).

2.5.1 Formation of lactic acid

Lactate is a product of glycolysis and glycogenolysis (Brooks, 1985). It is both produced and used by the muscles; its rate of production increases as the exercise rate increases and as more carbohydrate is used to fuel exercise (Noakes, 1992). During moderate levels of energy metabolism, the mitochondrial capacity for oxidative metabolism is adequate, and sufficient oxygen is available to the cells (McArdle et al., 1996). Consequently, the hydrogens stripped from glucose and carried by NADH are oxidised within the mitochondria and passed to oxygen from water (McArdle et al., 1996).

Any lactic acid that is formed is oxidised by other tissues at its rate of formation. In a biochemical sense, a "steady state" exists because hydrogen is oxidised at about the same rate, as it becomes available. Biochemists frequently refer to this condition as aerobic glycolysis, with pyruvate being the end product. Aerobic metabolism (at first mainly of glycogen, later increasingly of fat) is the principal route of ATP resynthesis in activities lasting longer than 2 min, but can only maintain work-rates about ¼ of those possible in very brief bursts (Spurway, 1992).

During strenuous exercise, when energy demands exceed either the oxygen supply or its rate of utilisation, the rate of production of hydrogen joined to NADH exceeds the rate at which it can be processed through the respiratory chain (McArdle et al., 1996). Under the conditions of anaerobic glycolysis, NAD^+ is regenerated as pairs of "excess" hydrogens combine with pyruvate in one additional step catalysed by the enzyme lactic dehydrogenase (LDH). This forms lactic acid in the reversible reaction. The terminal enzyme of the glycolytic pathway (LDH) has the greatest catalytic activity of any glycolytic enzyme (Brooks, 1985). Therefore at concentrations of pyruvate found in muscle during sub-maximal exercise, substrate concentration is sufficient to support maximal catalytic activity of LDH in the production of lactate. Thus, during oxygen deficit, lactic acid, rather than carbon dioxide and water, is the end product of cellular respiration of glucose (Marieb, 1989). By comparing the ATP generated in the breakdown of carbohydrate and fatty acids, it is clear that anaerobic metabolism provides only 5.5% as much energy as aerobic metabolism (Martin & Coe, 1997).

Once lactic acid forms in the muscle, it diffuses rapidly into the blood, where it is buffered to form lactate, and is then transported from the site of energy metabolism (McArdle et al., 1996). Fatigue is largely mediated by increased acidity which inactivates various enzymes involved in energy transfer and interferes with the muscle's contractile properties.

Lactic acid should not be viewed as a metabolic "waste product". Rather, it is a valuable source of chemical potential energy that is continually utilised by the body in moderate exercise and accumulates during heavy exercise (Noakes, 1992). Most of the lactic acid diffuses out of the muscles into the bloodstream. When oxygen is again available, the lactic acid is reconverted to pyruvic acid and oxidised via the aerobic pathways to carbon dioxide and water. Thus, when large amounts of ATP are needed for moderate periods (30–40 seconds) of strenuous muscle activity, the anaerobic pathway can provide most of the ATP needed.

2.5.2 Blood lactate accumulation

An untrained individual who has fasted overnight and who has a sample of blood collected in the morning from an arm vein before any exercise, has a lactate level ranging from 0,44 to 1,7 mmol/L. Martin & Coe (1997) also found a lactate range between 0,3 to 0,6 mmol/L for

trained athletes if they are not overtrained. However, one residual effect of either a very hard single training session or a period of overtraining is morning postabsorptive lactate level that is either very high normal or clinically elevated (Martin & Coe, 1997).

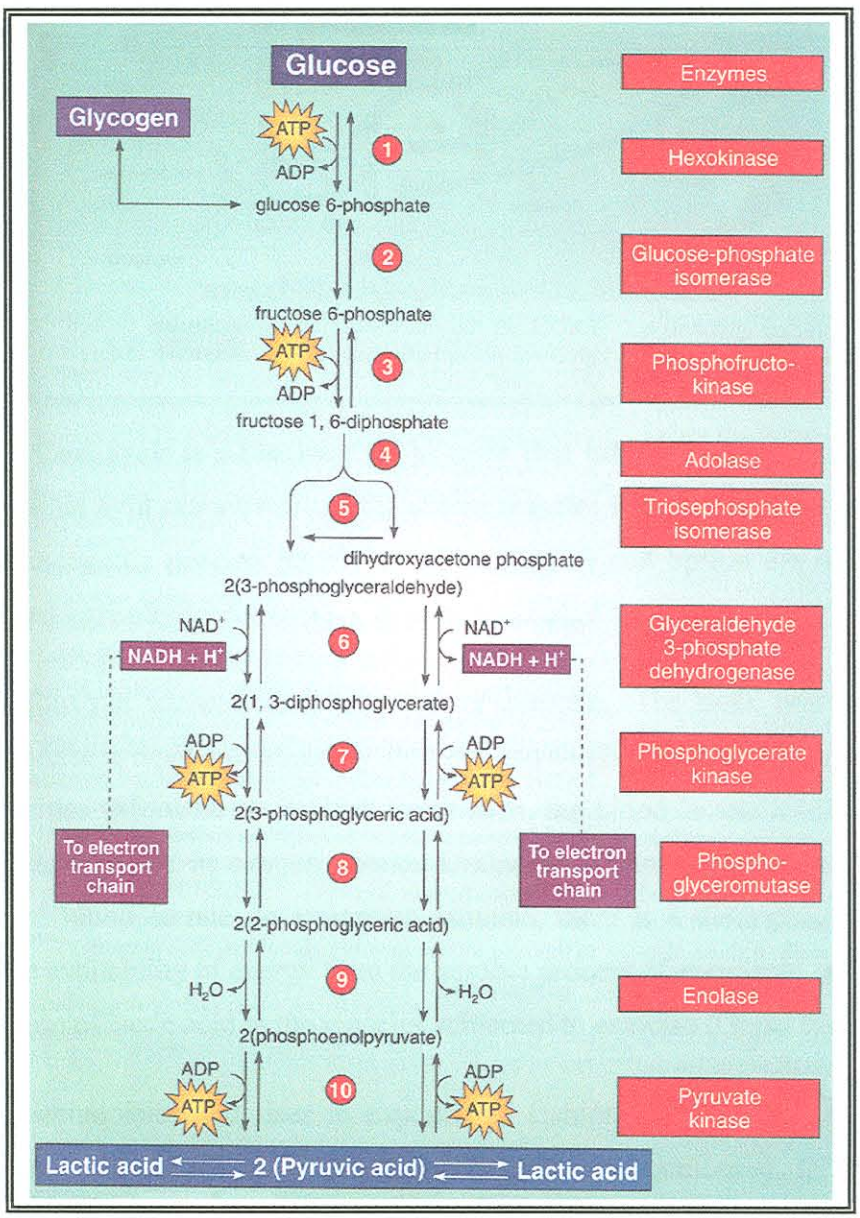


Figure 4: Glycolysis is a series of 10 enzymatically controlled chemical reactions that occur during the anaerobic breakdown of glucose to two molecules of pyruvate. Lactic acid is formed by the process of anaerobic glycolysis when the oxidation of NADH does not keep pace with its formation in glycolysis (McArdle et al., 1996).

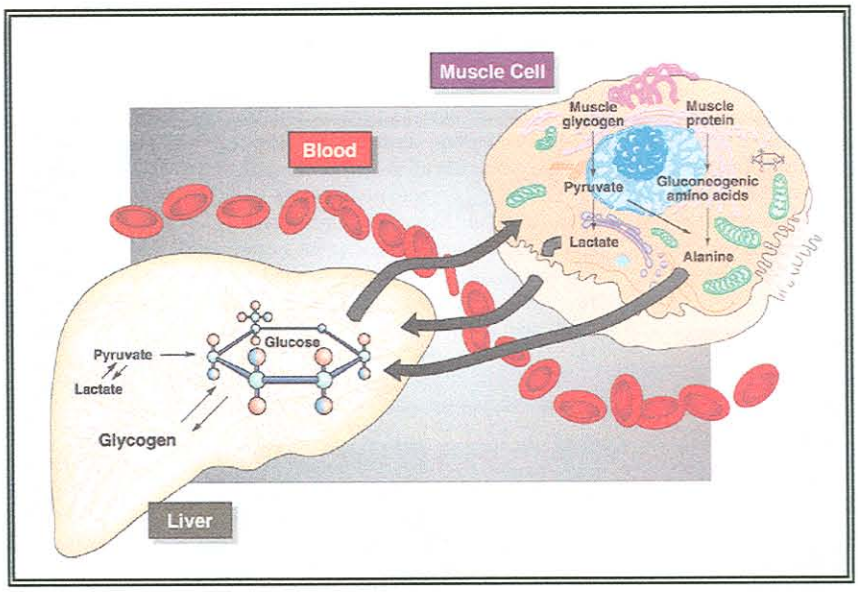


Figure 5: The Cori cycle is a biochemical process that takes place in the liver in which the lactic acid released from the active muscles is synthesised to glucose. This gluconeogenic process provides the body with and option for maintaining its limited carbohydrate reserves (McArdle et al., 1996).

Blood lactate does not accumulate at all levels of exercise. The lactic acid that is formed during light exercise is rapidly oxidised by the heart and neighbouring muscle fibres with high oxidative capacities (McArdle et al., 1996). As such, the blood lactate level remains fairly stable despite an increase in oxygen uptake. Under these conditions, there is little or no accumulation of blood lactate. In strenuous exercise, there is a discrepancy between the demand and the availability of energy from the aerobic process of exercising, which results in a large production of lactic acid in the muscles subjected to exercise (Gupta et al. 1996).

Blood lactate accumulates and rises in exponential fashion at about 55% of the healthy, untrained person's maximal capacity for aerobic metabolism (Costill et al., 1973). One of the factors limiting exercise from the athlete's perspective is an increasing subjective ventilatory discomfort; this heightened respiratory stress may be a more important variable to measure than a blood chemistry variable such as lactate, although is presumably the acidosis that initiates the increased ventilatory drive. The usual explanation for a lactic increase is based on an assumed relative tissue hypoxia during heavy exercise (McArdle et al., 1996). The

accumulation of an excess amount of lactic acid in muscles under stress is a contributing factor to fatigue (Gupta et al. 1996).

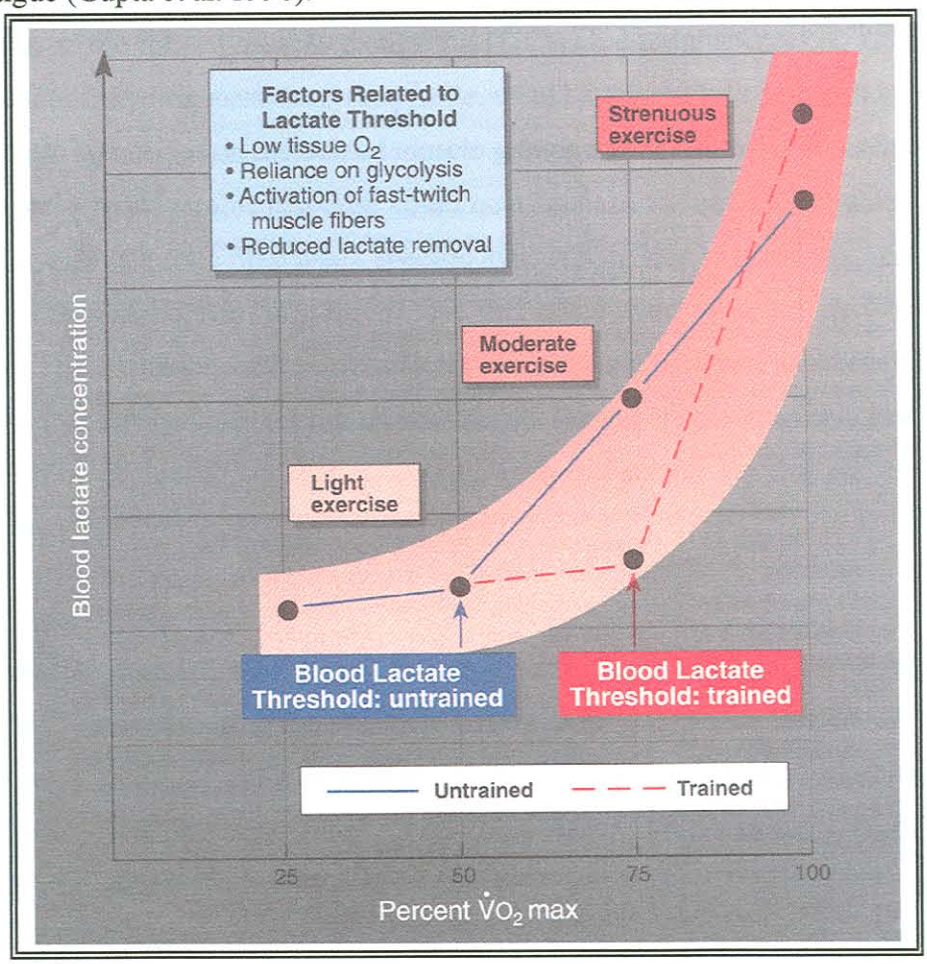


Figure 6: Blood lactate concentration at different levels of exercise expressed as a percentage of maximal oxygen uptake for trained and untrained subjects (McArdle et al., 1996).

Most of the lactic acid produced during vigorous exercise is removed by direct oxidation (55%-70%) while the balance amount is converted to glycogen (<20%), protein constituents (5%-10%) and other compounds (<10%) (Gupta et al. 1996). Lactic acid produced in working muscles is almost completely dissociated into H+ and lactate within the range of physiological pH, which contributes to the metabolic acidosis (Hirokoba et al. 1992).

With aerobic training, cellular adaptations provide for a high rate of lactate turnover, so accumulation occurs only at higher exercise levels. The increased oxidative potential and capillarization of the trained muscle groups together with a training-induced shift to a more oxidative lactate dehydrogenase isozyme profile, would favour enhanced availability, uptake, and oxidation of lactate within the trained muscle groups during exercise (McLellan & Jacobs, 1989). The favourable aerobic response could be a result of the endurance athlete's specific genetic endowment (muscle fibre type), specific local adaptations with training that favour the production of less lactic acid, or a more rapid rate of its removal at any particular level of exercise intensity. Trained endurance athletes, for example, exercise at intensities that are between 80% and 90% of their maximum capacity for aerobic metabolism and there is little or no increase in blood lactate concentration until an exercise intensity that elicits 70-85% of VO_2 max (Hawley, 1995).

2.5.3 Lactate Steady State

During steady-rate exercise, aerobic metabolism is matched to the energy requirements of the active muscles. Under these conditions, there is little or no accumulation of blood lactate; any lactic acid that is produced is either oxidised or reconverted to glucose, predominantly in the liver and possibly in the kidneys. Blood lactate does not accumulate to any appreciable extent under steady-rate metabolic conditions. In the steady-state, it is assumed that the rates of appearance in and disappearance from the blood equal the intracellular production and removal rates (Brooks, 1985). Theoretically, once a steady state has been attained, the athlete could continue indefinitely if he has the willpower to continue (McArdle et al., 1996). Other factors, however, should also be considered: these include fluid loss and electrolyte depletion, adequate fuel reserves, particularly liver glycogen, blood glucose and muscle glucose.

After the onset of exercise below the lactate threshold, VO_2 rises monoexponentially until a steady state is reached, usually within 2-3 min (Jones et al., 1999). For running speeds above lactate threshold the VO_2 at 6 min was consistently higher than the VO_2 at 3 min, suggesting the existence of a slow component that increases the VO_2 requirement of the exercise above that which would be predicted from the VO_2 response to sub lactate threshold exercise (Jones et al., 1999).

Although mean lactate values representing a maximal steady-state during continuous exercise were found to be close to 4 mmol/L, individual values varied from 3–5.5 mmol/L (McLellan & Cheung, 1992). Stegman et al. (1981) recognised the extent of this individual variation in maximal lactate steady-state values and introduced the concept of the individual anaerobic threshold (IAT)

Several studies have documented that endurance performance is more strongly related to the metabolic rate associated with various indices of lactate kinetics during submaximal exercise than with maximal oxygen consumption. Prolonged exercise at individual anaerobic threshold for 50 minutes has been shown to result in steady-state lactate values (McLellan & Jacobs, 1989). Stegman et al. (1981) reported that well-trained athletes could exercise at their IAT for 50 minutes with individual steady-state blood lactate values varying from 2 to 7 mmol/L. Especially in highly trained endurance athletes the maximum lactate steady state seems to be reached at lower threshold intensities (Urhausen et al., 1993).

Blood lactate does not accumulate to very high levels during exercise that lasts more than an hour. A good example of this is during marathon running. At the end of a marathon, trained athlete's blood lactic acid is only two to three times that found at rest (Costill et al., 1967).

The changes in blood lactate concentration during incremental exercise tests to exhaustion have been examined in many ways in order to identify significant levels that might have a bearing on sustained physical performance (Orok et al., 1989). In general, the incremental intensity exercise tests are based on exercise periods of between 3-5 minutes. These durations are considered as adequate when measurements of oxygen consumption and heart rate are performed, because these variables usually are in steady state after 3-5 min (Orok et al., 1989). However, Foxdal et al. (1996) found that incremental exercise with durations shorter than 8 minutes is usually not in steady state. Several possible explanations can be postulated when considering results from previous studies. Firstly, there is the release of lactate from the muscle to the blood, which can be dependent on different transport capacity over the muscle sarcolemma. Such a dependency could possibly cause a time dependent rate-limiting barrier for the lactate to reach the interstitium and the blood vessels. Secondly, there is a time dependent dilution effect of the released muscle lactate into the blood, which is also related to

the microcirculation of blood through the active muscle tissue and the capillary density. Thirdly, there is the capacity for lactate elimination in both active and inactive muscles, in the heart and in other organs (Foxdal et al., 1996).

Research into ventilation and the maximal lactate steady-state (MLSS) indicates that ventilation exhibits a threshold phenomenon and that the breathing frequency of 32 breaths/min is the rate most associated with the maximal lactate steady-state (Palmer et al., 1999). Palmer et al. (1999) also reported significant correlations between endurance race pace velocity and the MLSS.

2.5.4 Onset of blood lactate accumulation (OBLA)

The term lactate threshold refers to the highest exercise level or level of oxygen uptake that is not associated with an elevation in blood lactate concentration above the pre-exercise level (or with an increase of less than 1,0 mM) (McArdle et al., 1996). The region in which blood lactate shows a systematic increase equal to or above a level of 4,0 mM is termed the point of onset of blood lactate accumulation or simply OBLA (Rieu et al., 1990; Seip, 1991; Foxdal et al., 1996). Often the terms lactate threshold and OBLA are used interchangeably. In adults, distance-running performance is related more to submaximal effort measurements, such as the onset of blood lactate accumulation and anaerobic threshold, than to VO_2 max (Maffulli et al., 1991).

The 4mmol/L value for OBLA implies the maximum exercise intensity that a person can sustain for a prolonged period. In reality, this maximum stable lactate level is probably quite variable among individuals (Noakes 1988; Orok et al., 1989; Mognoni et al., 1990). The higher the running speed at which the lactate concentration exceeds the 4 mmol/L threshold, the higher the aerobic capacity. Spurway (1992) called 2 mmol/L the aerobic and 4 mmol/L the anaerobic threshold.

Almost all the lactic acid generated in anaerobic metabolism is buffered to lactate in the blood by sodium bicarbonate in the following reaction:

$$\text{Lactic acid} + NaHCO_3 \rightarrow \text{Na Lactate} + H_2CO_3$$
$$\downarrow\uparrow$$
$$H_2O + CO_2$$

The excess, non-metabolic carbon dioxide released in this reaction stimulates pulmonary ventilation, and CO_2 is exhaled into the atmosphere. Because blood lactate accumulation is associated with changes in carbon dioxide production (respiratory exchange ratio) via blood buffering, blood pH, bicarbonate, and H^+ concentration, these variables have been used to indirectly assess OBLA (McArdle et al., 1996). "Bloodless" techniques such as changes in the R, V_E/VO_2 or various fractional concentrations of expired gas during incremental exercise to signal the onset of metabolic acidosis can also be used (McArdle et al., 1996).

Long-distance runners have somewhat lower maximal oxygen uptake values than do middle-distance runners, but they run at exceptionally high percentages of VO_2 max before the onset of blood lactate accumulation occurs (Louanne et al., 1989). Schneider & Pollack, (1991) has defined the anaerobic threshold as the VO_2 at which lactic acid begins to accumulate in the blood during incremental exercise. At that point, lactic acid diffuses into the blood at a faster rate than it can be cleared. Generally speaking, threshold heartrate is between 82%-88% of maximum. However, for some people it is as low as 65%, while the Kenyan runners do not appear to reach the threshold until around 94% (Weight & McGee, 1998).

In young adults the anaerobic threshold corresponds to an oxygen uptake of about 70% of the aerobic power, the point of inflexion of the lactate curve has been shown to correspond to 82% of aerobic power in 11 year old boys (Mocellin et al., 1990). The anaerobic threshold is much lower in boys compared to adults in absolute terms, but it is distinctly higher in terms of percentages of VO_2 max. Incremental intensity blood lactate exercise tests, such as the 4,0 mmol/L OBLA test, have been accepted as valid and reliable estimators of aerobic endurance performance. Moreover, these tests have been used for setting aerobic training intensities not exceeding the intensity corresponding to a maximum lactate steady state (Foxdal et al., 1996). Intensities above the anaerobic threshold led to a distinct increase in the number of athletes showing progressive lactate acidosis and premature break-off - when anaerobic threshold was

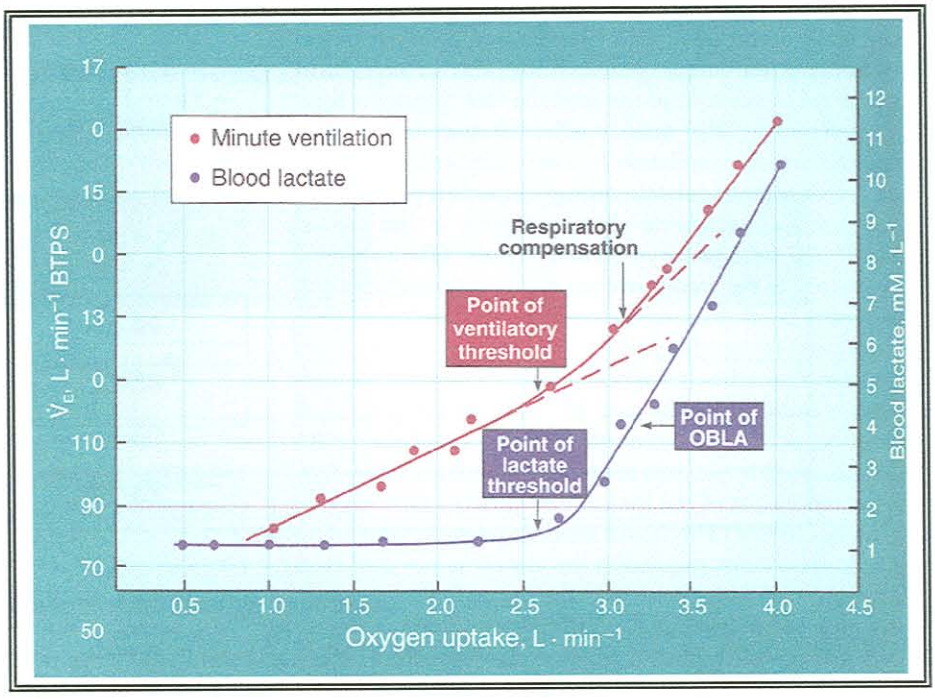


Figure 7: Pulmonary ventilation, blood lactate, and oxygen uptake during graded exercise to maximum (McArdle et al., 1996).

exceeded by only by 5% (Urhausen et al, 1993). Optimal times in marathon and similar events are achieved by performing at 97 - 100% of lactate threshold (Hagberg, 1984) while events of 5 - 10 000 m type require running speed nearer OBLA (Davis, 1985).

Training can improve OBLA without a concomitant increase in the VO_2 max. This indicates that the OBLA and VO_2 max are determined by somewhat different factors. The muscle mass activated during exercise and the muscle fibre type, capillary density, mitochondrial size and number, and alterations in a muscle's enzymatic and oxidative capabilities, play a major role in establishing the percentage of aerobic capacity that can be sustained in exercise with little lactate accumulation. According to McArdle et al. (1996), two important factors that can influence endurance performance are the VO_2 max (the maximal capacity to consume oxygen) and the maximal level for steady rate exercise or OBLA.

The table below illustrates how faster runners over 16 km had lower blood lactate concentrations than their slower counterparts, despite running at the same relative intensity of 85% of VO_2 max (Costill et al. 1973).

Time over 16 km	Blood lactate concentration at 85% of VO_2 max
48-49	2.7
53-56	3.7
57-60	5.0
61-68	6.2

2.5.5 Lactate removal after exercise

When exercise intensity exceeds 60%–75% of VO_2 max, a steady rate of aerobic metabolism is no longer maintained, lactic acid formation in muscle exceeds its rate of removal, and blood lactate accumulates (McArdle et al., 1996). The blood lactate level does provide an objective indication of the relative strenuousness of exercise and may also reflect the adequacy of the recovery process (Jacobs, 1987). Performing active aerobic exercise in recovery (McLellan & Jacobs, 1989; Falk, 1995) accelerates blood lactate removal. Blood lactate will continue to rise and will peak at about 5 minutes post exercise, therefore the importance of a cool down session after exercise (Martin & Coe, 1997).

The optimal level of recovery exercise is between 29%–45% of VO_2 max on a bicycle and 55%–60% for running (Karlsson & Jacobs, 1982; Jacobs, 1987). Fox et al. (1993) suggested that active recovery should be nearer 70% of VO_2 max for the first few minutes, then about 40% of VO_2 for the later recovery period. Data from Orok et al. (1989) also indicated that blood lactate concentration decreases most rapidly during exercise at approximately 40% VO_2 max. Athletes with higher fitness levels also have a quicker recovery of lactate removal because of their greater mitochondrial density, blood perfusion, and enzyme capacities.

Research clearly show that moderate aerobic exercise during recovery facilitates lactate removal more effectively than passive recovery. The combination of higher-intensity followed by lower-intensity exercise was of no more benefit than a single level of exercise of

moderate intensity (McArdle et al., 1996). If recovery exercise is too intense and is performed above the lactate threshold, it is of no added benefit and may even prolong recovery by initiating lactate formation (McArdle et al., 1996; Gupta et al., 1996).

The facilitated removal of lactate with recovery exercise is likely the result of an increased perfusion of blood through "lactate using" organs such as the liver and heart. Increased blood flow through the muscles during active recovery certainly would enhance lactate removal because this tissue can oxidise lactate via Krebs cycle metabolism (McArdle et al., 1996). Most of the lactic acid produced during rigorous exercise is removed by direct oxidation (55%-70%) while the balance amount is converted to glycogen (<20%), protein constituents (5%-10%) and other compounds (<10%) (Gupta et al., 1996).

Lactic acid, rather than being viewed simply as a "waste product" of metabolism, should be thought of as a fuel source for muscle and as a source for the partial regeneration of liver and muscle glycogen. During recovery from high-intensity exercise, blood lactate is both oxidised to CO_2 and resynthesized to glycogen.

When a running test is done on a treadmill until exhaustion, at least one hour is required to remove all the lactic acid (Fox et al., 1993). In general, 25 minutes of rest-recovery are required following maximal exercise, to remove half of the accumulated lactic acid. This means that about 95% of the lactic acid will be removed in 1 hour and 15 minutes of rest-recovery from maximal exercise.

During the transient phase (first 4 minutes), lactate during isolated exercise increased considerably, but decreased markedly if supramaximal exercise had been performed previously (Rieu et al., 1990). It may be assumed that the energy contribution of anaerobic glycolysis, and hence lactate production, are negligible in the first minutes of exercise preceded by previous supramaximal exercise on the treadmill, inasmuch as there is no accumulation of lactate in the blood. If the energy expenditure and efficiency are unmodified, the minimal contribution of anaerobic glycolysis could be explained by the fact that the oxygen deficit of the first 4 minutes is reduced (Rieu et al., 1990).

2.5.6 Buffering of lactic acid

The normal pH of arterial blood is 7,4, that of venous blood and interstitial fluid are 7,35, and intracellular fluid averages 7,0 (Marieb, 1989). The lower pH of cells and venous blood reflects their greater amounts of acidic metabolites (such as lactic acid) and carbon dioxide, which combines with water to form carbonic acid. Whenever the pH of arterial blood rises above 7,45, a person is said to have alkalosis. A drop in pH to below 7,35 results in acidosis.

Chemical buffers act within a fraction of a second to resist a pH change and are the first line of defence. Adjustments in respiratory rate and depth begin to compensate for acidosis and alkalosis within 1-3 minutes (Marieb, 1989). Studies of Martin & Coe (1997) indicated that intracellular muscle pH may fall as low as 6,8. Athletes working very hard sense this acidosis subjectively. Thus, the increasing metabolic acidosis becomes an intolerable stress. Exactly what limits exercise is therefore probably more easily answered subjectively as a symptom-limiting situation involving an intolerable effort sense in either the limb muscles or the breathing muscles. When anaerobic energy-transfer predominates, lactic acid accumulates and the acidity of muscle and blood increases (McArdle et al., 1996). This has a dramatic negative effect on the intracellular environment and the contractile capability of active muscles. This factor has led to the speculation that anaerobic training may enhance short-term energy capacity by increasing the body's alkaline reserve (McArdle et al., 1996). Such a training adaptation would theoretically enable greater lactic acid production because it could be buffered more effectively. Although this reasoning seems appealing, only a small increase in alkaline reserve has been noted in athletes compared with sedentary counterparts (McArdle et al., 1996).

Furthermore, there is no appreciable change in alkaline reserve following hard physical training. The consensus is that trained people have a buffering capability that is within the range expected for healthy untrained individuals (McArdle et al., 1996). High-intensity anaerobic exercise performance can be enhancing by temporarily altering the acid-base balance in the direction of alkalosis. Hirokoba et al. (1992) reported that it was unlikely that body-buffering capacity could be changed by endurance training for only two months. The increase in carbon dioxide excess per unit of body mass per lactate accumulation after

endurance training may be accounted for by a factor other than body buffering capacity, which accompanies endurance training (Hirokoba et al., 1992).

This enhanced performance was achieved through ingestion of a buffering solution of sodium bicarbonate prior to an 800 metre race (McArdle et al., 1996). The significantly faster run times were accompanied by higher levels of blood lactate and extra-cellular H^+ concentration, suggesting an increased anaerobic energy contribution to this exercise (McArdle et al., 1996). The hydrogen ions of lactic acid are buffered by bicarbonate, producing excess (non-metabolic) carbon dioxide. This excess carbon dioxide is thought to stimulate an increased rate of ventilation (Schneider & Pollack, 1991; Hirokoba et al., 1992). McNaughton et al. (1999) conclude that the addition of sodium bicarbonate to a normal diet proved to be ergogenic benefit in the performance of short-term, high-intensity work.

Thus, the presence of accumulating H^+ ions helps to initiate a marked increase in local blood flow to the working muscles and to enhance their oxygen supply. Both H^+ ions and carbon dioxide are potent inhibitors of smooth muscle tension generation in blood vessels. This action dilates the small, local blood vessels in the working skeletal muscles, which increases local blood flow. Greater oxygen availability from increases circulation and breathing permits more aerobic metabolism. Removal of CO_2, lactate and H^+ ions helps to slow the development of acidosis in these active tissues. Eventually, increases in circulation and respiration permit O_2 delivery to catch up with demand almost completely. It has been thought by some that the so-called second-wind phenomenon – which can be defined as a sudden improvement in general comfort and ability to tolerate pace following several minutes of running – may correlate with initial achievement of this aerobic metabolic dominance, when external respiration catches up with internal respiration (Martin & Coe, 1997).

2.5.7 Measuring blood lactate

A number of difficulties are associated with the measuring of blood lactate. First, athletes prefer a racelike, uninterrupted treadmill protocol, which obviously cannot be maintained if athletes have to stop periodically for fingertip or earlobe blood collection. MacDougall et al. (1991) stated that progressive work increments must be small enough to avoid undue increases in lactate and local muscle fatigue. Also the initial work rates must be of a low enough

intensity to serve as a warming up. Beginning at high work intensities presents the risk that oxidative energy production will be unable to increase to a maximal rate before lactate accumulation or other factors force cessation of the exercise. Secondly, there are technical quality-control problems in analysing capillary as opposed to venous blood. Capillary blood collected following lancet puncture will certainly be contaminated with interstitial fluid and possible with sweat as well. Only if the lancet puncture is firm enough to provide plenty of free-flowing bloods will it be essentially arterialised rather than predominantly capillary blood. If the first drop is wiped away, because it will be contaminated with interstitial fluid, collection of the next drop must be rapid to prevent clotting at the puncture site. The site must not be massaged to enhance flow, because this will alter the blood composition (Martin & Coe, 1997). An alcohol swab applied to the site between lactate samples will avoid lancing for each sample, particularly when the work intensity becomes high, but thorough drying is necessary to avoid diluting the blood (MacDougall et al. 1991)

Anaerobic responsiveness can be measured in several ways, according to Martin & Coe (1997). One is to note the length of time the VO_2 max plateau is maintained. Another is to compare the length of time the athlete is working with a respiratory exchange ratio greater than 1,0, which indicates a respiratory compensation to increasing metabolic acidosis, and to identify the maximum R value achieved during the test. A third is to measure VCO_2 max. A fourth is to compare the 5 min post-test maximal blood lactate level. Although effective endurance training ought to lower blood lactate concentrations observed at any given submaximal work load, a higher maximal lactate suggests greater tolerance to anaerobic work. Finally, a fifth observation is to evaluate subjectively the athlete's stability during the final few moments before test termination.

2.6 FATIGUE

2.6.1 Definition

Historically, muscle fatigue has been defined as the failure to maintain force output, leading to a reduced performance (Fitts, 1994). More recently, fatigue is defined as failure to maintain the required or expected power output. This definition recognises the ability to sustain a given work capacity without decrement requires the maintenance of both force and velocity.

2.6.2 Oxygen consumption and fatigue

Fatigue induced by submaximal long duration exercise significantly increases the aerobic demand of running. Following prolonged activity, increases in VO_2 have been associated with increases in heartrate, core temperature, fat catabolism and blood catecholamines levels as well as decreases in biomechanical efficiency, muscle glycogen and liver glycogen content (Zavorsky et al., 1998). The rise in VO_2 indicated worsened running economy and the increase in heartrate is to compensate for a decreased stroke volume.

Zavorsky et al., (1998) reported a rise in VO_2 by 5% during 3 hour steady state exercise in moderate environmental conditions. An increase in core temperature induces an increase in minute ventilation with accompanying oxygen cost for the respiratory muscles. Increased ventilation, enhanced oxygen extraction, or a combination of these mechanisms should account for the increase in VO_2. Billat et al. (1994) indicate that the average time to exhaustion at VO_2 max ranges between 2 min 30 s and 10 min.

2.6.3 Lactic acid and fatigue

Fatigue caused by lactic acid accumulation has been suspected for many years. However, only recently has a relationship between intramuscular lactic acid accumulation and decline in peak tension been established (Fox et al., 1993). The accumulation of an excess amount of lactic acid in muscles under stress is a contributing factor to fatigue (Gupta et al., 1996). When an athlete starts a race too fast, lactate will accumulate to very high levels and muscle glycogen stores will be depleted early in the race. Costill (1967) found only a 2–3 times increase in lactate values after trained athletes completed a marathon. The fatigue experienced by these runners is therefore caused by factors other than high blood lactic acid levels. Other factors that could lead to fatigue are:

- low blood glucose levels owing to depletion of liver glycogen stores;
- local muscular fatigue owing to depletion of muscle glycogen stores;
- loss of water and electrolytes, which leads to a high body temperature; and
- boredom.

In addition, interindividual variability in the degree of fitness could affect the relationship between lactate concentration in capillary blood at a given exercise intensity during

incremental exercise and the resistance to exercise fatigue (Mognoni et al., 1990). Lactic acid, which causes the pH value in muscles to drop, causes extreme fatigue, which limits the usefulness of the anaerobic mechanism for ATP production. During the transmission of action potentials, potassium is lost from the muscle cells and excess sodium enters. As long as ATP is available to energize the Na^+ and K^+ pump, these slight ionic imbalances are corrected. However, in the absence of ATP, the pump is inactive, and ionic imbalances finally cause the muscle cells to become non-responsive to stimulation (Marieb et al., 1989). The lactate accumulation is represented as the ratio of lactic acid concentrations in fast twitch (FT) and slow twitch (ST) fibres. This means that as the ratio increases, more lactic acid is being produced in FT fibres in comparison with ST fibres. This greater ability to form lactic acid might be one contributing factor to the higher anaerobic performance capacity of the FT fibres. As the lactic acid FT : ST ratio increases, the peak tension of the muscle decreases. This may be interpreted to mean that the greater fatigability of FT fibers is related to their greater ability to form lactic acid (Foss & Keteyian, 1998).

Barstow et al. (1996) showed that the relative magnitude of the slow component during heavy exercise was significantly correlated with the proportion of FT muscle fibers. This study strongly suggest that the VO_2 slow component is related in some way to the recruitment of FT motor units during heavy exercise. Study of Foss & Keteyian (1998) indicate that the composition of fiber types varies within different regions of the same muscle, between different muscles within the same person, and certainly within the same muscles of different people. It is thus important to consider such distributions in athletes and their potential impact on performance.

2.6.4 Time to exhaustion

According to Kindermann et al. (1979) exercise characterised by a lactate concentration of 4 mmol/L may be carried out for 45-60 min and occasionally, longer. The results of Mognoni et al. (1990) also found the time to exhaustion 45-60 min with a lactate concentration of 4.3 mmol/L. The data of Stegmann & Kindermann (1982) are not in agreement with the above results. In fact, most of their athletes were no able to carry out a steady state lactate intensity equal to 4 mmol/L over 50 min. They were exhausted after 14 min. These results are probably the consequence of an overestimation of the threshold intensity.

Mognoni et al. (1990) conclude that the effect of blood and muscle lactate on resistance to fatigue decreases rapidly with time. The anaerobic threshold is very often determined in order to obtain the corresponding heart rate value which is used to set the exercise intensity in endurance training so that there is no, or little lactic acid accumulation. However, the exercise duration should also be kept in mind as lactate increase with prolonged exercise.

2.6.5 Differences between black and white runners

In South Africa, black runners dominate all running distances greater than 3 to 5 km. Interestingly, black South African runners regularly dominate the annual world listings for the 15 and 21 km. For example black South Africans recorded 11 of the top 15 times at 15 km in 1990. This suggests that the same genetic pool for distance running excellence that exists in East and North Africa must also be shared by black South Africans (Noakes, 1992)

Bosch et al. (1990) showed that sub-elite black runners matched with white runners for best 42 km marathon time had slightly lower VO_2 max values than white runners but compensated for this by sustaining a significantly higher % VO_2 max during marathon races. Bosch et al. (1990) reported 89% of VO_2 max for the black runners and 81% of VO_2 max for the white runners in which both groups ran at the same percentage of their best marathon race speed. The black runners in Coetzer et al. (1993) also study reported that they trained at high average exercise intensity than the white runners. Thus, a better resistance to fatigue. When the very best white and black athletes, with the same VO_2 max compete against each other, then the black runners were outperform the white runners in endurance events like marathons. Because of their greater capacity to run for longer at a higher percentage VO_2 max and hence at a faster running speed (Noakes, 1992).

Despite the superior fatigue resistance of the black distance athletes, however, their skeletal muscle fiber composition did not show a preponderance of type I fibres as might have been expected. In both the black and white runners, there was a high proportion of FT fibres, similar to that described for middle-distance runners (Coetzer et al., 1993). However the FT fibers of the black runners are fundamentally different from those of the white middle-distance runners, and are characterised by extreme fatigue-resistance (Noakes, 1992).

The main anthropometric differences between the runners in Coetzer et al. (1993) study were that the black runners were significantly shorter and lighter than the white middle-distance track athletes and had a considerably smaller muscle mass and lean thigh volume. They also had smaller front thigh and medial calf skinfold thickness. Thus the inertia of the limbs would be less and so theoretically less energy would be expended when moving the limbs (Bosch et al., 1990).

Lower blood lactate concentrations were found in the black runners. The lower blood lactate concentration at any given running speed might have contributed to the superior fatigue resistance of the black athletes. The lower peak RER values in black athletes were due to a lesser hyperventilatory response to maximal exercise, which in turn was possibly related to their lower blood lactate concentration. Bosch however found that the difference in blood lactate was so small in black and white runners, that the physiological importance is questionable. Coetzer et al. (1993) also measured a large difference between the quadriceps strength in black and white runners. Strength was better in the white athletes and fatigue better in the black athletes.

Bosch et al. (1990) stated that during a marathon black runners had lower V_T but higher breathing frequency than white runners. This combination resulted in similar minute ventilation volume. The higher breathing frequency in the black runners is almost certainly due in part to a smaller V_T as a result of a smaller vital capacity compared to the white runners.

It seems very probable that these specific characteristics explain the remarkable dominance of distance running by black East Africans athletes, especially Kenyans. Whether they are the result of both a culture in which running is a natural daily activity and residence at altitude, or whether they are inherited traits, remains to be established (Noakes, 1992).

2.6.6 Difference between male and female runners

In the study by Ramsbottom et al. (1989) women demonstrated lower respiratory exchange ratios and lower blood lactate concentrations throughout the test. Thus at a running speed equivalent to a blood lactate concentration of 2 mmol/L the women were able to utilise a higher proportion of their maximal oxygen consumption than were the men. The ability to

maintain a high proportion of VO_2 max has been suggested to be a better predictor of conditioning or training status than a determination of the maximal oxygen uptake alone (Noakes et al., 1990). Therefore using this criterion, the women appear to have a higher aerobic capacity than the men in this study of Ramsbottom et al. (1989).

2.7 VELOCITY

2.7.1 Oxygen consumption and velocity

There is no doubt that a high VO_2 max constitutes a kind of membership card for entrance into the world of top level middle-distance and distance running excellence. But anaerobic aspects of performance also contribute to the difference between finishing first and second in a race, because they interact with VO_2 max.

Noakes et al. (1990) found that peak treadmill velocity reached during the VO_2 max test was a better predictor of running performance than VO_2 max for all distances from 10-90km. Noakes et al. (1990) reported that race time at 10 km or 21 km is the best predictor of performance in both the 42.2 km marathon in specialist marathon runners and in the 90 km ultra-marathon in the specialist ultra-marathon runners. That indicates that in trained marathon and ultra-marathon runners are also the fastest over the shorter distances. Thus, in agreement with Hawley & Noakes (1992) the relation between power output and oxygen uptake is linear. Kuipers & Arts. (1994) reported that the relationship between the power output and oxygen uptake and heart rate is linear for the absolute values as well as the percentages of maximum power output, VO_2 max, and maximum heart rate.

Thus, two important physiological variables are important in evaluating distance-running abilities. One is the velocity at anaerobic threshold – the pace at which blood lactate just starts to rise substantially. Marathon pace is slightly slower than this. The other is the velocity at VO_2 max, which typically is close to 3000m race pace (Martin & Coe, 1997).

The larger the VO_2 max, the smaller the runners total anaerobic contribution will be at any given pace, or the faster they can run before anaerobic effects start to impair performance. But once VO_2 max has been elevated about as high as possible without inordinate additional training volumes, anaerobic development will make the additional difference between being

optimally fit and marginally fit. Noakes et al. (1990) study showed that the physiological variables determining success at distances from 10-90km are not different, at least in marathon and ultramarathon specialists. This suggests that with appropriate training for longer distance events, the fastest 10km runners will also be the fastest marathon and ultramarathon runners. Billat et al. (1994) reported that the subjects capable of sustaining the maximal aerobic speed for longer period of time were also those who displayed a marked increase in lactate concentration at a later stage of a progressive exercise test and those who run a 21.1 km race faster.

Daniels (1985) suggested that the interplay of VO_2 max and running economy could be expressed by calculating the predicted running velocity at VO_2 max. According to Daniels, this derived variable may be useful in explaining performance similarities among competitive distance runners who differentially possesses the desirable attributes of VO_2 max and running economy.

Research by Morgan et al. (1989) indicated that there is a significant relationship between 10 km run time and velocity at VO_2 max and appears to be mediated to a large extent by running economy. This data from Morgan et al. (1989) suggest that velocity at VO_2 max is a useful index of training status and therefore a non-invasive predictor of distance running performance. Nevill et al. (1992) stated that subjects with higher VO_2 max values produced steeper regression lines (together with lower intercepts) than subjects with lower VO_2 max measures.

2.7.2 Lactate levels and velocity

Anaerobic metabolism may will occur among the Comrades athletes at the start, or while running up hills, but the lactic acid concentration in the blood immediately after a standard marathon (Costill, 1970) and the Comrades Marathon (Jooste et al., 1981) is fairly low. These facts emphasise the conviction that the Comrades Marathon is run mainly on aerobic energy. The pace of each athlete is thus limited largely by his lactic acid turning point and in such a way that this point is not exceeded (Jooste et al., 1981). Palmer et al. (1999) reported that marathon runners finished their races in a time 3 to 7 min faster if they had been running at

velocities above the maximal lactate steady state. This indicate that lactate may build during the race to reach a level above threshold by the finish.

2.9 RUNNING ECONOMY

2.9.1 Definition

Hawley (1995) defines economy of motion as the cost (i.e. oxygen uptake) required to produce a specific work rate or speed of movement. The best endurance athletes are usually the most efficient (Noakes, 1988). Better economy (i.e. lower oxygen cost) is advantageous during endurance exercise because it is associated with a slower rate of energy utilisation (i.e. muscle glycogen). Daniels & Daniels (1991) defines running economy as the relationship between oxygen consumption and velocity of running, or as the aerobic demands of running.

Previously published results show a decrease in energy cost of running across speed ranging from 40%-60% VO_2 max and an optimal zone of energy cost between 60% and 80% VO_2 max in trained runners (Brisswalter et al., 1996). This indicates that elite distance runners have a physiologically non-optimal speed (too slow a pace), and a physiologically optimal speed.

2.9.2 Factors that affect running economy

The question that remains unresolved is why some individuals demonstrate markedly better economy when compared with counterparts exhibiting similar fitness and performance backgrounds. An alternative hypothesis is that successful long distance runners may have a structural or anatomical makeup, which genetically predisposes them towards better economy. Bailey & Pate (1991) introduced the concept of "external energy" – the energy needed to overcome external resistance, and "internal energy" the energy used in the production of external energy. In this way, running economy could, theoretically, be improved by reducing the demand for external energy, internal energy or both at any given submaximal running velocity. Previous research has indicated that the between-subject variation in running economy can be as much as 20-30% among trained male and female runners of similar ability (Williams et al., 1991).

There are many factors that affect running economy i.e. age, sex, training, stride rate and frequency, shoe weight, wing are air resistance, including lower density found at altitude (Daniels & Daniels., 1991). Furthermore, clothing, footing, terrain, and possibly fatigue are additional factors that can change the cost of running. Brisswalter & Legros. (1994) indicate that elite runners display a wide range of daily variation in the energy cost of running that is independent of variation in stride rate or respiratory parameters. The application of this information should help coaches to design better training programs that will improve the running economy and endurance performance of both male and female athletes.

2.8.2.1 Age

Children are less economical than adults are; running at common submaximal speeds elicits a greater relative demand for oxygen in children (Krahenbuhl & Williams., 1991). They also concluded that running economy improves steadily with age in normally active children. This improvement occurs with or without participation in formal running training programs. Some of the reasons why children are less economical than adults are that when compared with adults they exhibit higher resting metabolic rates, greater ventilatory equivalents for oxygen at a given running pace, and disadvantageous stride lengths. Cross-sectional research (Morgan et al., 1989) indicates that the gross energy cost of running increases 2% per year from 8-18 years of age.

2.8.2.2 Sex

Daniels & Daniels. (1991) concluded that at absolute running velocities, men are more economical than women, but when expressed in ml/kg/min there are no gender difference at similar relative intensities of running. Also, when men and women of equal VO_2 max or equal economy are matched, the men show a better aerobic profile. Some studies however showed that there are no differences in running economy (Pyne, 1994). Daniels & Daniels. (1991) recommended the economy date must be collected up to speeds equal to over 90% of VO_2 max. Much of the variance in physiological parameters can be accounted for by differences in body composition and the proportions of fat free mass. Pyne (1994) proposed that smaller individuals possess a relatively greater amount of his or her body mass in the extremities, and would therefore perform a relatively greater amount of work moving body segments during running than larger individuals. Given that female runners are, on average, smaller than their

male counterparts, it is possible that this might be one explanation for the relatively poorer economy in female runners.

Morgan et al. (1989) speculated that the higher stride frequency and greater oxygen debt exhibited by the females might have contributed to the higher overall energy cost of running. Morgan & Craib (1992) also suggested that females may exhibit greater vertical displacement of the body during running, which would theoretically require a higher aerobic demand because of the added muscular effort needed to lift the body a greater vertical distance. Other factors observed were differences in stride frequency, running-experience and training intensity.

2.8.2.3 Training

In a statistical sense, approximately 64% of the total variation in 10km running performance among athletes can be explained by the variations in running economy. (Morgan & Craib, 1992). Noakes et al. (1990) noted a relationship between peak treadmill running velocity and running economy; those athletes who reached the highest treadmill running velocities were also the most economical. This suggests that with appropriate training for longer distance events, the fastest 10km runners will also be the fastest marathon and ultramarathon runners. The positive correlation found between running economy and run times from Housh et al. (1988) further substantiates that the faster runner were also more metabolically economical. Morgan & Craib (1992) stated that athletes who specialists in shorter distance events have been shown to exhibit better economy at faster speeds, whereas long-distance specialists tend to be more economical at slower running speeds.

Krahenbuhl & Williams (1991) found that instruction on techniques of running, at least over a short term (2-3 months) is ineffective in bringing about improvements in running economy. However running training results in little or no improvement in running economy during childhood and adolescence. Over the longer term (years), improvements in running economy may be augmented through participation in running training programs (Krahenbuhl & Williams, 1991). Morgan et al. (1989) concluded that growth-related factors and training were likely causes for the enhancement in running economy.

There is no single biomechanical factor that accounts for individual differences in running economy (Martin & Morgan, 1992; McArdle et al., 1996). Significant variation in economy observed at a particular running speed occurs even among trained runners. (Conley & Krahenbuhl, 1980; McArdle et al., 1996). In general, improvements in running economy can result from long-term programme of running (Conley & Krahenbuhl, 1980; McArdle et al., 1996). Morgan et al. (1995) stated that trained distance runners are more economical compared with untrained subjects and that economy differences between trained and untrained subjects may be a function of repeated exposure to moderate training loads.

Ideally, runners are most concerned with being optimally economical at race pace. The longer the race and the smaller the anaerobic racing component, the greater will be the influence of running economy on performance quality. Thus marathoners can probably benefit most either from above-average running economy through genetic factors or from specific training to improve it. This has been offered as an explanation for the rather low VO_2 max values recorded among some top-level marathon runners (Martin & Coe, 1997). Better running economy was associated with a lower heartrate and ventilation (Bailey et al., 1991). Training-induced reductions in heartrate and ventilation might produce an overall drop in total body VO_2 leading to lower aerobic demands (Morgan & Craib, 1992).

2.8.2.4 Stride rate and frequency

Elite runners also appear to choose an optimal stride length at which they are most efficient and, when forced to take either longer or shorter strides for the same running velocity, they require an increased oxygen uptake, thus becoming less efficient (Hawley, 1995). Based on these results, Morgan et al. (1989) concluded that there is little need for a coach to dictate a particular stride length profile in most athletes since they tend to display nearly optimal stride lengths. They suggested that this phenomenon might be due to two mechanisms. The first states that runners may gravitate naturally toward an optimal stride length/stride rate combination over time an interactive process based on perceived exertion. A second possibility is that runners may adapt physiologically through repeated training at a particular combination of stride length and stride frequency for a given running speed.

In the study by Rowland et al. (1987) both adults and children elected to increase stride length rather than frequency as treadmill speed increased. This further supports the idea that greater running economy is achieved by increasing stride length rather than frequency. Bailey & Pate (1991) shown that stride length and running economy differ between experience and novice runners, with experienced runners possessing longer stride lengths and greater running economy. The results indicated that the most economical runners possessed a significantly lower force peak at heel strike, greater shank angle with vertical at heel strike, smaller maximal plantar flexion angle following toe off, greater forward trunk lean, and lower minimum velocity of a point on the knee during foot contact.

2.8.2.5 Fatigue

Economy takes on considerable importance during longer-duration exercise, where success depends largely on the aerobic capability of the individual and the oxygen requirements of the task (McArdle et al., 1996). Zavorsky et al. (1998) also stated that fatigue induced by submaximal long duration exercise significantly increases the aerobic demand of running. For exercises lasting more than two hours (e.g. marathon), it has been shown that the running economy decreased at the end of a long-distance run (Hausswirth et al., 1996). Thus fatigue affects economy in a negative way, increasing aerobic demand through the use of increasingly tired prime movers plus others brought into action to help maintain pace.

Following prolonged activity, increases in heart rate, core temperature, fat catabolism and blood catecholamine levels as well as decreases in biomechanical efficiency, muscle glycogen and lower glycogen content (Bailey and Pate, 1991). An increase in core temperature induces an increase in V_E with accompanying oxygen cost for the respiratory muscles. Increased ventilation, enhanced oxygen extraction, or a combination of these mechanisms should account for the increase in VO_2 (Zavorsky et al., 1998).

Possible explanations of fatigue:

- include inadequate recovery time following intense training periods to permit proper nutrient, electrolyte, and fuel replenishment (Martin & Coe, 1997);

- use multiple muscle groups during exercise (running up hills with a vigorous arm swing to accompany leg motion), thereby diminishing the fall in muscle cell glycogen for any particular muscle groups (Martin & Coe, 1997);
- the contractile ability of the muscle falls progressively during prolonged exercise possibly on the basis of thermal damage to muscle (Davies and Thompson, 1986);
- increase stored fuel supplies in the working muscle before a major competition;
- inadequate fluid intake while running (Noakes, 1992)

Data obtained on elite and trained endurance runners performing longer runs have produced conflicting results, which one study reporting higher aerobic demands following a competitive distance race (Cavanagh et al., 1985), and others demonstrating no change in economy 1 day after a hard training workout (Martin et al., 1987). Morgan et al. (1990) replicated the Martin et al. (1987) study by expanding the experimental design. These findings suggest that an intense 30-min training run or a competitive 10 km race would not raise the aerobic demand of running by increasing dependence on fat metabolism or disrupting the gait pattern in subsequent submaximal runs over the short term. Viewed from a theoretical perspective, these results demonstrate the imperturbability of the metabolic and biomechanical profiles of trained runners following a prolonged maximal run. In a more recent study by Zavorsky et al. (1998) they conclude that running economy is worsened after repeated hard efforts, quantification of the time needed to re-establish baseline economy values would assist athletes in optimising training.

2.8.2.6 Temperature

A number of studies have documented the effect of increased core temperature (Q10 effect) on VO_2. Morgan et al. (1989) reported a 5% rise in VO_2 during 3 hours of constant-load exercise under normal conditions. Variety of reasons for this rise in VO_2, including an increased energy requirement for peripheral circulation, increases sweat gland activity, hyperventilation and a decreased efficiency of energy metabolism. Bailey & Pate (1991) have suggested that training-induced adaptations to exercise in the heat, such as an increased plasma volume, may attenuate the magnitude of the thermoregulatory response and reduce attendant energy requirements.

2.8.2.7 Body mass

Davies (1980) found that children carrying 5% of their body weight on their trunk while running displayed a lower aerobic demand at faster speeds compared to unloaded running. Morgan et al. (1989) also observed a modest inverse relationship between body mass or weight and economy in elite female runners. However, greater body mass in the trunk area appears to be advantageous in terms of running economy. Conversely, those individuals who possess greater percentages of their body mass in the arms and legs may be able to obtain higher VO_2 max values because a greater proportion of their lean muscle mass is active during running (Bailey & Pate, 1991).

2.8.3 Determination of running economy

Martin & Coe (1997) estimate the submaximal O_2 demand (economy) at each pace as the average of three 20-s expired gas samples collected during the final minute of running at a pace. Using the statistical technique of regression analysis, an equation can be written using the pace and VO_2 data that best describe each runner's O_2 consumption with increasing work load. This regression equation determined from data obtained during level running permits extrapolation to the level-ground pace at which the athlete would be running at VO_2 max intensity. Thereby, *velocity at VO_2 max* or *v- VO_2 max* can be calculated.

The question asked now is, is the relationship between running velocity and O_2 consumption linear or curvilinear? The present evidence seems almost in favour of curvilinearity. If the relationship were linear throughout, the slope of the regression lines obtained by most workers who evaluate the economy of distance runners would be essentially parallel, differing only in that the more efficient runners would be positioned lower than the less efficient runners because of their decreased O_2 cost at submaximal paces (Martin & Coe, 1997).

However, studies by Kearney & Van Handel (1989) state information from other published studies suggesting that a range of faster running velocities results in a steeper slope than does a range of lower running velocities. Thus, although some studies quoted frequently regarding the relationship whereby 1 kcal of energy is required per kilogram of body weight per kilometre of distance covered submaximally, this linearity may not necessarily be true for energy demands beyond the lactate/ventilatory threshold and approaching VO_2 max.

Morgan et al. (1990) reported that a stable measure of running economy could be obtained in a single data collection session if the testing environment is controlled to minimise nonbiological variability. Also to maximise the likelihood of securing accurate baseline measures of running economy and running mechanics, it is recommended that subjects be evaluated at the same time of the day, in the same footwear and in a nonfatigued state (Williams et al., 1991). It appears that the importance of economy may be expressed only when performers are of comparable ability with similar maximal aerobic capacities (Conley & Krahenbuhl, 1980).

Costill (1979) provides the following information regarding oxygen uptake at set speeds:

Type of runner (running speed)	12 km/hr	14.5 km/hr	16 km/hr	19.2 km/hr
VO_2 ($mlO_2/kg/min$)				
Very efficient	35	42	48	58
Average efficient	38	46	52	61
Inefficient	43	51	57	66

2.9 TRAINING

Over the past century, there have been massive assaults on athletic performance records. For example, the world's best running performance in the marathon and the 100 m, 400 m, and 1500 m races have all fallen sharply from the 1900s to the present, with an average of about 25% (Foss & Keteyian, 1998). Athletes who strive to compete at the highest levels must realise that it takes time to build the excellence required. Long-term goal setting permits an athlete to assign relative importance to the various aspects of a training year. The need to simply score points for the team or to be seen in a sponsoring firm's new line of sportswear without being prepared to do one's best is a very difficult pill to swallow for an athlete who desires excellence. Racing and training are very different entities and demand different mental attitudes. Nowadays, extreme pressures are placed on top-level athletes to disrupt their

development and overall goals by travelling to far-flung destinations and racing for huge sums of money to satisfy their sponsoring firms.

Martin & Coe (1997) said that it was important to focus on a career plan for an athlete with potential. A career plan is important to identify both the competitive event for which excellence can most likely be achieved and the age at which athletes typically will be at their best for that distance. The average age of career-best performance in marathon running is 29,3 ± 4,9 for men and 26,8 ± 3,9 for women (Martin & Coe, 1997). Thus, it is important to organise an athlete's career effectively.

Without question, improved training techniques and methods, resulting partly from out better understanding of the physiology of exercise, have played a pivotal role in improving athletes record breaking performances (Foss & Keteyian, 1998).

2.9.1 Principles of training

The objective of training is to bring an athlete to a peak fitness level at the proper time, with all the requirements for good performance brought along in balance.

The four primary aspects of an adaptation to training are:

- initial tissue catabolism that occurs from the load applied and causes an initial reduction in performance capabilities;
- adaptation to the stress of training as a result of tissue recovery and improved mental outlook from having successfully completed the work;
- retention and likely improvement in such performance characteristics following a tapering of training; and
- reduction in performance if training volume is decreased for too long a period (Martin & Coe, 1997).

Thus, the training life of an athlete is a constant cycle of hard work, recovery (regeneration), improvement in performance, and a brief layoff (for mental and physical rest) to permit another cycle to repeat. Martin & Coe (1997) define *multi-tier* training as the organisation of training around several levels of which builds on the preceding one. The effectiveness of multi-tier training is scientifically based. Multi-tier training continually exposes athletes to a

wide range of training stimuli with varying emphasis, thereby decreasing risk for injury or excessive fatigue.

2.9.1.1 Intensity

Training intensities are commonly defined as percentages of maximal oxygen uptake or maximal heart rate. But according to Meyer et al. (1999) should not only these parameters alone be used to determine exercise intensities. Individualised concepts bases on lactate measurements are preferable.

McArdle et al. (1996) named seven ways to express exercise intensity:

- calories expended per unit time (kcal/min or kJ/min);
- power output (kg-m/min or W);
- relative metabolic level as a percentage of VO_2 max;
- level of exercise below, at or above the lactate threshold;
- percentage of maximum heart rate;
- multiplies of resting metabolic rate (METs)
- rate of perceived exertion (RPE)

Londeree (1995) provides the selected values for percent VO_2 max and corresponding percentages of heart rate max:

Percent HR max	Percent VO_2 max
50	28
60	40
70	58
80	70
90	83
100	100

Gilman & Wells (1993) suggested that energy production is primarily aerobic when exercise intensity is below the ventilatory threshold. Ventilatory threshold is considered the upper limit for easy intensity exercise. Hard intensity exercise may be identified by fatigue in the legs after a few minutes. Gilman & Wells (1993) considered OBLA as the lower limit of hard

intensity. Physical activity requiring a metabolic rate between ventilatory threshold and OBLA is considered moderate intensity exercise.

The metabolic reference points occur not only at different percentages of VO_2 max, but also at different percentages of maximal heart rate. Therefore, it may be possible to design individualised training programs based on heart rates at ventilatory threshold, OBLA, and VO_2 max. These reference heart rates can then be used to monitor training intensity relative to metabolic factors.

Gilman & Wells (1993) study shown that the subjects in his study spent nearly all their training time performing easy to moderate intensity running where as the majority of their race time occurred at a high intensity level. Thus training prescriptions based on heart rate at designated metabolic markers with subsequent heart rate monitoring will enable coaches and athletes to accurately monitor training intensity. The question still remains whether there is a single best training intensity for promoting endurance performance.

2.9.1.1.1 Long-duration, moderate intensity training

This method of endurance training involves 30 min to 2 hr or more of continuous exercise, usually performed over relatively long distances and is often referred to as LSD training (Foss & Keteyian, 1998). The intensity of exercise generally increases heart rate to 75% to 85% of maximum or 60% to 70% of VO_2 max. For most athletes this pace is below lactate threshold, and as a result, it is slower than race pace (Foss & Keteyian, 1998).

However, the optimum training intensity for improving endurance performance remains established; on theoretical grounds it has been suggested to be the maximum intensity that can be maintained in a steady state. Jacobs (1986) suggested that a blood lactate concentration of 4 mmol/L represent the optimum intensity. But according to Jacobs (1986) trained endurance runners lower than the optimum training intensity. According to Noakes & Granger (1995) the focus in the base training period should be on the time spent in the exercise zone, rather than the speed at which the athlete run. The intensity should be slower than race pace (85% to 95% of race pace), athlete should be able to do the talk test (Noakes & Granger, 1995). However, Sjodin et al. (1982) presented early evidence the training at an intensity approximating the maximal lactate steady state may be superior to other training.

2.9.1.1.2 Moderate-duration, high intensity training

This method of training is often referred to as pace or tempo training. Exercise intensity is set very near an athlete's lactate threshold – as heart rates that approach 85% to 90% of maximum (Foss & Keteyian, 1998). Exercise is still continuous in nature but duration is shortened (30 – 60 min). The approach is similar to interval training but differs in that the:

- length of the work interval is longer;
- and the degree of recovery between repetitions is shorter.

The main objective of aerobic interval training is to improve tolerance to racing at lactate threshold. This method of training is very effective for improving VO_2 at lactate threshold and/or economy of movement (Foss & Keteyian, 1998). The heart rate method of determining intensity will establish a level of exercise stress for central circulation (for example stroke volume, cardiac output), whereas adjustment to lactate threshold are dictated by the capability of the periphery (local vasculature and active muscles) to sustain steady-rate aerobic metabolism (McArdle et al., 1996).

Heck et al. (1985) suggesting that elite runners who were stagnated in their performance improved only after decreasing the intensity of their training to that associated with the aerobic anaerobic threshold. Weltman et al. (1992) have demonstrated that at least some training above the lactate threshold is required for improvement. However, Lehmann et al. (1992) suggests that athletes respond better to increases in training intensity than training volume. If training at the maximal steady state was the "best" method to exercise one would have hypothesised that the increased volume study would have been tolerated better than increases in the homeostasis disturbing training intensity (Snyder et al., 1994). However if training intensity associated with maximal lactate steady state is not the best form to exercise in, it may be that this intensity simply represents the most time effective way of integrating training volume and intensity (Snyder et al., 1994). Yoshida et al. (1992) demonstrates the training-induced improvement in VO_2 kinetics was more apparent for the OBLA exercise intensity than for the initial lactate threshold intensity. Robinson et al. (1991) found that the mean intensity of steady state running for the subjects in this study is considerable lower than the optimal training intensity.

2.9.1.1.3 Short-duration, very high intensity training

This method of training involves a modification of interval training; in fact it is often called interval sprinting. The session involves for example a 50 m sprint then a 60 m jog over a distance of 4 – 8 km. Runners often incorporate hills into this type of training (Foss & Keteyian, 1998). Improvements in performance occur in trained runners when intensity of training increased and it is known that optimum improvements in cardiorespiratory fitness occur when training is at an intensity corresponding to 90-100 % of maximum oxygen uptake (Robinson et al., 1991). Noakes & Granger (1995) reported that high intensity training should never last longer than 14 weeks and this period should include at least three recovery weeks. Evidence of Noakes (1992) proves beyond doubt that the faster the athlete at short distances, the greater his or her potential in the marathon and ultimately in the ultramarathon. Matthews Temane, who has run the fastest mile at altitude, holds the world 21.1 km best; Bruce Fordyce had the fastest mile, 5 000 m and 10 000 m times of all finishers in the Comrades Marathon and Firth van der Merwe and Eleanor Adams are one of the fastest female ultra athletes and both also compete in shorter events (Noakes, 1992).

2.9.2 Methods of training

The old question thus remains: How much training and what kind of training are ideal and sufficient? Optimal training can be defined as doing the least amount of the most specific work that will continually bring improvement in fitness (Martin & Coe, 1997).

Gustav Brink, Chairman of the Road Running Coaches Committee, reported a number of different training phases for a distance runner:

- base training phase;
- endurance training phase;
- speed phase;
- tapering phase; and
- racing phase (Brink, 1999).

A runner has to build a proper base before attempting to enter the next phase. It is preferable that the phases be followed in the order indicated above in order to peak for a specific event.

In the case of ultra distances, such as Comrades, there may not be sufficient time remaining after "loading" i.e. running extreme weekly distances, to properly incorporate a speed phase, and this phase may be done before the endurance phase.

2.9.2.1 Base training phase

Base training should be at a comfortable level, i.e. it should not leave the athlete fatigued and the athlete should recover from any base training session within 24 hours. Base training for a 10km runner would differ considerably from that of an ultra runner. A 10km runner would do approximately 30-50km per week with strides once or twice a week. The long run would not exceed a maximum of 15 km. In the case of club running for the Comrades i.e. someone who would run 100 to 120km per week for a period of 4 to 6 weeks in the build-up to Comrades. The base training for this athlete should be at least 50 to 60km per week, incorporating a long run of at least 18 to 20km. Evidence form Hickson & Rosenkoetter (1981) suggest training frequency rather than training intensity is responsible for the increase mitochondrial enzyme concentration and endurance exercise capacity.

Martin & Coe (1997) question the benefit of more than 115 to 120 km a week at low intensity aerobic conditioning paces for distance runners seeking to improve their VO_2 max. Although marathon runners are special cases in requiring very high training volumes in order to stimulate greater fuel storage abilities in their working muscles. Thus, once aerobic conditioning has provided the initial stimulus, it should then be followed by lower total weekly volume with higher-intensity aerobic capacity training sessions to bring VO_2 max to its peak for that particular training period.

2.9.2.2 Endurance training phase

Endurance exercise training leads to modest improvements in maximal oxygen uptake but large increases in the capacity to sustain submaximal exercise to exhaustion (Ramsbottom et al., 1989). The large increase in submaximal endurance capacity is thought to be closely related to the increases in muscle oxidative capacity. It is well documented that endurance training results in lowered blood lactate concentrations at the same absolute and relative exercise intensity after training which suggests an increased proportion of the energy supply derived from fat metabolism or greater aerobic catabolism of substrates *per se*.

Over the shorter events, most serious runners would train "over distance" i.e. runs longer than the end goal. Accordingly, a half marathon runner may run training runs of up to 25km, while a 5000m runner will occasionally train over distances of up to 15km. The great New Zealand coach, Arthur Lydiard, had even his 800m runners, such as Peter Elliot, occasionally run distances of up to the marathon during their endurance training. Long runs for the 5km to 21.1km runner will invariably be run at a pace 20 to 30 seconds/km slower than race pace. A marathon runner, mindful of the effect that very long runs have on the body and the recovery time required, may choose not to train beyond 35 to 38km in preparation for a hard marathon, although some of the elite athletes do train over distances of up to 45km. Serious Comrades runners invariably train over distances of up to 75km, but usually have just one or two runs, five or six 60-70km runs, and several shorter 40-50km runs. Even the serious Comrades runner would not train "over distance". Most of the long runs are at a pace slightly slower than race pace, while the very long training runs (over 60km) would be run significantly slower than race pace (Brink, 1999).

Noakes (1992) stated that elite runners perform best in the marathon and ultramarathon races when they train between 120 to 200 km per week, with an increasing likelihood that they will perform indifferently when they train more than 200 km per week.

It is possible that increased volumes of aerobic training alone can improve fitness and provide a similar economy of motion. The studies of Scrimgeour et al. (1986), which report that athletes training less than 60 km a week have as much as 19% less running economy than athletes training more than 100 km a week, might support this suggestion. Martin & Coe (1997) report that as athlete became better trained, not only does the VO_2 max rise, but so does the lactate/ventilatory threshold, both in absolute terms and as a percentage of VO_2 max. These long runs are nor just important for endurance development but also for mental preparation for the Comrades.

A good rule of thump is to have run 5-6 runs during the endurance phase and to add another 2-3 such runs during the strength and speed phases of training. The last run should be 3-4 weeks prior to the race to give the body the opportunity to fully recover before the race. The distance

of long run should be as least 80% or more for the half marathon and shorter distances, and 60% of the distance in the case of a marathon and 50% for the Comrades (Brink, 1999).

Muscle endurance can also be increased through track work, by doing long repeats on the track at a pace slightly (5-10 seconds/km) faster than race pace. If the race goal is a marathon at a speed of 4 min/km, a sample track session in this phase would be 5x2000m at 3:50-3:55/km, with 1:00 passive or 400m active recovery. Muscle endurance can also be trained through cross training such as cycling, swimming, and gym, provided the sessions are done at a similar intensity and for reasonably long periods of time. In the gym the athlete would work with a lighter weight, and then do 2-3 sets of 20-40 repetitions with 60-65% of maximum weight (Brink, 1999).

2.9.2.2.1 Strength training phase

Resistance training is one component that will result in a stronger endurance runner. Runners who avoid resistance training fear it will compromise their performance but fail to realise that resistance training leads to physiological adaptations, both acute and long-term, that may actually improve performance. Dolezal & Potteiger (1996) named several physiological adaptations that take place when doing resistance work.

- ***Muscle enlargement***

  Increased muscle size usually means increased strength, which can enhance performance.
- ***Muscle contraction time***

  Research reveals that strength training does not affect a muscle's contraction time and therefore will not slow one down. In fact, many athletes have shown increases in speed of movement.
- ***Body composition changes***

  Strength training increases lean body mass and decreases fat weight and the percentage of body fat. This improvement help maximise performance.
- ***Neural factors***

  Initial strength gains may be attributed to the recruitment of additional motor units and reductions of inhibitory impulses. These changes enhance motor skill performance.
- ***Fuel availability***

Significant increase in the intramuscular stores of energy leads to increased performance and delayed fatigue response.

- ***Enzymatic changes***
  Increased enzymatic activity leads to more energy production and more efficient energy use.
- ***Bone density***
  There is strong evidence that strength training can increase bone mineral content, thus guarding against stress fractures or providing a safety factor in the case of falls.
- ***Connective tissue***
  Physiological adaptations in ligaments and tendons due to strength training may help prevent injuries.

Johnston et al. (1995) found that low volume training of moderate to high intensity, when incorporated in an endurance training programme will significantly improve upper and lower body strength as well as running economy. They stated that the benefits from increased upper body strength help delay fatigue in the arms and postural muscles during a race. As the muscles become fatigued, they may compromise the efficiency of movement and increase the oxygen demand for running because additional motor units are recruited. Oxygen cost at each running speed may be reduced if a more efficient pattern is induced through an increase in leg strength. However some studies showed no improvement in endurance performance after resistance training (Hawley, 1998). It appears that there is a minimal amount of muscle strength required performing well in any endurance event. This explains why the greater muscle power seen after most resistance training increases short-term endurance capacity in these athletes.

Runners are much less out of breath during a marathon, particularly during the latter stages, but are far more fatigued in their legs after a 10km. This indicates the importance of muscle endurance. Muscle endurance is the ability of a muscle to endure a sub-maximal workload over an extended period of time. The impact on an athlete's legs when running on a level surface is four times the athlete's bodyweight. The impact on a downhill can increase as much as eight times the athlete's bodyweight (McGee, 1998). Therefore the importance of eccentric contraction exercises to improve muscle endurance and decrease risk of injury.

2.9.2.3 Speed training phase

Running at faster speeds demands the utilisation of a greater proportion of the maximal oxygen uptake and an increased contribution to the energy supply from anaerobic metabolism. The end result is that lactate accumulates in the blood in increasing concentrations (Ramsbottom et al., 1989). A strong correlation has been reported between the onset of plasma of blood lactate accumulation and distance running performance (Rieu et al., 1990; Seip, 1991; Foxdal et al., 1996).

Long-distance racing is about endurance and speed together. Jenkins & Quigley (1992) reported that endurance training appeared to induce a fall in the y-intercept (anaerobic capacity), and although the change was not significant, the implication is that the improved endurance ability diminished the capacity of high-intensity exercise. Thus, endurance athletes have higher anaerobic thresholds than non-athletes, where the anaerobic threshold is expressed as a fraction of VO_2 max (Haffor et al., 1991). Endurance will get a runner to the finishing line, but speed will get a runner there quicker. This means that speed and varying degrees of speed must be practised to be done well.

2.9.2.4 Tapering phase

Endurance runners often reduce training volume for 5 – 21 days prior to an important competition in an effort to enhance performance. Houmard et al. (1990) found that a 70% reduction in training volume, while maintaining training intensity, resulted in unchanged aerobic power and 5 km race performance. In a more resent study by Houmard et al. (1994), reported that a 7 day systematic reduction in training volume of low intensity (60% VO_2 max) did not improve treadmill time to exhaustion at a fixed pace (1 500m race pace). In contrast a 7 day high intensity taper improved this index of performance by 22% (Shepley et al., 1992). Hickson (1981) found that one of two thirds reduction in training frequency or duration had no affect on VO_2 max over 15 weeks as long as training intensity was maintained. However, the ability to sustain a high percentage of this oxygen uptake appears to have been diminished based on elevated RER and blood lactate levels during submaximal exercise (McConnel et al., 1993). Shepley et al. (1992) findings suggest that a high-intensity taper provides an unique stimulus that may lead to enhanced distance running performance.

Intense distance running has been demonstrated to decrease muscular power (Houmard et al., 1994), however muscular power is positively associated with distance running performance. Therefore any effort to maximise power should be made. A 7 day taper consisting of high-intensity running increased muscle citrate synthase activity by 18% in will-trained distance runners (Shepley et al., 1992). An elevation in mitochondrial capacity could minimise lactate production and any corresponding inhibition of the contractile process and thus improve both performance and running economy. Mode-specific training during taper is necessary for performance enhancement (Houmard et al., 1994).

Top-level marathon runners who remain injury free with excellent general fitness can work well with a cycle that repeats every four to five months. There are 10 to 12 weeks of intense preparation, a few weeks of tapering, then the race, and a month of mental and physical recovery (Lenzi, 1987). This is why the world's healthiest and most consistent marathoners typically compete no more than two to three time a year.

Newton's tapering approach was to reduce his training by 15% the third week before the race, and by a further 10% in the second week before the race, so that still running 75% of his usual mileage only seven days before the race. By comparison, Bruce Fordyce rests much more during the last ten days before Comrades than did Newton, despite the fact that Fordyce runs less total mileage. This suggests that Newton probably did not rest sufficiently before races and this might have explained the occasions when his performance was not optimal (Noakes, 1992).

2.9.2.5 Training summary

Endurance training is the most crucial element in any long distance training programme and becomes more important the longer the goal race distance is. A runner can, within the limits, still run a reasonable marathon or Comrades without doing strength or speed work, but without the necessary endurance work, the athlete will fail to finish the race.

2.9.3 Physiological consequences of training

2.9.3.1 Effect of endurance training on the VO_2 parameters

It has been will documented that cardiorespiratory and metabolic responses to exercise may be modified rapidly by exercise training. The study by Ramsbottom et al. (1989) demonstrated a decrease in the ventilatory equivalent for oxygen with endurance training. This decrease represents a more efficient utilisation of oxygen and, may reflect an increased mechanical efficiency of the running action. Therefore increased aerobic capacity of human skeletal muscle. Wasserman et al. (1986) suggest that lactate production during exercise depends mainly on the availability of oxygen in the active tissue. Therefore the decrease in blood lactate concentrations with endurance training.

The well-documented adaptation within endurance trained muscle suggest greater perfusion and an increased transit time for blood through muscle after training. Such adaptations would facilitate an improved availability of oxygen and substrates to the active muscle cell while promoting the rapid removal of metabolic waste products (Ramsbottom et al., 1989). Such adaptations within human skeletal muscle may be especially important during marathon and ultra running events like the Comrades Marathon.

Yoshida et al. (1992) named three factors that contribute to the improvement of VO_2 parameters after training, i.e.:

- an improved capacity for mitochondrial respiration in muscle;
- an increased availability of blood and / or muscle O_2 stores; and
- an elevation of cardiac output and / or an increase in muscle blood flow.

In this context, Yoshida et al. (1992) documented that endurance training induces an improvement of mitochondrial respiratory function resulting in a reduced production of lactate during heavy exercise.

2.9.3.2 Can VO_2 max be raised by training?

The answer relates to inherent genetic endowment, but the level of fitness at which one begins the training programme also plays a role. Recent studies of Makrides et al. (1985) have shown that untrained people over a broad age range who embark on a serious aerobic fitness

development programme can raise their sedentary-lifestyle VO_2 max values by as much as 40% or more. Among already established top-level runners, this percentage increase is considerably less.

Although some researchers claim that VO_2 max among elite-level runners changes little over the course of a year, Martin & Coe (1997) find substantial differences as either training load or training emphasis shifts. As an example, the VO_2 max of one of their male middle-distance runners increased by 18%, from 4,695 ml/min to 5,525 ml/min, over a seven-month period. Ramsbottom et al. (1989) stated that training status could be better explained in terms of the highest proportion of the VO_2 max at which a steady-state could be achieved rather than VO_2 max alone. Earlier investigations all demonstrated that training induced improvements in endurance capacity were accompanied by an increase in exercise intensity at which blood lactate accumulates and at which ventilatory threshold was achieved. Weltman (1990) reported that it is possible to increase velocity and VO_2 at OBLA without changing VO_2 max.

Housh et al. (1988) has reported that trained males exhibited lower submaximal steady-state VO_2 responses than untrained males. Therefore, while short term endurance training may not be effective in modifying running economy, it is possible that prolonged training could result in improved biomechanical efficiency and therefore lower submaximal steady-state VO_2 values.

2.9.3.3 Effect of training on heart rate response

It has been well documented that heart rate at a given exercise intensity decreases significantly after endurance training. Yoshida et al. (1992) demonstrated that as well as a reduction in heart rate during constant exercise test; the increase of heart rate at the onset of exercise was speeded up by the endurance training. Furthermore, since endurance training has been shown to increase stroke volume during constant exercise, it is postulated that cardiac output kinetics at the onset of exercise might be improved by endurance training.

2.9.3.4 Interval training versus long slow distance training

Interval training appears to enhance running economy, and improvements seem to occur when the number of interval sessions per week is increased (Douglas et al., 1981). It is probably best to include some interval work at speeds equal to or slightly faster than the pace at which

optimal economy is desired. Endurance training has little effect on VO_2 max, whereas interval training brings about greater, more rapid improvement. It appears, therefore, that interval training is essential for the development and maintenance of maximal aerobic capacity.

2.9.3.5 Effect of training on lactate and ventilatory threshold

Training at or slightly above OBLA has been found to be the most effective way to develop aerobic fitness (Spurway, 1992). It is well established that exercise of differing durations, number of repetitions, or levels of severity can bring about different degrees of glycogen depletion in the active muscles. Glycogen depletion was shown to cause depression of glycolysis and lactate production, resulting in a rightward shift in the lactate response curve and OBLA point (Dotan et al., 1989). Yoshida et al. (1990) reported that in distance runners and competitive walkers, blood lactate variables such as lactate threshold (VO_2 at threshold, and velocity at threshold) and OBLA (VO_2 at OBLA, velocity at OBLA), and running velocity were significantly improved after extra endurance training, but VO_2 max was not significantly improved. Yoshida et al. (1992) also stated that during the first few weeks of training decreases have been observed in blood lactate concentration and heart rate during submaximal exercise, and increases in lactate threshold.

2.9.3.6 Effect of endurance training on weight loss

Chad & Wenger (1985) reported that the duration of activity rather than the intensity have a greater effect on the post-exercise oxygen consumption. The implications of the metabolic after-effects of exercise making a contribution in a weight reduction program, is in considering the total energy expenditure of prolonged exercise and the importance of extending the work time for elevating energy expenditure. Hagberg (1980) found that rectal temperature is significantly higher following longer duration exercise (20 minutes versus 5 minutes) at work intensities of 50% to 80% of VO_2 max and elevated temperatures after exercise are associated with elevated post exercise VO_2. Previous studies indicated that lower fat-free body weight is one of the variables primarily characterising the faster endurance runners (Costill, 1967; Housh et al., 1988).

2.9.3.7 Fast (FT) and Slow twitch (ST) fibres

Recent research studies involving comprehensive physiological evaluation of male Kenyan distance runners have been quite illuminating in helping to explain whether it is genetics (FT/ST muscle fiber ratios) or lifestyle (altitude residence, a hard lifestyle in younger years), or both that make the Kenyans such good distance runners. As seen by recent studies from Saltin et al. (1995), from a physiological perspective, the most important characteristic of these runners is their slightly higher running economy, higher anaerobic threshold, and higher VO_2 max. These physiological advantages most likely are developed from the utilization of running as a part of their childhood lifestyle, before they begin more structured training intended to improve fitness for competitive racing. Thus it may be simply the situation of successful accommodation to stress loading in the formative years, rather than genetic factors, that sets the stage for their success (Noakes, 1992).

Studies by Costill et al. (1973) have demonstrated a strong relationship between ST fibres in the lateralis muscle and distance running performance. The fastest runners were found to possess 70-80% ST fibres while slowest runners had 40-45% ST fibres. It is possible therefore, that the lower blood lactate values observed in the better distance runners might be a function of fibre population and/or the oxidative-glycolytic qualities of the running musculature.

Rice et al. (1988) also demonstrated that the lateral head of the gastrocnemius muscle of 14 elite male long-distance runners showed a range of 50% to 98% ST fibers compared to a range of 50% to 64% among the untrained population. However, because the ST fibers in the elite runners were 29% larger than the FT fibers, on the average 82% of the muscle cross-sectional area was composed of ST muscle. Thus training can selectively increase the size of muscle fibers (Rice et al., 1988).

Jenkins & Quigley (1992) used critical power (CP), that was determine in exhaustive tests at different outputs, to measure a athletes aerobic and anaerobic capacity. CP as proposed by Jenkins & Quigley (1992), is the power represented by the coefficient of linear regression of maximum work on maximum time. As hypothesised, the slope of the CP function increased as a result of continuous training at or near CP without any significant change in the y-

intercept. This confirms now the well-established finding that continuous training results in increases in endurance performance, the percentage of maximal oxygen uptake that can be maintained continuously, and the lactate and ventilatory thresholds, all of which exceed the increase in maximal oxygen uptake.

2.9.4 Overtraining

Competitive endurance athletes typically undergo large increases in training volume and intensity early in their competitive season. Training stress during this period can exceed the athlete's ability to adapt, leading to decrements or stagnation in performance, injury, and complex of symptoms commonly referred as the overtraining syndrome (Pizza et al., 1994). Morgan & Craib (1992) defined overtraining as an imbalance between exercise and recovery, resulting in severe and prolonged fatigues. A problem exists in attempting to steadily increase aerobic and anaerobic capabilities through training. The greater the increase in such performance indicators as VO_2 max, lactate / ventilatory threshold pace, and whatever maximal anaerobic work indicators are used, the greater the subsequent intensity and volume of training required for any further increases. Kuipers & Keizer (1988) have hypothesised that inadequate recovery from a rapid increase in training volume would increase the aerobic demand of running at any given workload due to changes in the stimulation and recruitment of motor units.

Thus, higher volumes of aerobic running bring so little performance benefit that the increasing risks of overuse injury of development of symptoms of overtraining outweigh the potential performance gains (Martin & Coe, 1997). In other words, the risk-benefit ratio becomes excessively high.

Coaches and athletes commonly use an elevation in resting heart rate as an indicator of training stress. However, Pizza et al. (1994) reported that moderate to large increases in training intensity and volume have failed to produce significant elevations in resting heart rate despite changes in other indices of training stress. Therefore, in highly conditioned endurance athletes, monitoring resting heart rate may not be a useful indicator of training stress.

2.9.5 Cross training

In an attempt to minimise the potential detrimental effects of a large increase in training stress via the same training mode (overtraining), endurance athletes are currently implementing cross training regimens into their training. The popular media has endorsed cross training as a means of improving total body fitness and performance in runners (Pizza et al., 1994). However, in the presence of well-developed literature on the principle of specificity of training, the idea that the practice of non-running training alternatives in improving running performance is tenuous and potentially detracts from training time that could be spent more specifically on running needs to be investigated.

Walker et al. (1993) reported that cross training does not exhibit this optimisation of stride length due to the detracting influence of:

- interaction of training methods, specifically running and cycling that utilise the leg musculature in quite a different range of motion; and
- inadequate volume of running specific training relative to their other training necessary to produce an optimal running economy.

However, Mutton et al. (1993) support the use of cross training as an alternative to increasing performance while adding variety to the training program and perhaps reducing the potential for injuries due to overuse or high intensity activity. They showed improvements in running speed, % VO_2 utilised and a decrease in blood lactate values with the cross training group. Cross training could be advantageous to an athlete with smaller increases in training volume or during the "off season" by creating variety in their workouts, training accessory muscles, and by providing a training stimulus when an injury may not permit them to train in their competitive mode. However, in the "high volume phase" of a competitive season, the athlete may benefit more from reducing the volume and increasing the intensity of training, as opposed to, increasing their training volume via the same training mode or by cross training (Pizza et al., 1994).

2.9.6 Middle distance and long distance running

There is no doubt that a high VO_2 max constitutes a kind of membership card for endurance into the world of top level middle and long distance running excellence. But anaerobic aspects

of performance also contribute to the difference between finishing first and second in a race, because they interact with VO_2 max (Martin & Coe, 1997). It is thus important to measure two physiological variables in evaluating long and middle distance runners. One is velocity at anaerobic threshold. Marathon pace is slightly slower than this. The other is velocity at VO_2 max, which is close to 3 000 m pace (Billat & Koralsztein, 1996).

Middle distance runners have lower VO_2 max values than long distance runners but complete at a higher percentage of VO_2 max and incur a greater energy cost for unit distance run. They have the ability to compete at intensities up to 110% of VO_2 max for a duration of as long as 10 or 11 minutes, while long distance runners typically compete at intensities of between 75% and 90% of VO_2 max (Brandon, 1995). Athletes attempt to compete at the fastest sustainable aerobic pace, through the 800-m, where they must cope with additional large anaerobic accumulation over a few minutes time. The larger their VO_2 max, the smaller their total anaerobic contribution will be at any given pace, or the faster they can run before anaerobic effects start to impair performance. But once VO_2 max has been elevated about as high as possible without inordinate additional training volumes, anaerobic development will make the additional difference between being optimally fit and marginally fit (Martin & Coe, 1997).

The energy requirements and metabolic support for optimal run performance are functions of the length of the race and the intensity at which it is completed (Brandon, 1995). The energy requirement for long distance running can, to a great extent, be supplied by the aerobic metabolism, as the rate of energy production necessary to sustain the run velocity is less at longer than at shorter distances (Lacour et al., 1990). Brandon (1995) found that distances requiring up to 10 minutes to complete are quite dependent on both aerobic and anaerobic metabolism.

Many studies have shown that the muscles of outstanding athletes show specific and predictable patterns of muscle fibre content according to the sports in which they excel. Middle distance runners, cyclists and swimmers tent to have equal proportions of both FT and ST fibers. In long distance runners and cross-country skiers, on the other hand, the percentage of ST fibers is high (Noakes, 1992).

Since anaerobic threshold occurs at 60% to 90% of VO_2 max it is important factor in long distance run performance, as distance running is associated more with submaximal efforts. On the other hand, middle distance runners run for shorter time periods at higher velocities and apparently are less sensitive than long distance runners to high lactate levels. Middle distance runners are able to maintain high velocities in races lasting 8 to 11 minutes with relatively high levels of lactic acid (Brandon, 1995).

2.10 ENVIRONMENTAL STRESS

2.10.1 Altitude

The density of air decreases progressively as one ascends above sea level. The barometric pressure at sea level averages 760 mm (Hg), whereas at 3048 m, the barometer reads 510 mm Hg; at an elevation of 5486 m, the pressure of a column of air at the earth's surface is about one-half its pressure at sea level. Ambient air at both sea level and altitude contains 20,93% oxygen, the PO_2 or density of the oxygen molecules in air is reduced in direct proportion to the fall in barometric pressure upon ascending to higher elevations. Ambient PO_2 averages approximately 150 mm Hg at sea level, and 107 mm Hg at 3048 m. At the summit of Mount Everest (8848 m), the alveolar PO_2 is 25 mm Hg. This represents only approximately 30% of the oxygen available in air at sea level. It is this reduction in PO_2 that precipitates the immediate physiologic adjustments to altitude as well as the longer-term process of acclimatization (McArdle et al., 1996).

The relatively poor performances of men and women in middle-distance and distance running and swimming during the 1968 Olympics in Mexico City (altitude 2300 m) have been attributed to the small reduction in oxygen transport at this altitude. Acute exposure to 4300 m causes a 32% reduction in aerobic capacity, compared with sea level values. At extreme altitudes (8848 m), VO_2 max will reduce by approximately 70% (Groves, 1987).

Classification of altitude elevation (Curtis, 1998)

Low	1000 – 1500 m
Moderate	2300 – 3000 m
High	3100 – 4000 m
Extreme	5300 – 8800 m

2.10.2 Acclimatization

The adaptive responses in physiology and metabolism that improve one's tolerance to altitude hypoxia are broadly termed acclimatisation (McArdle et al., 1996). Rapid ascent to high altitudes may be associated with acute mountain sickness, a syndrome including various combinations of headache, anorexia, vomiting, fatigue, insomnia, dyspnoea, ataxia, oliguria and oedema (Berrè et al., 1999). There are many uncertainties about when to have an athlete go to an area of high altitude to compete. Some believe that 2 – 3 weeks before competition provides the best adjustment period, whereas other believe that, for psychological as well as physiological reasons, three days before competition is enough time (Arnheim & Prentice, 1993).

The length of the acclimatisation period depends on the altitude. As a broad guideline, approximately 2 weeks are required to adapt to altitudes of up to 2300 m. Thereafter, for each 610 m increase in altitude, an additional week is necessary to fully adapt up to an altitude of 4600 m. The benefits of acclimatization are lost within 2 –3 weeks after a return to sea level (McArdle et al., 1996).

Bailey & Pate (1991) stated that the economy of heat stress environment should be improved by heat acclimatisation. Acclimatisation, accompanied by exercise training, can increase plasma volume up to 12%. Increased plasma volume assisted in the maintenance of stroke volume and consequently minimises myocardial work in a heat stress environment. Further, an increased plasma volume improves sweating capacity and enables the body to tolerate greater internal heat production.

According to Levine & Stay-Gundersen (1992) 2200-2500 m is approximately the "altitude threshold". It is thus unlikely that physiological adaptations that might improve exercise performance would develop at altitudes below this range. Above 2500 m however, such adaptations are likely to increase linearly with increasing altitude unless acute mountain sickness intervenes or extreme hypoxia (above 5500 m) causes frank deterioration. It would appear that as far as altitude acclimatisation for sea level performance is concerned, within a reasonable range between 2200 m – 4000 m, "higher is better"

2.10.3 Adjustments to altitude

- ***Acid-Base Readjustment***

  Although hyperventilation at altitude favourably increases alveolar PO_2, it has the opposite effect on carbon dioxide. During a prolonged stay at high altitude, the pressure of alveolar carbon dioxide falls to as low as 10 mm Hg. This control of respiratory alkalosis is accomplished slowly, since the kidneys excrete base through renal tubules (McArdle et al., 1991).

- ***Reduced Buffering Capacity***

  A general depression in maximum lactate concentration is apparent during maximal exercise at altitudes above 4000 m. This reduction in exercise blood lactate levels during chronic high-altitude hypoxia is not accompanied by an increase in VO_2 max or by an increase in oxygen delivery to active tissues after acclimatization (McArdle et al., 1991). It seems that lactate threshold is better maintained in males then females with increasing altitude. Comparing the same active, untrained altitude residers from above, increasing hypoxia detrimentally influenced lactate kinetics in females. This difference was independent of the reductions in VO_2 max (Seiler, 1997).

- ***Hematological Changes***

  The most important adaptation for the endurance athlete is an increase in the number of red blood cells, which are produced in response to greater release of the hormone erythropoietin (EPO) by the kidneys (Baker & Hopkins, 1998). An increase in the blood's oxygen-carrying capacity is the most important longer-term adaptation to altitude. The two factors that are responsible are the initial decrease in plasma volume and the increase in the erythrocytes and haemoglobin. After one week at an altitude of 2300 m, the plasma volume decreased by 8%, haematocrit increase by 4% and haemoglobin increased by 10%. During the 1973 Mount Everest Expedition, a 40% increase in haemoglobin concentration and a 66% increase in haematocrit were noted for climbers acclimatised at 6500 m. This probably approaches the upper limit of a beneficial concentration of red blood cells. Any further erythrocyte packing would increase the blood's viscosity and probably restrict blood flow and oxygen diffusion to the tissues. Athletes with low iron stores may not respond to the acclimatisation process as well as individuals who arrive at altitude with

iron reserves adequate to sustain an increase in red blood cell production (McArdle et al., 1996).

- ***Cellular Adaptations***

  Increasing red cell mass is recognised as a potent means of improving athletic performance (Buick et al., 1980). Numerous studies have shown that red blood cell production is stimulated during exposure to very high altitude - 4 000 m or higher (Ashenden et al., 1999). An increase in the concentration of erythropoietin (EPO), which is one of the hormones that stimulate red blood cell production, has been documented in athletes living at simulated altitudes of between 2500 and 3000 m and training at near sea level (Laitinen et al., 1995). Ashenden et al. (1999) found no increase in the reticulocyte production of elite female cyclists following 12 nights of sleeping at a simulated altitude of 2650 m compared to control subjects undertaking the same training loads but sleeping near sea-level conditions. The reticulocyte parameters are known to reflect an increase in red blood cell production.

  Muscle biopsies from humans living at high altitude indicate an increase in myoglobin by as much as 16% after acclimatisation. Such adaptations increase the "storage" of oxygen in specific muscles and facilitate intracellular oxygen release and utilisation at low PO_2. Adaptations that depend on oxygen delivery to peripheral tissues increase linearly with oxyhemoglobin desaturation. Thus, red cell mass does not increase until PaO_2 decreases below approximately 65mmHg (Levine & Stay-Gundersen, 1992).

- ***Changes in Body Mass and Composition***

  A substantial increase in heart rate when the athlete stands up may be an early warning of the onset of overtraining and a sign that the athlete should return to sea level (Rusko, 1996). A related sign of living to high may be wasting of muscles. Athletes living at 3000 m or higher would be wise to include some form of monitoring for overtraining: feelings of fatigue during training, reduction in performance of criterion tests, and possibly changes in heart rate, body mass or muscle mass (MacDougal et al., 1991). Long-term exposure to high altitude produces a significant loss in lean body mass and body fat. The higher basal metabolic rate also plays a role in weight loss. Severe high altitude is thus catabolic. In

fact the decrease in muscle fiber size may be directly responsible for increasing capillary density and reducing effective diffusion distance to muscle mitochondria (Green et al., 1989).

Baker & Hopkins (1998) named various methods to get altitude exposure:

- Use of a mountain and a valley;
- stay high and train hard with oxygen;
- live in a nitrogen house;
- rest and sleep in a nitrogen tent;
- breathe through a nitrogen mask intermittently;
- live in a large barometric chamber;
- rest and sleep in a personal barometric chamber; and
- use erythropoietin or blood doping.

However the ethics of altitude exposure should also be taken in consideration. Altitude chambers, nitrogen houses and nitrogen tents would be dangerous if the simulated altitude was high enough and long enough to raise the thickness of blood to an unsafe level (Baker & Hopkins, 1998).

2.10.4 Altitude training and performance

From a theoretical viewpoint, training at altitude could produce more rapid and even greater physiological changes than could training at sea level only. The reason is that altitude hypoxia is a stress that produces physiological changes similar to those caused by physical training. To a certain extent, this research has been supported experimentally. (Roskamm et al., 1969; Banister & Woo, 1978), but most of the research indicates that enhancement in performance is more apparent in unconditioned non-athletic individuals.

Although some adaptations during acclimatisation to altitude should enhance aerobic capacity and endurance performance upon return to sea level, research results do not support this effect (Banister & Woo, 1978). This is probably the result of the altitude-related decrease in both maximum heartrate and stroke volume. For the highly trained athlete, the training intensity required for maintenance of peak performances cannot be achieved at altitude. In contrast,

probably the most important adaptation that might improve sea level performance is an increase in hemoglobin and hematocrit which improves the oxygen carrying capacity of the blood (Levine & Stay-Gundersen, 1992).

Despite the fact that there is very little scientific evidence showing that altitude training improves sea-level performance, the prevailing opinion among coaches, athletes and many uninformed sports administrators is that it works for all athletes, irrespective of their event or ability. Special altitude training centres are usually located in exotic mountainous regions and the athletes who attend these camps do not have too many distractions. They are there to train. Life at altitude consists of running, eating sleeping and more of the same. Athletes are highly motivated and usually working towards a common competitive goal, an environment highly conductive to quality living and training. John Hawley stated in the Runners World magazine that it may will be that the isolation of these altitude camps is the main reason that many athletes improve their performance when they return to sea level. Therefore Hawley reported that isolated sea-level training camps would probably have just the same positive effect whether the camp is at altitude or not (Hawley, 1997). However Baker & Hopkins (1998) found that living and training at altitude is less effective than living at altitude and training near sea level, because the lack of oxygen at altitude results in detraining through reduction in intensity of training

Trained runners have revealed lower aerobic demands during treadmill running at altitude compared with sea-level treadmill running, despite a greater ventilatory effort at altitude (Morgan & Craib, 1992). Levine & Stay-Gundersen (1992) study showed that athletes train faster and at greater oxygen uptakes near sea level than at altitude. A lowered overall work of breathing due to the reduced air density at altitude an environmental differences in anaerobic energy contribution may partly account for enhance treadmill running economy at altitude. Sea level aerobic demands measured in trained runners during track running are also significantly greater when compared with track running at altitude. A potential explanation for this finding is that a decreased energy demand is required to move through the less dense air of altitude (Morgan & Craib, 1992). Kayser et al. (1993) stated that high-altitude natives are also characterised by low levels of lactic acid at exhaustion, although the underlying mechanism that might be different from that for acclimatised lowlanders. Levine & Stay-

Gundersen (1992) stated that the substrate utilisation also improved by increasing mobilisation of free fatty acids and increasing dependence on blood glucose thus sparing muscle glycogen. This is manifested by decreased accumulation of metabolites such as lactate or ammonia during submaximal exercise.

Muscle pH after exhaustive exercise is higher at altitude than at sea level. As a consequence, decreases intra- and extracellular buffer capacities do not appear to be responsible for the decrease in low lactate values at maximum intensity. It is important however, that altitude training is not a substitute for a focused, well designed training program and must be undertaken with specific attention to adequate nutrition, particularly ensuring adequate iron stores (Levine & Stay-Gundersen, 1992). Stay-Gundersen et al. (1992) demonstrated that runners, who begin a period of altitude training with low iron stores, do not increase their red cell volume after a month living and training above 2500 m.

2.10.4.1 Training intensities at altitude

During high intensity interval workouts, absolute workloads, peak heart rate, and blood lactate concentrations were lower during training at altitude than near sea level. Thus interval training at high altitudes is unlikely to be as effective as interval training at lower altitudes (Levine & Stay-Gundersen, 1992).

It seems that lactate threshold is better maintained in males than females with increasing altitude (Seiler, 1997). Base training near sea level will allow relatively normal training intensity and may prevent the loss of plasma volume that often accompanies altitude acclimatisation (Withey et al., 1983). In contrast, base training at altitude, as long as it occurs at a low enough altitude to maintain similar running speeds and absolute workloads as at sea level, may facilitate an increase in mitochondrial aerobic enzyme activities and maximise peripheral oxygen utilisation.

The shortage of oxygen at higher altitudes appears to be a stressor that these athletes cannot adapt to. The result is inability to sustain previous training may loads and gradual loss of fitness. A substantial increase in heart rate when the athlete stands up may be an early warning of the onset of such overtraining and a sign that the athlete should return to sea level. Thus it would be wise to include some form of monitoring for overtraining: feelings of fatigue

during training, reduction in performance of criterion tests, and possibly changes in heart rate, body mass, or more sophisticated measures of muscle mass (Baker & Hopkins, 1998).

2.10.5 Alternative approaches

The optimal approach to altitude training would be to acclimatise to altitude, but train as close to sea level as possible thereby maximising running speed and maintaining aerobic fitness (Levine & Stay-Gundersen, 1992). Especially high intensity and interval workouts should be conducted as close to sea level as possible, preferable below 1500 m, to maximise running speed and training intensity. However, Ashenden et al. (1999) found no increase in the erythropoietic stimulus when the athletes participate in a "live high, train low" experiment.

2.11 USE OF HEARTRATE MONITORS

Yamaji et al. (1992) found excellent correlation between heart rate monitors and heart rate readings as computed form the ECG using chest electrodes. These findings support thus the accuracy of the use of pulse monitors. Today, heartrate monitors are widely used by amateur and professional athletes in many different sports in an effort to guide training, optimise race performance and monitor recovery. Heartrate monitoring allows a more precise control of intensity than would be possible from subjective monitoring of effort alone. During running competitions, it has become common practice for athletes to guide pace by setting relatively narrow heartrate limits (10 bpm) on their heartrate monitors. (Toole et al., 1998). They attempt to stay within these limits by continually adjusting pace throughout the course of the race. Thus, heartrate monitoring provides the key to regulate the quantity and intensity of workouts and racing performance by providing accurate and immediate biofeedback data.

2.11.1 Factors that affect heart rate response

One of the major problems is determining the degree of similarity between the laboratory test used to establish the heartrate – oxygen uptake line and the specific activity to which it is applied. Comrades Marathon star Nick Bester said after the 1997 Comrades that in the last few kilometres he was unable to keep his heart rate up at the remarkable 170 beats per minute he had sustained the whole race, "because my legs were too tired, I just couldn't move any faster" (Comrades Marathon update, 1997).

Many factors have an effect on a person's heartrate response and therefore need to be considered when training. The increased heart rate found in warm conditions is probably the result of increased body temperature rather than any tendency for the subjects to push themselves harder in warmer weather. Increased fluid loss through sweating might also contribute to the increased heart rate in warmer conditions (Robinson et al., 1991; Seiler, 1997). Heartrate increases from 5-10 beats per minute at any speed when the ambient temperature increases to about 25°C. Heartrate is the lowest in the morning and increases by about 10 beats per minute by late afternoon, thus time of the day is also a factor to consider when training. Dehydration affects the heartrate response by an increase in heartrate (about 7 beats per minute) at any speed for every litre of fluid lost while training. Heart rate decreases as a result of an improvement in an athlete's fitness level (Robinson et al., 1991). Daily heat exposure of ten days is superior to intermittent exposure for inducing acclimatisation (Seiler, 1997)

Yamaji et al. (1992) report a direct linkage between central and cardiovascular fatigue, and respiratory and peripheral sensations. Robinson et al. (1991) named other factors that produced significant correlations with heart rates were distance, duration, willingness to train, number of companions, temperature, terrain and wind. They stated that the higher heart rates recorded in steep terrain and wind conditions may have been the result of subjects attempting to maintain a constant pace during periods of increased work load. Also the effect of running with companions may reflect a tendency of elite athletes to compete against each other even on training runs.

Why use a heartrate monitor:

- to give a precise measurement of the exercise intensity
- to individualise the training program
- to monitor progress, and witnessing improvement is motivation
- to introduce objective observation

2.11.2 Different heart rate formulas for exercise intensity prescription

Several formulas are routinely used to determine an individual's exercise intensity and are based upon maximal heart rate, either measured directly during a graded exerciser test or

estimated for age (220-age). The Karvonen method, which is the most widely accepted, the resting heart rate is a second variable used in the calculation of the targed heart rate intensity (DiCarlo et al., 1991; Foss, & Keteyian, 1998). By doing an incremental maximum oxygen consumption test, the lactate and ventilatory threshold can be used to determine target exercise zones.

Conconi developed a method by which it was possible to establish the anaerobic threshold without measuring lactate. Conconi made use of the existing correlation between activity intensity and pulse rate. He found that at very intensive activity pulse rate and intensity no run parallel. The straight line at the onset deflects at high intensities. In other words, the intensity may be increased but the increase of pulse rate lags at a certain point. This point is the PR point. The workout intensity corresponding to this point is the maximum activity that can be done with aerobic energy supply (Janssen, 1987).

For each person, heart rate and oxygen uptake tends to be linearly related throughout a wide rage of aerobic exercises (McArdle et al., 1996). If this precise relationship is known, the exercise heart rate can be used to estimate oxygen uptake during similar forms of physical activity. This approach has been used when the oxygen uptake could not be measured during actual activity. Each athlete's heart rate increased linearly, with an increase in oxygen uptake being accompanied by a proportionate increase in heart rate. However, even though both heart rate and oxygen uptake lines are linear, the same heart rate does not correspond to the same level of oxygen uptake. The slope or rate of change of the line differs considerably from person to person (McArdle et al., 1996).

Heart rates generally rise quickly to a steady-state heart rate that is maintained early in exercise (i.e. up to approximately 30 minutes), followed by gradually increasing heart rates if exercise is continued for prolonged periods (Toole et al., 1998). Although specific reasons for slowing pace during prolonged exercise may vary among individual athletes, potential contributing reasons include substrate depletion, altered muscle efficiency, fluid and electrolyte imbalances, thermoregulatory problems, cardiac fatigue and psychological factors (Toole et al., 1998). The ability, then, to sustain high heart rates during prolonged exercise could be hypothesised to be necessary for fast paces and fast finish times.

2.11.3 Relationship between oxygen consumption (VO_2) and heart rate

Heart rate is recognised as the most useful method for determining training intensity, because it displays a fairly linear response to increasing work loads and normally reaches maximum values at the same exercise intensity that produces maximum aerobic power (Astrand & Rodahl, 1986). Thus, by simultaneously measuring maximum aerobic power and exercise heart rate in the laboratory, the scientist can estimate the relative intensity of exercise in the field on the basis of heart rate alone and indirectly determine maximum aerobic power (MacDougall et al., 1991).

Common tests to predict VO_2 max use exercise heart rate during a standardised regimen of submaximal exercise performed either on a bicycle ergometer, a treadmill or a step test. Using the linear relationship between oxygen consumption and heartrate, VO_2 max can be determined. Although each person's heartrate-VO_2 line tends to be linear, the slope of the individual lines can differ considerably (McArdle et al., 1996). Also heart rate reflects the relative difficulty or intensity of the exercise rather than the absolute work being performed. This explained the fact that the lower body usually represent a larger proportion to the total body mass and, in the case of equal fitness in upper and lower body musculature, is capable of achieving a higher work rate before the aerobic production of ATP becomes limiting (MacDougall et al., 1991).

Londeree (1995) provides the selected values for percent VO_2 max and corresponding percentages of heart rate max:

Percent HR max	Percent VO_2 max
50	28
60	40
70	58
80	70
90	83
100	100

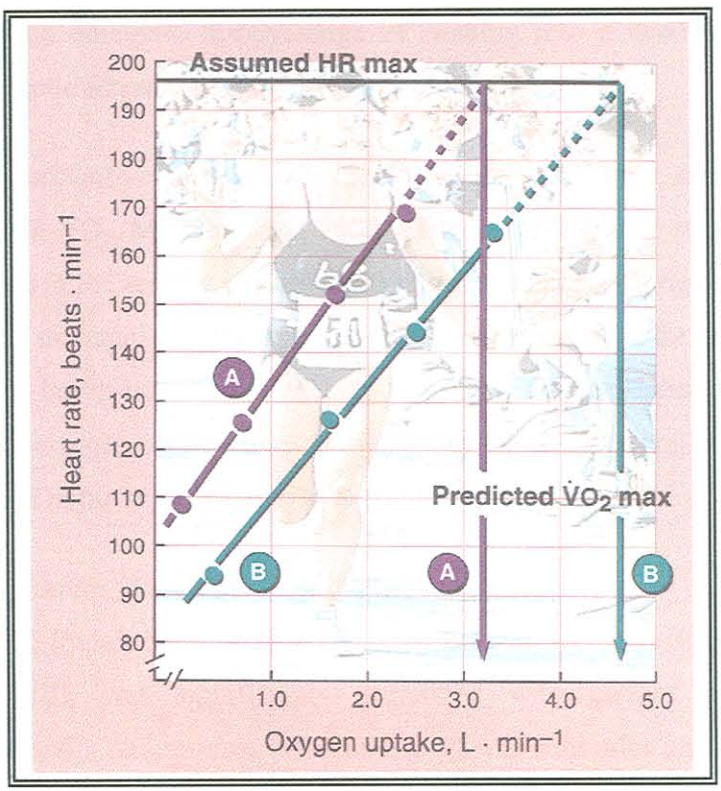


Figure 8: Heart rate in relation to oxygen uptake during upright exercise in endurance athletes (brown line) and sedentary college students before (blue line) and after (green line) 55 days of aerobic training (McArdle et al., 1996).

2.12 TREADMILL VS OVERGROUND CONDITIONS

Due to the difficulty of obtaining metabolic date in field situations, measurements have typically been made indoors on treadmills. Since air and wind resistance are not factors during indoor testing, caution must be used in applying treadmill date to overground conditions (Morgan et al., 1989).

While most studies have indicated that overground running is more costly than indoor treadmill running, some investigators have reported no significant differences between the two conditions. Morgan et al. (1989) estimated that 2% to 8% of the total energy demands of long- and middle-distance track running is expended overcoming air resistance. Results from Daniels (1985) indicated that at fast running speeds, the cost of overground running in calm air was 7.1% greater compared to indoor treadmill running. It was observed that as wind

velocity increased, the energetic disadvantage of running into a headwind was relatively greater than the energetic advantage gained by running with a tailwind.

Interestingly, when running velocity and tailwind velocity were equal, overground VO_2 was equivalent to treadmill VO_2 (Morgan et al., 1985). Bassett (1985) also stated that there is no measurable difference in the aerobic requirements of submaximal running on the treadmill and track, or between the maximal oxygen uptakes measured during both forms of exercise. It is still possible that at faster running speeds the impact of air resistance on a calm day is considerable and the oxygen cost of track running may be greater compared to "stationary" running on a treadmill at the same speed (Bassett, 1985).

2.13 Psychological factors influence running performance

Bailey & Pate (1991) found that a more positive mood state, as measured by the Profile of Mood States (POMS), was significantly correlated with greater running economy. Furthermore, correlation's determinate between the POMS subscales and running economy indicated that tension held the strongest association. Mood state during exercise and running economy may be similarly affected by changes in heartrate, ventilation, running mechanics and substrate utilisation.

CHAPTER 3

PROCEDURES AND METHODS

3.1 INTRODUCTION

Laboratory tests are widely used by athletes of many different endurance sports in an effort to guide training, optimise race performance and to monitor recovery. The science of sport is particularly exciting and challenging. Measuring anaerobic and aerobic fitness helps not only to characterise an individual's ability to perform a certain task but it can also be used to quantify the effect of a change in training regimen on performance. Testing helps to describe the characteristics of the athlete but can also help to access the effects of exposure to altitude and pollution and the effect of ergogenic aids. Determining one's cardiovascular fitness also has both general health and clinical applications. The extent to which these and other factors are "trainable" as opposed to genetically determined is a topic of considerable debate (Bouchard et al., 1992).

3.2 TESTING PROCEDURES

3.2.1 Subjects

Five male marathon athletes volunteered to take part in the different tests and were informed in detail about the nature of the experiment and about possible risks. All the subjects were training for the 1998 Comrades Marathon and all of them had run the Comrades before. They had been following a training programme for a minimum of 4 years and were experienced treadmill runners. All the subjects were non-smokers and were free of any disease. The day before each test, no intensive training was allowed. Their regular training regimens included running of at least 80 km weekly, with workouts of moderate to high intensity.

3.2.2 Experimental environment

Laboratory tests were administered in an air-conditioned laboratory at a temperature of 20°C, and a relative humidity of 45%-55%. Analysers were calibrated before each subject's test by

using gases of known concentration. The same biokineticist was employed in subsequent testing.

3.3 TESTING

3.3.1 Anthropometrical measurements

The evaluation of body composition permits quantification of the major structural components of the body – muscle, bone and fat (McArdle et al., 1996).

3.3.1.1 Anthropometry equipment

Anthropometry tape: A flexible steel tape calibrated in centimetres with millimetre gradations. Weighing scales: A medical balance scale calibrated to the nearest 100 g. Detecto standing scale was used to measure body weight.

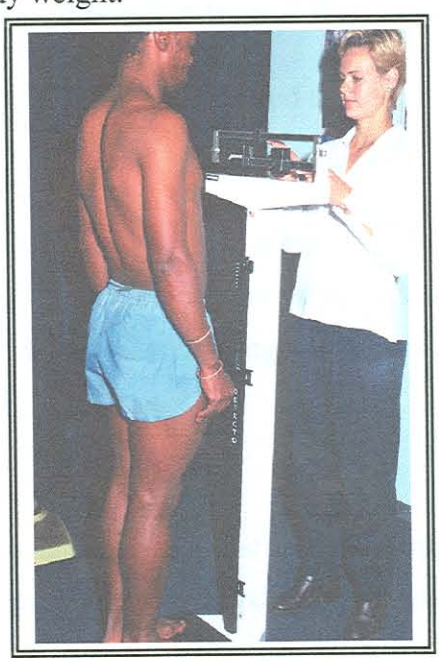


Figure 10: Detecto standing scale to determine body mass

Anthropometer: Anthropometer with foot plate, calibrated in centimetres with millimetre gradations.

Skinfold caliper: Harpenden caliper calibrated to 50mm in 0,2mm divisions, accurately interpolated to the nearest 0,1 mm.

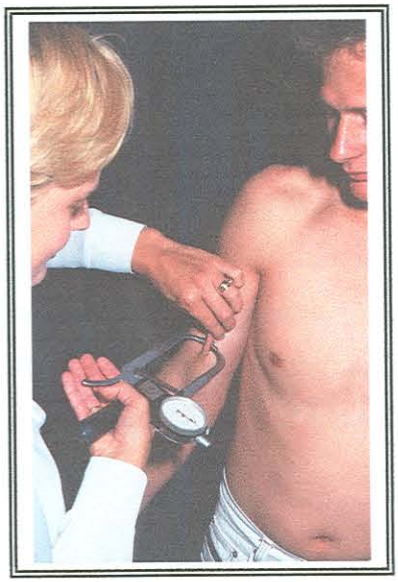


Figure 9: Measuring body composition using a Harpenden skinfold caliper

Wide-spread calipers: Widespread calliper calibrated to the nearest 0,2 mm.

3.3.1.2 Anatomical landmarks

Landmarks are identifiable skeletal points, which generally lie close to the body's surface and are the "markers" which identify the exact location of the measurement site. All the variables unless stated otherwise, were measured according to the procedures of the anthropometric manual of Lohman et al. (1988).

a. Skinfolds

1. *Triceps:* The fold is vertical and parallel to the line of the upper arm, on the posterior-mid-acromiale–radiale line. Taken on the most posterior surface of the triceps muscle. Arm relaxed with the shoulder joint slightly externally rotated and elbow extended by the side of the body.
2. *Subscapular:* Inferior angle of the scapula along a line running laterally and obliquely downwards from the subscapular landmark at an angle (approximately 45°) as determined by the natural fold lines of the skin.
3. *Biceps:* Mid-acromiale-radiale line with the fold running vertically parallel to the axis of the upper arm. Arm relaxed with the shoulder joint slightly externally rotated and elbow extended. Located on the most anterior aspect of the biceps.

4. *Iliac crest*: Superior to the iliocristale on the ilio-axilla line. The fold runs slightly downwards toward the medial aspect of the body.
5. *Abdominal:* Vertical fold 5 cm in the midline of the belly of the rectus abdominus from the right-hand side of the omphalion (midpoint of the navel).
6. *Front thigh*: Parallel to the long axis of the femur at the mid-point of the distance between the inguinal fold and the superior border of the patella (when the knee is bent).
7. *Medial calf*: Vertical fold on the medial aspect of the calf at a level where it has maximal circumference. Subject seated and knee 90° flexed and calf relaxed.

b. Girths

1. **Forearm:** Measurement is taken at the maximum girth of the forearm with the subject holding the palm up. Usually occurs just distal to the elbow.
2. *Wrist:* Distal to the styloid processes. It is the minimal girth in this region.
3. *Chest:* Girth is taken at the level of the mesosternal, with the tape around the chest in a near horizontal plane. Measurement is taken at the end of normal expiration.
4. *Waist:* Measurement is taken at the level of the narrowest point between the lower costal border and the iliac crest, at the end of normal expiration with the arms relaxed on the sides.
5. *Hip:* This is taken at the level of the greatest posterior protuberance of the buttocks which usually corresponds anteriorly to about the level of the symphysis pubis.
6. *Thigh:* The girth of the thigh is taken 1 cm below the level of the gluteal fold, perpendicular to the long axis of the thigh.
7. *Calf:* With the weight equally distributed on both feet. The maximal girth of the calf is taken.
8. *Ankle:* The minimum girth of the ankle is taken at the narrowest point superior to the sphyrion tibiale.
9. *Upper arm*: Maximum girth of the biceps muscle.

c. Breadths

1. *Biacromial:* The distance is measured between the most lateral points on the acromion processes.
2. *Bi-iliocristal*: Distance measured is between the most lateral points on the iliac tubercles.

3. *Transverse chest:* The distance is measured between the most lateral aspect of the thorax when the superior aspect of the calliper scale is positioned at the level of the mesosternal and the blades are positioned at an angle of 30° downward from the horizontal. The measurement is taken at the end of tidal expiration.
4. *Anterior-posterior chest*: The distance is measured between the recurved branches of the wide-spread calliper when it is positioned at the level of the mesosternal. The rear branch of the caliper should be positioned on the spinous process of the vertebra at the level of the mesosternal.
5. *Bi-epicondylar humerus*: Distance is measured between the medial and lateral epicondyles of the humerus when the arm is raised anteriorly to the horizontal and the forearm is flexed at right angles to the upper arm. Because the medial epicondyle is lower than the lateral epicondyle, the meas*ured distance is oblique.*
6. *Bi*-epicondylar femur: Distance is measured between the medial and the lateral epicondyles of the femur when the subject is seated and the leg flexed at the knee form a right angle with the thigh.

3.3.1.3 Calculations

The Heath Carter method was used to determine the athlete's body composition. There are two available procedures for calculating the anthropometric somatotype. The first is to enter measurements into the Heath-Carter rating form, and the second is to enter the measurements into equations derived from the rating form (Foss & Keteyian, 1998). The following measurements, all taken on the right side of the body, are required to complete the somatotype (Fox et al., 1993). The anthropometry measurements include height, body mass, four skinfold measurement (triceps, subscapular, supra-illiac, medial calf), two bone widths (humerus, femur), two bone girths (upper arm girth, calf) (Pate et al., 1991).

The Drinkwater and Ross method includes the following measurements: height, body mass, seven skinfolds (biceps, triceps, subscapular, supra-illiac, abdominal, frontal thigh, and medial calf), six bone widths (biacromial, bi-iliocristal, transverse chest, anterior-posterior chest, bi-epicondylar humerus, bi-epicondylar femur) and nine girths (forearm, wrist, chest, waist, hip, thigh, calf, ankle, upper arm). For research purposes, the investigator has had considerable

experience and was consistent in duplication values for the same subject on consecutive days (McDougal et al., 1991).

Data were entered in the labtest's computer program to determined the following:

Absolute fat mass (kg) and fat percentage (%)

$$\frac{(\text{sum of skinfolds}/6 \times 3.25) + 12.13}{(170.18/S)^3} = AFM$$

AFM	=	absolute fat mass (kg).
Sum of skinfolds	=	sum of triceps, subscapular, supra-illiac, abdominal, thigh, calf.
S	=	measured height (cm).
3.25	=	mean standard deviation for fat component.
12.13	=	mean phantom value for fat component

$$RFM = (AFM/MBM) \times 100$$

RFM	=	relative fat mass (%)
AFM	=	absolute fat mass (kg)
MBM	=	measured body mass (kg)

Absolute muscle mass (kg) and percentage (%)

$$\frac{(\text{Sum of CG} / 5 \times 2.99) + 25.55}{(170.18 / S)^3} = AMM$$

AMM	=	absolute muscle mass (kg)
Sum of CG	=	sum of corrected arm, chest, thigh and calf girths and the uncorrected forearm girth
S	=	measured height (cm)
1.99	=	mean phantom standard deviation for muscle component
25.55	=	mean phantom value for muscle component

$RMM = (AMM / MBM) \times 100$

RMM	=	relative muscle mass (%)
AMM	=	absolute muscle mass (kg)
MBM	=	measured body mass (kg)

Lean body mass (kg)

Lean body mass (g) $= S(0.0553CTG^2 + 0.0987FG^2 + 0.0331CCG^2) - 2445$

S = stature

CTG = corrected mid thigh girth

FG = forearm girth

CCG = corrected calf girth

2. Somatotype (endomorph, ecotomorph and mesomorph)

Endomorphy:

ENDO $= [-0.7182 + 0.1451\ (x)] - [0.00068\ (x^2)] + [0.0000014\ (x^3)]$

x = sum of triceps, subscapular and supra iliac skinfolds

Mesomorphy:

Meso $= [(0.858 \times H) + (0.601 \times F) + (0.188 \times AG) + (0.161 \times CG)] - (S \times 0.131) + 4.50$

H = largest humerus diameter (cm)

F = largest femur diameter (cm)

AG = corrected arm girth (cm)

CG = corrected calf girth (cm)

S = stature (cm)

Ectomorphy:

ECTO = (SMR x 0.732)- 28.38

SMR = stature (cm)/mass$^{0.333}$(kg)

Body mass index

(kg/m^2)

Ideal body mass (kg)

Ideal body mass (kg) = Fat free mass/ (100 – TF%)

TF% = target % body fat

3.3.2 Lung-function testing

Lung-function tests were performed on the Schiller CS 100 - Ergo-spirometry-module; Flow-sensor SP-160. All the subjects were familiar with the lung-function test procedures. Two trials were given to each subject, to allow a degree of learning. The results were also visible to the subjects, thus encouraging them to use maximum effort. Subjects perform the test standing in front of the recorder and wearing a noseclip. The subject exhales as rapidly and completely as possible, following a maximal inspiration. Forced vital capacity (FVC) was measured in litres. The tidal volume was then divided by the FVC to calculate the percentage of lung filling during the VO_2 max test.

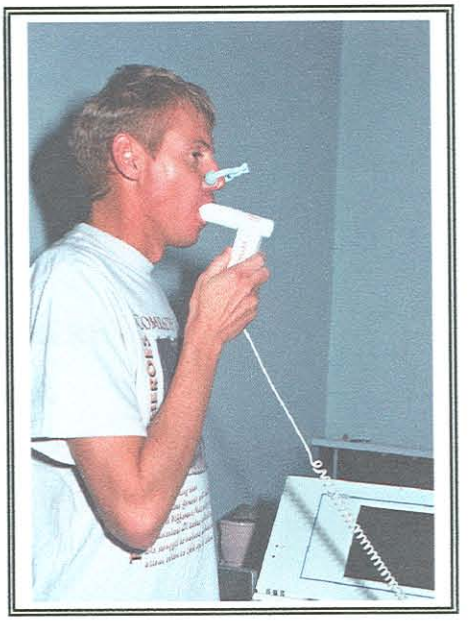


Figure 11 Lung-function test performed on the Schiller CS 100

3.3.3 Maximal oxygen uptake (VO_2 max)

The VO_2 max was determined by use of an inclined electrically driven treadmill protocol. Schiller CS 100 gas analyser, with ECG-Module, flow-sensor SP-160 and a Quintin treadmill

(model 24-72) were used to determine the endurance fitness of the marathon athlete. Before each test analysers were calibrated with gas mixtures of known concentrations.

After a five-minute warm-up period at 10 km/h, the subjects started the test running at 12 km/h on a constant slope of 2%. The speed was increased every 3 minutes by 2 km/h until 16 km/h was reached. The treadmill speed was then increased by 1 km/h every 2 minutes until exhaustion. The subjects received strong verbal encouragement to continue as long as possible. The subjects constantly wore a mask that covered the mouth and nose during the entire phase of gas collection. Gas samples was recorded every 10 seconds.

All the athletes reach a VO_2 max according to the following criteria:

1. heart rate of at least 95% of maximal heart rate of the subject;
2. respiratory exchange ratio at least 1.1;
3. severe exhaustion;
4. plateau or decreasing of the VO_2 max; and
5. lactate values of at least 8 mmol/L (MacDougal et al., 1991).

The physiological parameters measured were:

- oxygen consumption (VO_2 max);
- heartrate (HR);
- respiratory exchange ratio (RER);
- oxygen equivalent (VE/VO_2);
- carbon dioxide equivalent (VE/VCO_2);
- carbon dioxide production (VCO_2);
- respiration rate (RR);
- tidal volume (VT);
- lactate threshold;
- ventilatory threshold;
- oxygen pulse (ml O_2/HT); and
- metabolic equivalents (METS).

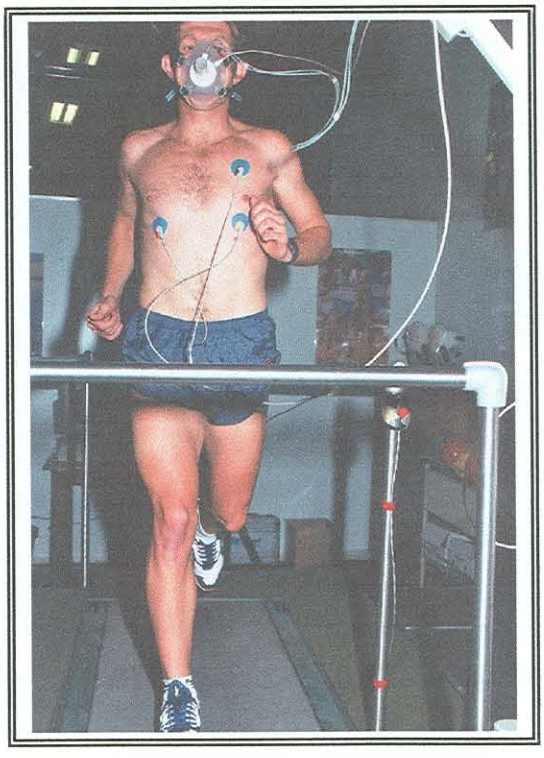


Figure12: VO_2 max test performed on the Quintin treadmill (model 24-72), using the Schiller CS 100-gas analyser, with ECG-Module and flow-sensor SP-160.

3.3.4 Measuring blood lactate

Lactate was determined in the same laboratory setting on the treadmill on a fixed 2% inclination simultaneously with the VO_2 max test. Blood lactate tests were performed with an Accurex BM lactate meter (manufactured by Boehringer and Mannheim), requiring a lancet puncture of a fingertip to obtain a small sample of peripheral blood. Blood lactate samples were collected during the test at the end of each workload. Measuring time is only 60 seconds. Lactate was plotted against heartrate to determine the onset of lactate accumulation (OBLA 4 mmol/L).

3.3.5 Distances and running times

Each subject's best running times for the 10, 21, 42, 50, 56 and 90 km had been recorded during the preceding year. Training distances were also recorded during the testing period (October 1997 to May 1998) as a total of kilometres done per month.

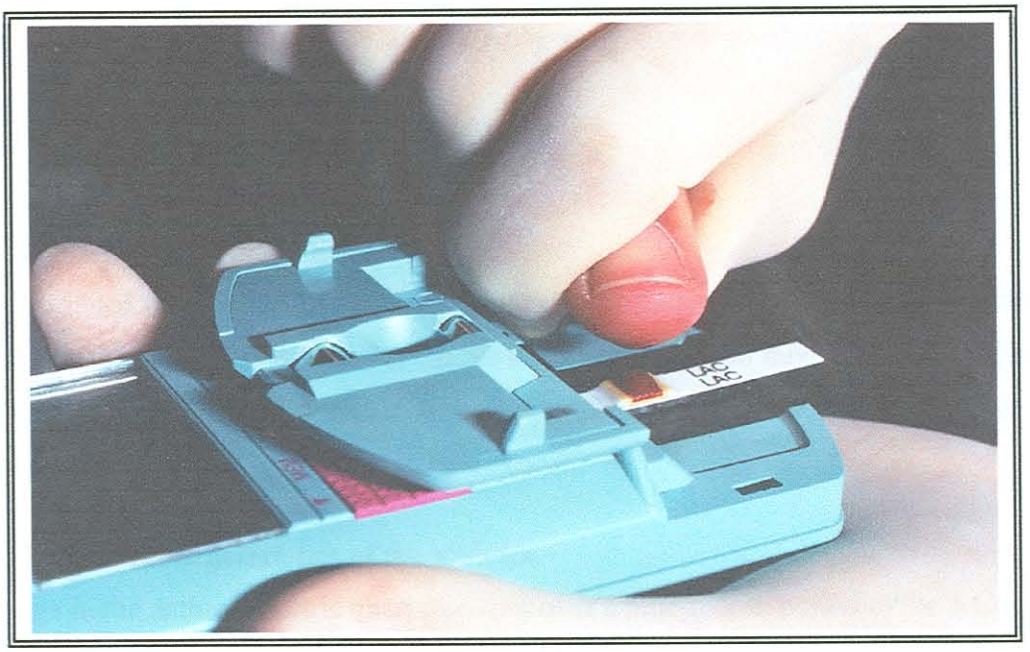


Figure 13: Blood lactate test performed with an Accurex BM lactate meter (manufactured by Boehringer and Mannheim).

3.3.6 Heartrate response during the Comrades Marathon

Each subject ran the Comrades Marathon with a Polar Vantage NV to determine race intensity. Data was there after analysed with the polar interface plus with training advisor software for Windows. Heartrate response was measured every 5 seconds during the race. The software of the interface program allows the user to calculate heartrate response as a percentage below the lactate threshold, percentage above the threshold, average heartrate and maximal heartrate. The athletes attempted to stay in the 2 to 3 mmol/L training zone by adjusting pace throughout the course of the Comrades Marathon.

3.3.7 Statistical analysis

Statistical calculations were done with time-related information data over a period of eight months. Progression analysis showed the percentage improvement in this time period. The test sample consists of 5 marathon athletes. Because of the small sample, the Spearman's rank correlation coefficient tests were used. It has to be stated that the results obtained from the test cannot be used to make conclusions about the population (all marathon athletes), because this is not a random sample.

CHAPTER 4

RESULTS AND DISCUSSION

4.1 GENERAL RESULTS:

It has to be stated that the results obtained from the tests cannot be used to make conclusions about all marathon athletes because the sample is not big enough or random enough. Therefore it is a **case study** of five Comrades Marathon athletes who were training for the 1998 Comrades Marathon.

4.1.1 Physical characteristics of the athletes

The change in the anthropometric characteristics over a nine-month period of the 5 Comrades Marathon athletes is summarized in Table 1. The results indicate an increase in muscle percentage (2.45%), and a decrease in lean body mass (-3.37%), BMI (-2.49%), body mass (-2.53%) and fat percentage (-4.09%).

Below (Table 1) is a summary of the percentage changes that took place from October 1997 to May 1998. Individual values for each athlete are shown.

TABLE 1:Percentage change in the anthropometric parameters over nine-months

Athlete #	% change in mass	% change in % muscle	% change in % fat	% change in BMI	% change in lean body mass (LBM)
#1	0.07 %	2.99 %	7.10 %	0.00 %	-2.02 %
#2	-3.15 %	2.45 %	-13.84 %	-3.15 %	-1.12 %
#3	-4.07 %	2.60 %	-4.19 %	-4.22 %	-2.16 %
#4	0.57 %	1.07 %	8.24 %	0.19 %	-9.63 %
#5	-6.06 %	3.12 %	-17.78 %	-5.99 %	-1.93 %
Average	-2.53 %	2.45%	-4.09 %	-2.49 %	-3.37 %

4.1.2 Changes in anthropometric parameters over the 5 test sessions

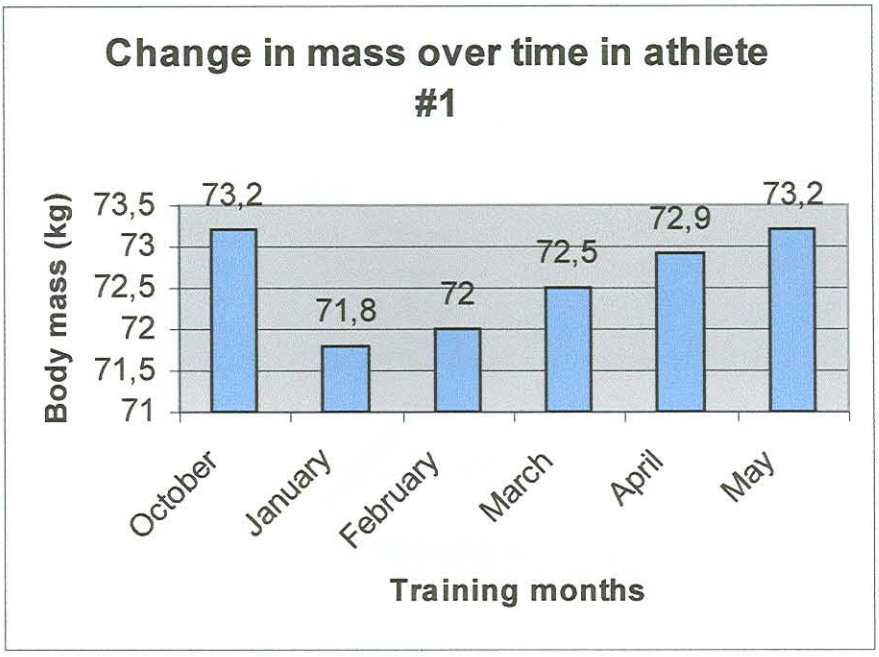
Tests were done 5 times before the Comrades Marathon in October, January, March, April and May. Changes in total body mass (kg), fat percentage (%) and lean body mass (kg) were measured during the training months.

4.1.2.1 Total body mass

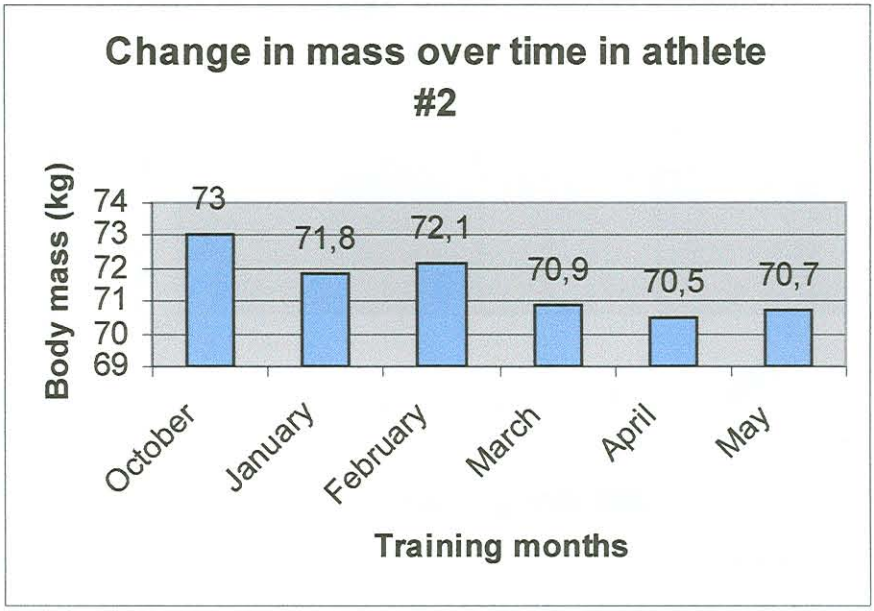
Several studies reported a high relationship between VO_2 max, body composition and endurance performance (Noakes, 1990; Brandon et al., 1995; Brisswalter et al., 1996). Berg et al. (1998) found a strong linear relationship between VO_2 max (L/min) and gross body mass for ectomorphs and mesomorphs, while these two variables are unrelated to endomorphs. VO_2 max (ml/kg/min) is dependent on gross body mass and increases with increasing body mass for ectomorphs and mesomorphs. The opposite is true of endomorphs in which group a strong linear decrease in VO_2 max expressed as ml/kg/min is observed with an increase in body mass.

The VO_2 max (mlO_2/kg/min) is therefore considered to be a weight adjusted expression of VO_2 max where the effects of differences in body mass have been factored out. VO_2 max is usually reported as an absolute volume per minute (L/min) for sports such as rowing, in which total work output is important, and as a volume per minute relative to body weight (ml/kg/min) in activities such as running, in which the body weight is supported during the performance (McDougal et al., 1991).

It is estimated that the energy cost of running at 4 min/km for a 50 kg person is 54kJ/min, and for a 60 kg person 65 kJ/min (Burke, 1988). Costill & Fox (1969) estimated that one of the subjects in their study, who completed the Boston Marathon in a time of 2 h 22 min and weighed 63 kg, expended an average of 74 kJ/min, which would equate to an energy expenditure of about 10 579 kJ for the entire race. According to the regression equation formulated by Costill & Fox (1969), the caloric energy expenditure per kilogram of body mass is about 1.26 kJ/kg body mass. The above calculation gives a useful estimate of the energy consumption of marathon running and thus on weight loss during training.



Figures 14 : Change in mass over time in athlete 1



Figures 15 : Change in mass over time in athlete 2

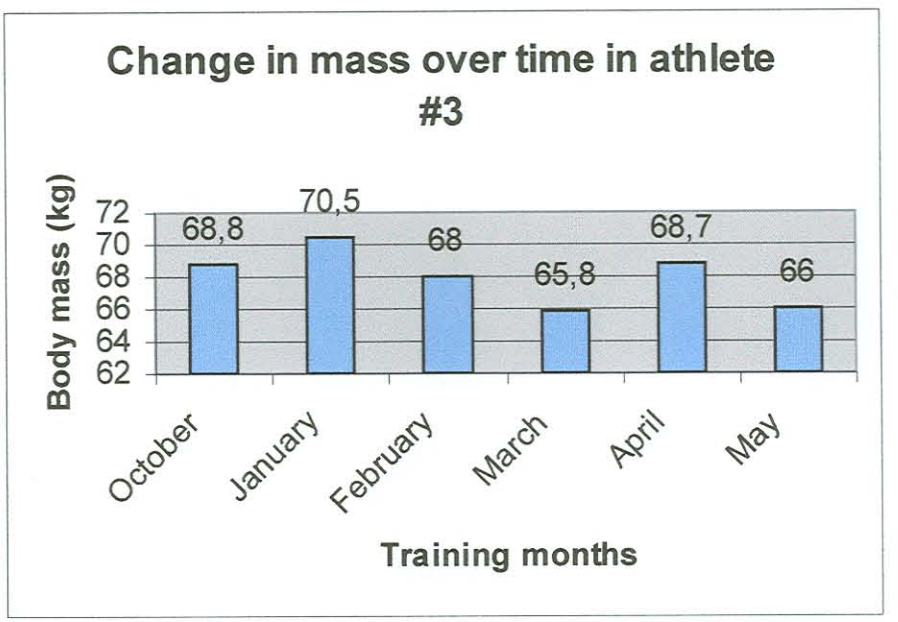


Figure 16: Change in mass over time in athlete 3

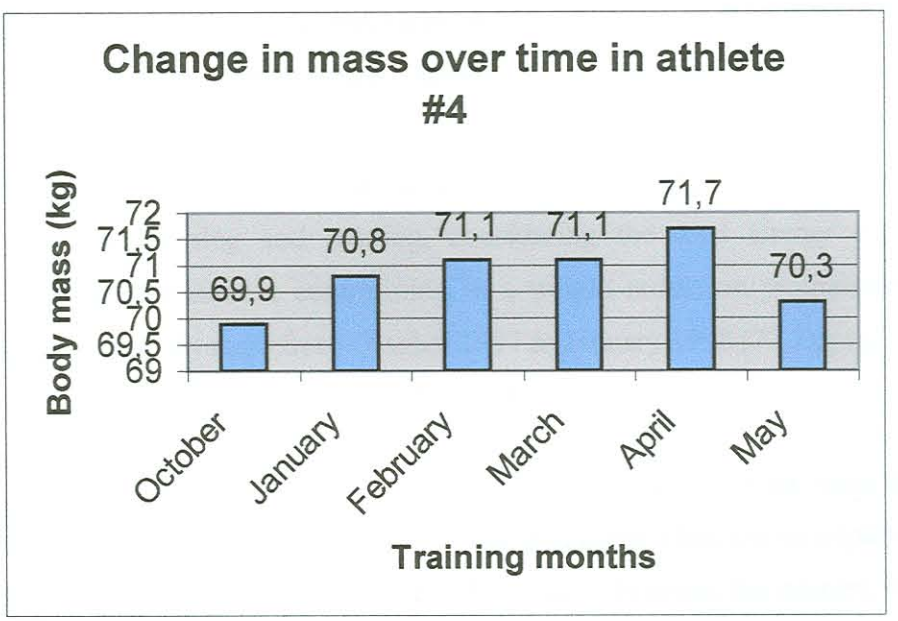


Figure 17: Change in mass over time in athlete 4

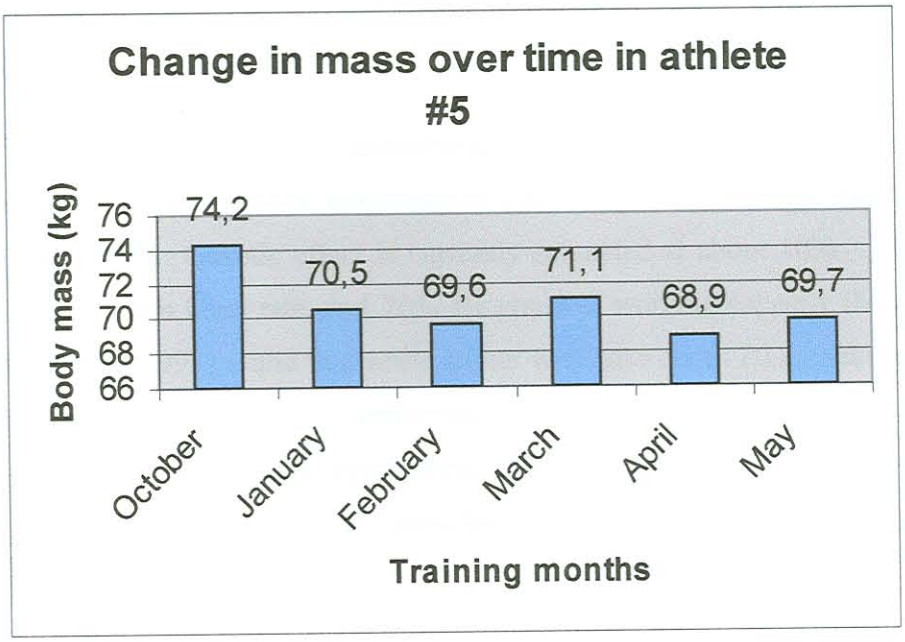


Figure 18: Change in mass over time in athlete 5

The change in mass in all the athletes are shown in Figure 14-18. The mean body mass (kg) of athletes 1 and 4 did not change much during the nine-month training period (0.07% and 0.57% respectively), while a significant decrease was shown in athletes 2, 3 and 5 (-3.15%, -4.07% and –6.06% respectively). Athletes 2 and 3 were also the athletes with the highest mileage in the nine-month period. Chad & Wenger. (1985) report that the duration of activity rather than the intensity has a greater effect on post-exercise oxygen consumption. The metabolic after-effects of marathon running and training, considering the total energy expenditure of prolonged exercise, can make a contribution in a weight reduction programme. Athlete 1 showed a decrease in body mass from October 1997 to January 1998 (73.2kg to 71.8kg) as he was training intensively for the Iron Man in February 1998.

Athlete 3's genetic endowment also plays a role in his body composition, since athlete 3 is a black runner. When trained white males were compared to their black counterparts, Coetzer et al. (1993) found that the main anthropometric differences between the runners in their study were that the black runners were significantly shorter and lighter than the white middle-distance track athletes and had a considerably smaller muscle mass and lean thigh volume. Athlete 3 was slightly lighter than all the other athletes and had the lowest fat percentage. They also had smaller front thigh and medial calf skinfold thickness. The inertia of the limbs

would be less and thus, theoretically, less energy would be expended when moving the limbs (Bosch et al., 1990).

Questions concerning the relative contribution of natural endowment (genotype) to physiologic function and exercise performance (phenotype) have frequently been raised (McArdle et al., 1996). Genetic effect is currently estimated at about 10% – 30% for VO_2 max, 50% for maximum heart rate, and 70% for physical working capacity (McArdle et al., 1996). Coetzer et al. (1993) found that white milers were also about 20 kg heavier (72 vs. 52 kg) than the black distance runners. Furthermore, in a test of muscle function, Coetzer et al. (1993) show that, when undergoing repeated cycles of contraction and relaxation, the muscles of the black distance runners were able to complete more contraction / relaxation cycles before they developed marked fatigue.

Although a vigorous programme of physical training will enhance a person's level of fitness regardless of genetic background, it is clear that the limits for developing fitness capacity are linked to natural endowment (McArdle et al., 1996). Genetic makeup plays such a predominant role in determining the training response that it is almost impossible to predict a particular individual's response to a given training stimulus (McArdle et al., 1996). It is clear that genetic endowment plays an important role in the potential of an athlete. There is evidence that the muscles of white middle-distance runners do not have the same resistance to fatigue as do those of the black distance runners, despite their having the same proportion of T fibres. Black runners are therefore characterised by their extreme fatigue-resistance (Noakes, 1992).

4.1.2.2 Percentage body fat

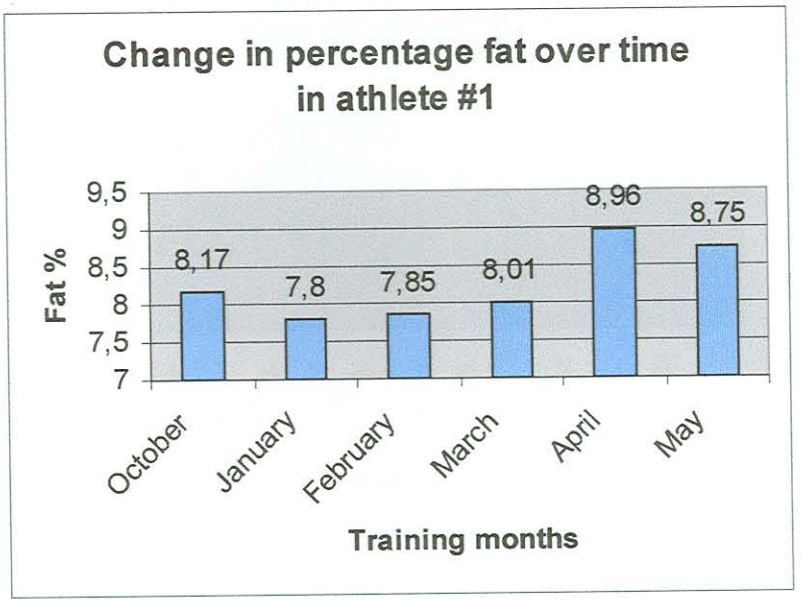


Figure 19: Change in percentage fat over time in athlete 1

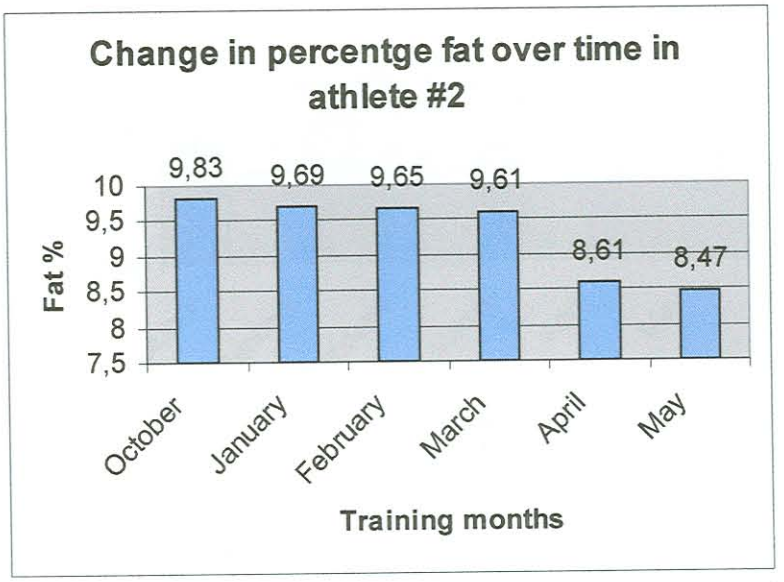


Figure 20: Change in percentage fat over time in athlete 2

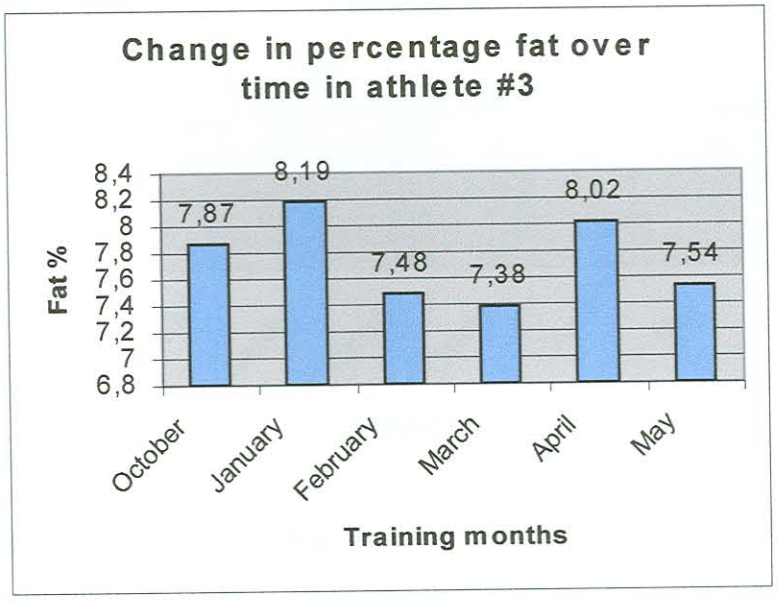


Figure 21: Change in percentage fat over time in athlete 3

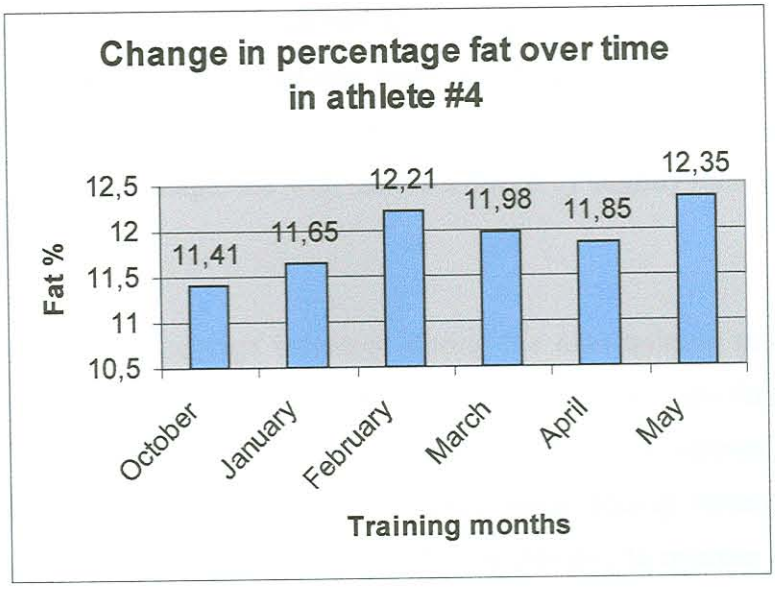


Figure 22: Change in percentage fat over time in athlete 4

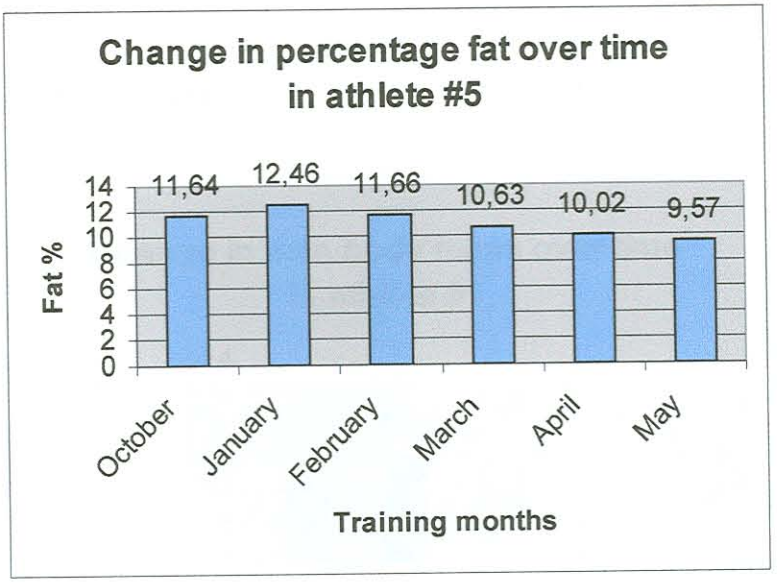


Figure 23: Change in percentage fat over time in athlete 5

Individual changes in fat precentage are shown in Figures 19-23. Lower fat percentges were shown in athletes 2, 3, and 5 (-13.48%, -4.19%, and-17.78% respectively). The fat percentage of athletes 1 and 4 increased with 7.10% and 8.24% respectively. This finding correlates with the increase in both these athletes' total body mass. Since the energy cost of running is a function of body mass, one way of maximising performance is to reduce excess body fat. Previous studies indicate that lower fat-free body weight is one of the variables primarily characterising the faster endurance runners (Costill, 1967; Housh et al., 1988). With every one percent increase in fat percentage, the VO_2 max is reduced by slightly more than one percent (Londeree, 1986).

Although athlete 1's fat percentage increased during the nine training months it was still within the normal range for marathon athletes. He started with a very low fat percentage as he was training for the Iron Man. The results of Mutton et al. (1993) support the use of cross training as an alternative to increasing performance while adding variety to the training programme and possibly reducing the potential for injuries due to overuse or high intensity activity. In the study of Mutton et al. (1993), the cross training group showed improvements in running speed, % VO_2 utilised and a decrease in blood lactate values. The increase in athlete 4's fat percentage can be related to a decrease in training distance as a result of a hamstring injury.

4.1.2.3 Lean Body Mass (LBM)

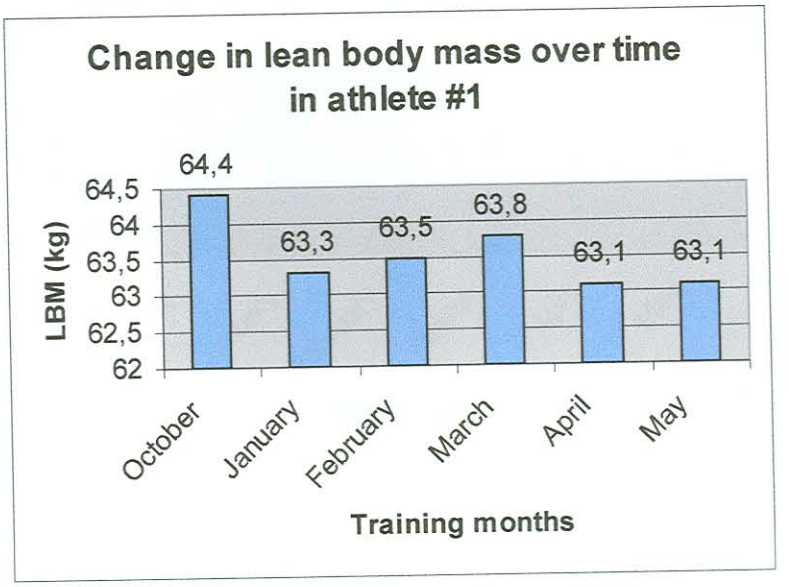


Figure 24: Change in lean body mass over time in athlete 1

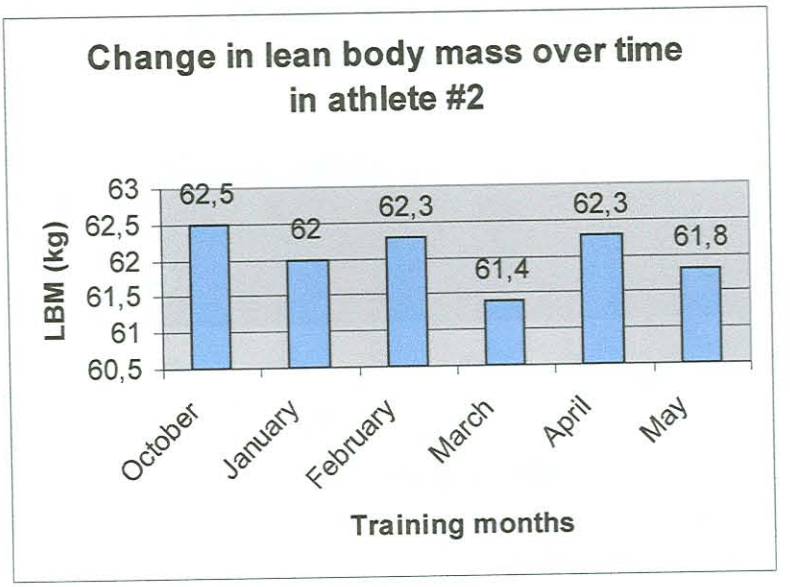


Figure 25: Change in lean body mass over time in athlete 2

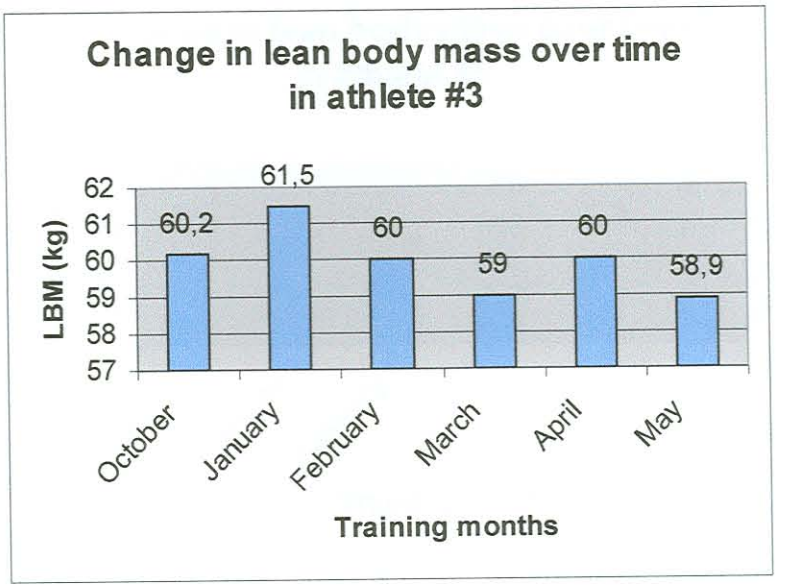


Figure 26: Change in lean body mass over time in athlete 3

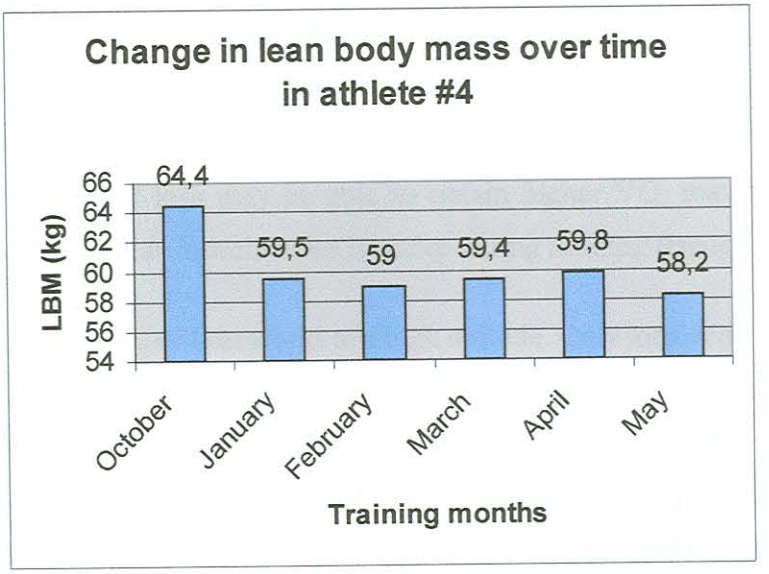


Figure 27: Change in lean body mass over time in athlete 4

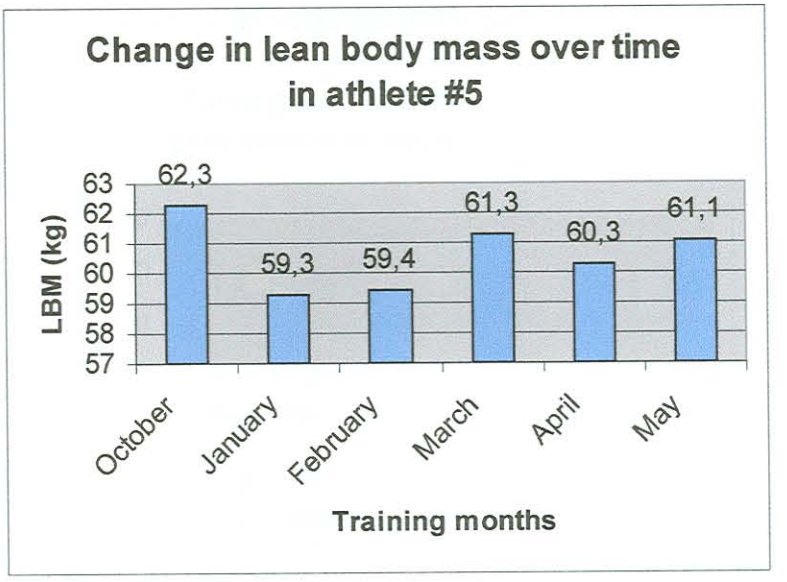


Figure 28: Change in lean body mass over time in athlete 5

All the athletes showed a decrease in lean body mass (Figures 24-28). Results indicate a decrease of 2.02%, 1.12%, 2.16%, 9.63% and 1.93% in athletes 1-5. Morgan et al. (1989) also observed a modest inverse relationship between body mass or weight and economy in èlite runners. However, greater body mass in the trunk area appears to be advantageous in terms of running economy. Conversely, those individuals who possess greater percentages of their body mass in the arms and legs may be able to obtain higher VO_2 max values because a greater proportion of their lean muscle mass is active during running (Bailey et al., 1991).

Athlete 2 trained for a period of four weeks at a high altitude. Only long-term exposure to high altitude produces a significant loss in lean body mass and body fat. The higher basal metabolic rate also plays a role in weight loss. Severe high altitude is thus catabolic. The decrease in muscle fibre size may be directly responsible for increasing capillary density and reducing effective diffusion distance to muscle mitochondria (Green et al., 1989). Thus the loss in lean body mass cannot be the reason for the athlete's lower lean body mass because of his relatively short stay at high altitude. It seems that more interval training and gymnasium work are necessary to build sufficient stamina endurance, which is an important parameter for Comrades athletes.

Below (Figure 29) is a summary of the anthropometric parameters in each individual.

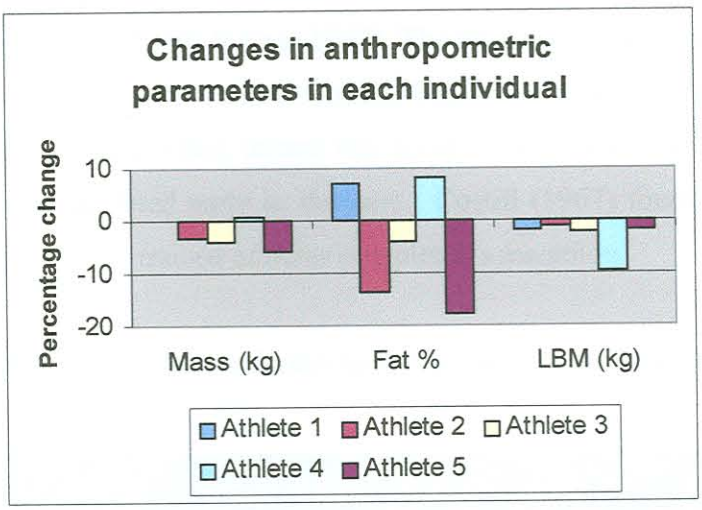


Figure 29 : Summary of the changes in anthropometric parameters in each individual

4.2 RUNNING PERFORMANCE RESULTS

4.2.1 Physiological characteristics of the athletes

The results indicated statistically that some of the VO_2 max parameters changed during the time period of eight months. It has been found, however, that some of the maximum parameters did not change to a great extent (VE/VO_2 = 0.01%, VO_2/HR = 1.3%, VO_2 max = 3.54%, VO_2 absolute = 0.98%, RQ = 0.58%, VT = 4.31%, VE = 0.73%; speed and heartrate showed a decrease at the maximal exercise intensity; speed = -4.94%, heartrate = -4.37%). There was a greater improvement in parameters measured at threshold level (VE/VO_2 = 1.45%, VO_2/HR = 5.43%, VO_2 max = 5.73%, VO_2 absolute = 6.62%, RQ = 1.70%, VT = 4.19%, VE = 7.78%, speed = 9.10% and heartrate = 6.34%).

Table 2-11 represents the improvements in speed, heart rate, VE/VO_2, VO2/HR, VO_2, VO_2 absolute, RQ, VT, VE and RR at maximum and at lactate threshold intensity from October 1997 to May 1998.

4.2.1.1 Speed

Two important physiological variables are important in evaluating distance-running abilities. One is the velocity at anaerobic threshold – the pace at which blood lactate starts to rise substantially. Marathon pace is slightly slower than this. When an athlete starts a race too fast,

lactate will rise to very high levels and muscle glycogen stores will be depleted early in the race. The other important physiological variable is the velocity at VO_2 max, which is typically close to a 3000m race pace (Martin & Coe, 1997). The accumulation of an excessive amount of lactic acid in muscles under stress is a contributing factor to fatigue (Gupta et al., 1996). When an athlete starts a race too fast, lactate will accumulate to very high levels and muscle glycogen stores will be depleted early in the race. Costill (1967) found only a 2–3 times increase in lactate values after trained athletes completed a marathon.

TABLE 2: Percentage change in maximum speed versus change in speed at lactate threshold intensity

SPEED (km/hr)						
Athlete #	**October max**	**May max**	**Maximum % change**	**October lactate threshold**	**May lactate threshold**	**Lactate threshold % change**
#1	20	18	-10%	18.4	17.5	-4.89%
#2	20	17	-15%	18	17	-5.56%
#3	18	19	5.56%	14.4	18	25%
#4	19	18	-5.26%	14	16	14.29%
#5	16	16	0%	12	14	16.67%
Average	**18.6**	**17.6**	**-4.94%**	**15.36**	**16.5**	**9.10%**

Athlete 1 presents a decrease in speed at both lactate threshold (-4.89%) and maximal intensity (-10%). However, his speed (17.5km/hr) at the lactate threshold in May before the Comrades Marathon was still very good for a marathon athlete. This correlates with the good Comrades Marathon time (6h12) which he achieved. Peaking for the Iron Man in February also played a role in his preparation for the Comrades Marathon. Top-level marathon runners who remain injuryfree with excellent general fitness can function well with a cycle that repeats every four to five months. There are 10 to 12 weeks of intense preparation and a few weeks of tapering; then the race, and a month of mental and physical recovery (Lenzi, 1987). This is why the world's healthiest and most consistent marathoners typically compete no more than two to three times a year.

Athlete 2 also shows a decrease in speed at both lactate threshold (–5.56%) and maximal intensity (–15%). He also did the highest training distances per week. Intense distance running has been shown to decrease muscular power (Houmard et al., 1994), however, muscular power is positively associated with distance running performance. Therefore, every effort to maximise power should be made.

Athlete's 3, 4 and 5 show a very positive improvement in speed at lactate threshold intensity (25%, 14.29% and 16.67% respectively). Thus, once VO_2 max has been elevated as high as possible without inordinate additional training volumes, anaerobic development will make the additional difference between being optimally fit and marginally fit. Noakes et al. (1990) show that the physiological variables determining success at distances from 10-90km are the same; at least in marathon and ultramarathon specialists. This suggests that with appropriate training for longer distance events, the fastest 10km runners will also be the fastest marathon and ultramarathon runners. Billat et al. (1994) report that the subjects capable of sustaining the maximal aerobic speed for a longer period of time were also those who displayed a marked increase in lactate concentration at a later stage of a progressive exercise test and who run a 21.1 km race faster. Athletes 1, 2 and 3 showed a very good speed at the lactate threshold (17.5-, 17.0- and 18.0 km/hr) in May before the Comrades Marathon. This correlates with the Comrades Marathon times that they achieved: 6h12 for athlete 1, 6h06 for athlete 2 and 6h08 for athlete 3. Athletes 4 and 5 indicate lower threshold speeds (16- and 14 km/hr). Their times achieved (8h41 and 9h18 respectively) were also much lower than those of athletes 1, 2 and 3.

Noakes et al. (1990) noted a relationship between peak treadmill running velocity and running economy; those athletes who reached the highest treadmill running velocities were also the most economical. The positive correlation found between running economy and running times by Housh et al. (1988) further substantiates the fact that the faster runners were also more metabolically economical. Morgan & Craib (1992) state that athletes who specialise in shorter distance events have been shown to exhibit a better economy at faster speeds, whereas long-distance specialists tend to be more economical at slower running speeds.

According to Kindermann et al. (1979), exercise characterised by a lactate concentration of 4 mmol/L may be carried out for 45-60 min and occasionally longer. Studies done by Mognoni et al. (1990) and Stegmann et al. (1982) are not in agreement with this statement; their results are, however, probably the consequence of an overestimation of the threshold intensity. The exercise duration should nevertheless be kept in mind, since lactate increases with prolonged exercise. Thus the duration of the test is also a variable when determining lactate threshold. The athletes with improved speed ability also present a better fatigue resistance. For exercises lasting more than two hours (e.g. a marathon), it has been shown that the running economy decreased at the end of a long-distance run (Hausswirth et al., 1996). Thus fatigue affects economy in a negative way, increasing aerobic demand through the use of increasingly tired primary movers (i.e. arms and legs) as well as other physical efforts to help maintain pace.

Bosch et al. (1990) report on the difference between black and white runners. Black athletes run at 89% of VO_2 max; white runners at 81% of VO_2 max, when both groups were running at the same percentage of their best marathon race speed. Coetzer et al. (1993) report that the black runners also trained at a higher average exercise intensity than the white runners. Lower blood lactate concentrations were found in the black runners. The lower blood lactate concentration at any given running speed might have contributed to the superior fatigue resistance of the black athletes. These findings are reinforced by this study, since athlete 3, who is a black athlete, performed in accordance with these trends.

Anaerobic metabolism may also occur among the Comrades athletes at the start, or while running up hills, but the lactic acid concentration in the blood immediately after a standard marathon (Costill, 1970) and the Comrades Marathon (Jooste et al., 1981) is fairly low. These facts confirm the conviction that the Comrades Marathon is run mainly on aerobic energy. The pace of each athlete is thus limited largely by his lactic acid turning point, since running above this point causes muscular fatigue (Jooste et al., 1981).

The optimum training intensity for improving endurance performance remains established; on theoretical grounds it has been suggested to be the maximum intensity that can be maintained in a steady state. Jacobs (1987) suggests that a blood lactate concentration of 4 mmol/L represents the optimum intensity. Aerobic interval training is necessary to improve tolerance

to racing at the lactate threshold. This method of training is very effective for improving VO_2 at lactate threshold and/or economy of movement (Foss & Keteyian, 1998). Heck et al. (1985) suggest that èlite runners who were stagnated in their performance improved only after decreasing the intensity of their training to that associated with the aerobic anaerobic threshold. Weltman et al. (1992a) have demonstrated that at least some training above the lactate threshold is required for improvement. Yoshida et al. (1990) report that in distance runners and competitive walkers, blood lactate variables such as lactate threshold (VO_2 at threshold, and velocity at threshold) and OBLA (VO_2 at OBLA, velocity at OBLA) as well as running velocity were significantly improved after extra endurance training, but VO_2 max was not significantly improved.

4.2.1.2 Heart rate

TABLE 3: Percentage change in maximum heart rate versus change in heart rate at lactate threshold intensity

HEART RATE						
Athlete #	**October max**	**May max**	**Maximum % change**	**October lactate threshold**	**May lactate threshold**	**Lactate threshold % change**
#1	176	176	0%	165	172	4.24%
#2	185	177	-4.32%	170	177	4.12%
#3	172	166	-3.49%	165	166	0.61%
#4	195	180	-7.69%	161	169	4.97%
#5	174	163	-6.32%	135	159	17.78%
Average	**180.4**	**172.4**	**-4.37%**	**159.2**	**168.6**	**6.34%**

Results indicated a decrease in the heart rate of all the athletes at maximal intensity and an increase in heart rate at lactate threshold intensity, except for athlete 1 who showed no difference at maximal intensity (see table 3). Thus, training prescriptions based on heart rate at designated metabolic markers with subsequent heart rate monitoring will enable coaches and athletes to monitor training intensity accurately. The ability, then, to sustain high heart rates during prolonged exercise could be hypothesised to be necessary for fast paces and fast

finishing times. According to this statement, athletes 1-5 improved threshold heart rate; an improvement which is very important for Comrades Marathon athletes.

Trained individuals have a much higher maximal cardiac output than untrained individuals - 40 versus 25 L/min. Maximal cardiac output is limited by the maximal rate of depolarisation of the sino-artrial (SA) node and the structural limits of the ventricle. It is estimated that 70 – 85% of the limitation in VO_2 max is linked to maximal cardiac output (David et al., 2000). Athletes 3 and 5 indicate a very low maximum heart rate (166 and 163 beats per minute); whereas the Comrades Marathon star Nick Bester said after the 1997 Comrades that in the last few kilometers he was unable to keep his heart rate up at the remarkable 170 beats per minute he had sustained the whole race, "because my legs were too tired, I just couldn't move any faster" (Comrades Marathon update, 1997). Heart rates generally rise quickly to a steady state heart rate which is maintained early in exercise (i.e. up to approximately 30 minutes), followed by gradually increasing heart rates if exercise is continued for prolonged periods (Toole et al., 1998). Although specific reasons for slowing pace during prolonged exercise may vary among individual athletes, potential contributing reasons include substrate depletion, altered muscle efficiency, fluid and electrolyte imbalances, thermoregulatory problems, cardiac fatigue and psychological factors (Toole et al., 1998). The ability, then, to sustain high heart rates during prolonged exercise could be hypothesised to be necessary for fast paces and fast finishing times.

Athlete 2, who exercise for a month period at high altitude, showed a decrease in his maximal heart rate (185-177 beats per minute). Although some adaptations during acclimatisation to altitude should enhance aerobic capacity and endurance performance upon return to sea level, research results do not support this effect (Banister et al., 1978). This is probably the result of the altitude-related decrease in both maximum heartrate and stroke volume. For the highly trained athlete, the training intensity required for the maintenance of peak performances cannot be achieved at altitude.

Heck et al. (1985) suggest that èlite runners who stagnated in their performance improved only after decreasing the intensity of their training to that associated with the aerobic anaerobic threshold. Weltman et al. (1992) have demonstrated that at least some training above the

lactate threshold is required for improvement. However, Lehmann et al. (1992) suggest that athletes respond better to increases in training intensity than in training volume. If training at the maximal steady state was the "best" method to exercise, one would have hypothesised that the increased volume study would have been tolerated better than increases in the homeostasis disturbing training intensity (Snyder et al., 1994). However, if training intensity associated with maximal lactate steady state is not the best form to exercise in, it may be that this intensity simply represents the most time effective way of integrating training volume and intensity (Snyder et al., 1994).

4.2.1.3 Ventilatory equivalent (VE/VO_2)

TABLE 4: Percentage change in maximum VE/VO_2 versus change in VE/VO_2 at lactate threshold intensity

VE/VO_2						
Athlete #	October max	May max	Maximum % change	October lactate threshold	May lactate threshold	Lactate threshold % change
#1	29.9	27.5	-8.03%	26.4	23.3	-11.74%
#2	25.6	26	1.56%	26.3	25.6	-2.66%
#3	27.4	27.2	-0.73%	23.9	27.2	13.81%
#4	33.3	30.5	-8.41%	25.7	28.3	10.12%
#5	28.3	32.7	15.55%	26.4	25.8	-2.27%
Average	**28.9**	**28.78**	**-0.01%**	**25.74**	**26.04**	**1.45%**

Only athlete 1 indicated a decrease in VE/VO_2 at both lactate threshold (-11.74%) and maximal intensity (-8.03%). Athletes 3 and 4 showed an increase in VE/VO_2 at lactate threshold intensity (13.81% and 10.12%). Athlete 5 showed an improvement only at the lactate threshold intensity (-2.27%). In healthy young adults, this ratio is usually maintained at approximately 25 L during submaximal exercise up to approximately 55% of the oxygen uptake. Thus, a decrease in VE/VO_2 at ventilatory threshold indicates a better oxygen extraction potential, and is therefore more advantageous for marathon athletes. Thus, although

athletes 3 and 4 indicate a increase in VE/VO_2, al the athletes' (1 – 5) ventilatory equivalent are still below 30L, which is ideal for marathon runners (see table 4).

Dempsey (1986) states that the ability of the skeletal muscle to adapt to training is far greater than what is observed in the lung. Thus, the main significance of the training-induced increase in capillary density is not to accommodate blood flow but rather to maintain mean transit. This enhances oxygen delivery by maintaining oxygen extraction even at high rates of muscle blood flow.

The study by Ramsbottom et al. (1989) demonstrates a decrease in the ventilatory equivalent for oxygen with endurance training. This decrease represents a more efficient utilisation of oxygen and may reflect an increased mechanical efficiency of the running action, and hence on increased aerobic capacity of human skeletal muscle. Wasserman et al. (1986) suggest that lactate production during exercise depends mainly on the availability of oxygen in the active tissue. Therefore there is a decrease in blood lactate concentrations with endurance training.

Endurance trained athletes demand a lower V_E than do untrained athletes (Bailey et al., 1991). Lower ventilation, particularly over a prolonged effort (e.g. the Marathon), would mean, on a ratio basis, less oxygen to the respiratory muscles and more to the working skeletal muscles (Fox et al., 1993). Thus manipulation of the amount of ventilatory work necessary at a given running velocity could alter overall running economy (Bailey et al., 1991).

4.2.1.4 Oxygen pulse (VO_2/HR)

TABLE 5: Percentage change in maximum VO_2/HR versus change in VO_2/HR at lactate threshold intensity

VO_2/HR						
Athlete #	**October max**	**May max**	**Maximum percentage change**	**October lactate threshold**	**May lactate threshold**	**Lactate threshold percentage change**
#1	25.5	27	5.88%	23.2	27.09	16.77%
#2	23.5	24.8	5.53%	20.5	21.5	4.88%
#3	27.7	28.4	2.53%	24.5	28.4	15.92%
#4	22.5	24.3	8%	22.1	23.3	5.43%
#5	25.9	21.9	-15.44%	24	20.2	-15.83%
Average	**25.02**	**25.28**	**1.3%**	**22.86**	**24.09**	**5.43%**

The VO_2 max is limited primarily by the rate of oxygen delivery, not by the ability of the muscles to take up oxygen from the blood. Therefore, the following factors could play a role in the limiting of VO_2 max: the pulmonary diffusing capacity, maximal cardiac output, the oxygen-carrying capacity of the blood, and skeletal muscle characteristics (David et al., 2000). Therefore, oxygen pulse plays a prominent role in the determination of aerobic endurance.

The predicted maximum oxygen pulse is the quotient of predicted maximum VO_2 and predicted maximum HR. In any given individual there is a close relationship between VO_2 and HR during exercise. The quotient of the VO_2 and HR is the oxygen pulse. The normal relationship of VO_2 to HR is linear over a wide range with a positive intercept on the HR axis. All the athletes showed an increase in the oxygen pulse at lactate threshold and maximal intensity, except for athlete 5. Athlete 5 indicates a lower maximum heartrate (174 – 163 beats per minute) and his training was not very high in terms of distance. The average improvement in lactate threshold is 5.43%, compared to 1.3% at maximal intensity (table 5).

The predicted O_2 pulse at any given VO_2, including maximum VO_2, is strongly dependent on the normal individual's body size, sex, age, degree of fitness, and hemoglobin concentration. The O_2 pulse can be considerably higher than predicted in the cardiovascularly fit person. All the athletes' (1 – 5) oxygen pulse is within the ideal range of 20 –25 mlO_2/HR.

4.2.1.5 Oxygen consumption (VO_2)

TABLE 6: Percentage change in maximum VO_2 versus change in VO_2 at lactate threshold intensity

VO_2						
Athlete #	**October max**	**May max**	**Maximum % change**	**October lactate threshold**	**May lactate threshold**	**Lactate threshold % change**
#1	61.5	65.3	6.18%	52.5	62.8	19.62%
#2	58.5	62.2	6.32%	56.9	50	-12.13%
#3	61.3	62	1.14%	58.8	62	5.44%
#4	62.4	62.7	0.48%	51.6	56.4	9.30%
#5	50.1	51.9	3.59%	43.8	46.6	6.39%
Average	**58.76**	**60.82**	**3.54%**	**52.72**	**55.56**	**5.73%**

A very positive improvement in the VO_2 is shown at lactate threshold (5.73%) and maximal intensity (3.54%) (table 6). Although some researchers claim that VO_2 max among èlite-level runners changes little over the course of a year, Martin & Coe (1997) found substantial differences as either training load or training emphasis shifts. Powers et al. (1983) demonstrated that the oxygen uptake measured at the ventilatory threshold was a better predictor of distance running success than either VO_2 max or running economy (Louanne et al., 1989; Schneider et al, 1991).

However, athlete 2 indicates a decrease (-12.13%) in VO_2 at lactate threshold intensity. Various reasons can be given for the decrease in VO_2 max. According to McArdle et al. (1996), maximal oxygen uptake will vary between 5% – 20%, depending on whether a person is "in shape" or "out of shape" at the time of measurement. Douglas et al. (1981) report that

distance runner Jim Rhyn's maximal aerobic capacity varied from 65-81 ml O_2/kg/min depending on his state of conditioning. Bouchard et al. (1992) state that there are high and low responders to training, and this factor is hereditary. This again demonstrates that VO_2 max is in fact a poor indicator of fitness, since one's ability to run both longer and faster will increase by more than 15% with training. For example, former mile world record holder, Jim Ryan, increased his VO_2 max from 65 ml O_2/kg/min in the partially trained state to 82 ml O_2 /kg/min in the trained state, a huge 26% increase (Noakes et al., 1992). This once again emphasises the ability of the èlite athlete to show a greater adaptation to a training response. Most of the increase in VO_2 max is due to an increase in muscle contractility, which increases the capacity of the muscles to produce power.

Day to day variation could also play a role in the decrease or increase of VO_2 max; however, a number of studies have examined the problem of day-to-day variability in oxygen consumption during submaximal running. Variability across subjects ranges from 0.30 to 4.40% in Morgan et al. (1991) and 1.20 to 5.80% in Williams et al. (1991). Pereira et al. (1991) demonstrated that intra-individual variation in VO_2 during steady-state graded treadmill running is small.

Weltman (1990) reports that it is possible to increase velocity and VO_2 at OBLA without changing VO_2 max. Housh et al. (1988) reports that trained males exhibit lower submaximal steady-state VO_2 responses than untrained males. Therefore, while short-term endurance training may not be effective in modifying running economy, it is possible that prolonged training could result in improved biomechanical efficiency and therefore lower submaximal steady-state VO_2 values.

Endurance training has been shown to delay the onset of both the lactate and the ventilatory thresholds (Schneider et al., 1991; Hoffmann et al., 1993). Top marathon runners (i.e. sub 2h20 min) can sustain 86% of VO_2 max for the duration of a race (Hawley, 1995), whereas slower runners (i.e. 2 h 45 min up to 3 h) can sustain only 75% of their VO_2 max for the same distance (Farrel et al., 1979; Hawley, 1995). The optimum training intensity for improving endurance performance remains established; on theoretical grounds it has been suggested to be the maximum intensity that can be maintained in a steady state.

Martin & Coe (1997) question the benefit of more than 115 to 120 km a week at low intensity aerobic conditioning paces for distance runners seeking to improve their VO_2 max. The studies of Scrimgeour et al. (1986), which report that athletes training less than 60 km a week have as much as 19% less running economy than athletes training more than 100 km a week, might support this suggestion. This can explain athlete 5's lower lactate threshold as athlete 5's average training distances were lower than 60 km per week. Athletes 2 and 3 trained more than 140 km per week from January to May. However, both these athletes' VO_2 max increase slightly, but athlete 2's lactate threshold VO_2 max did not improved. Athlete 1 trained on average 100 km per week and peaked on 160 km the last two months before the Comrades. He indicated the greatest improvement (19.2%) at lactate threshold intensity (52.5 to 62.8 $mlO_2/kg/min$). Martin & Coe (1997) report that as athletes become better trained, not only does the VO_2 max rise, but so does the lactate/ventilatory threshold, both in absolute terms and as a percentage of VO_2 max. These long runs are not only important for endurance development but also for mental preparation for the Comrades.

Thus, endurance athletes have higher anaerobic thresholds than non-athletes, where the anaerobic threshold is expressed as a fraction of VO_2 max (Haffor et al., 1990). Ramsbottom et al. (1989) states that training status could be better explained in terms of the highest proportion of the VO_2 max at which a steady-state could be achieved rather than in terms of VO_2 max alone. Most of the athletes indicate a greater improvement in the fraction of VO_2 max at threshold intensity (5.73%) than in the change in VO_2 max (3.54%). Weltman et al. (1990) report that it is possible to increase velocity and VO_2 at OBLA without changing VO_2 max.

Yoshida et al. (1990) report that in distance runners and competitive walkers, blood lactate variables such as lactate threshold (VO_2 at threshold, and velocity at threshold) and OBLA (VO_2 at OBLA, and velocity at OBLA), as well as running velocity, were significantly improved after extra endurance training, but VO_2 max was not significantly improved.

Yoshida et al. (1992) name three factors that contribute to the improvement of VO_2 parameters after training, namely:

- an improved capacity for mitochondrial respiration in muscle;

- an increased availability of blood and/or muscle O_2 stores; and
- an elevation of cardiac output and/or an increase in muscle blood flow.

In this context, Yoshida et al. (1992) document that endurance training induces an improvement of mitochondrial respiratory function, resulting in a reduced production of lactate during heavy exercise. According to Yoshida et al. (1992) the positive improvement in the VO_2 max (5.73%) and speed (16.67%) at lactate threshold intensity can be related to athletes' endurance training.

2.4.1.6 Respiratory quotient (RQ)

TABLE 7: Percentage change in maximum RQ versus change in RQ at lactate threshold intensity

RQ						
Athlete #	**October max**	**May max**	**Maximum % change**	**October lactate threshold**	**May lactate threshold**	**Lactate threshold % change**
#1	1.1	1.03	-6.36%	1.08	0.98	-9.26%
#2	0.96	0.98	2.08%	0.95	0.97	2.11%
#3	1.1	0.99	-10.00%	1.07	0.99	-7.48%
#4	1.13	1.1	-2.65%	0.97	0.99	2.06%
#5	1.07	1.22	14.02%	0.98	1.02	4.08%
Average	**1.072**	**1.064**	**-0.58%**	**1.01**	**0.99**	**-1.70%**

The application of the RQ is based on the assumption that the exchange of oxygen and carbon dioxide measured at the lungs reflects the actual gas exchange from nutrient catabolism in the cell (McArdle et al., 1996). This assumption is only valid during steady state or resting conditions (lactate threshold intensity). When other factors such as high intensity exercise or hyperventilation affect the RQ so that it no longer reflects only the substrate mixture in energy metabolism, it is described as respiratory exchange ratio (R).

A decrease in the RQ values (at lactate threshold intensity) in marathon athletes is more advantageous; thus the decrease in both lactate threshold (-1.7%) and maximal intensity (-0.58%) is an improvement. Athletes 1, 3 and 4 indicate a decrease in RQ at maximum intensity. As a result of high volumes of endurance training and less speed work it is possible to have a decrease in the lactate tolerance and therefore a decrease in RQ values. Their speed at lactate threshold had, however, improved; this is therefore an indication of an improvement in their aerobic system.

Only athletes 2 and 5 showed an increase in their RQ values at maximum intensity. During exhaustive exercise, R can rise significantly above 1,00. The lactic acid generated during anaerobic metabolism is buffered by sodium bicarbonate in the blood to maintain the acid-base balance. Because of the buffering effect, the CO_2 values rise to very high levels, above the quantity normally released during energy metabolism. Carbon dioxide elimination increases during hyperventilation, and as a result of that, the normal level of carbon dioxide in the blood is reduced. This elimination is not accompanied by a rise in the oxygen uptake; thus, the rise in the RQ does not represent the oxidation of food.

However, McArthur et al. (1983) found that higher muscle glycogen levels occurred after and higher RQ values during marathon races in the better runners. Hirokoba et al. (1992) found that endurancetrained men generate more CO_2 excess at the same blood lactate concentration when compared with non-endurancetrained and untrained men. There are two possible explanations for this:

- the increase in CO_2 excess per unit of body mass per lactate accumulation may be due to the decrease of buffering in the non-bicarbonate system; or
- the increase of buffering in the bicarbonate system.

These reasons can possibly explain the RQ increase in athletes 2 and 5.

4.3 BREATHING DYNAMICS

The primary task of the pulmonary system in accommodating the needs of an endurance athlete for either hard training or competition is to provide adequate gas exchange between alveoli and arterial blood with minimal work required by the lungs and chest. The ventilatory

system's efficiency for providing airflow is self-optimising (Martin & Coe, 1997). Expired ventilation (V_E) is the product of breathing rate (RR) and tidal volume (V_T).

4.3.1 Tidal volume (VT)

TABLE 8: Percentage change in maximum VT versus change in VT at lactate threshold intensity

VT						
Athlete #	October max	May max	Maximum % change	October lactate threshold	May lactate threshold	Lactate threshold % change
#1	2883	2903	0.69%	3204	3240	1.12%
#2	2138	2354	10.10%	1831	1980	8.14%
#3	1933	2165	12.00%	2073	2165	4.44%
#4	2885	2884	-0.03%	2844	2783	-2.14%
#5	2617	2585	-1.22%	2520	2757	9.405%
Average	2491.2	2578	4.31%	2494.4	2585	4.19%

During exercise that elicits VO_2 max, as much as 8% – 11% of the total oxygen uptake is required for respiratory muscle work. The respiratory muscles use approximately 40% – 60% of their maximum capacity to generate pressure at this exercise level (Aaron, 1992). Breathing can be optimized when the tidal volume is never more than 60% to 65 % of the vital capacity, defined as the maximum amount of air that can be exhaled after a maximal inspiration (Martin & Coe, 1997). Breathing rate values recorded in highly trained athletes were no greater than 55 per minute (Martin & Coe, 1997).

Lower ventilation, particularly over a prolonged effort (e.g. the Comrades Marathon), would mean, on a ratio basis, less oxygen to the respiratory muscles and more to the working skeletal muscles (Fox et al., 1993). Thus, manipulation of the amount of ventilatory work necessary at a given running velocity could alter overall running economy (Bailey et al., 1991).

Tidal volume increased in both maximal (4.31%) and lactate threshold intensity (4.19%). The increase indicates a better percentage lungfilling with exercise at maximum and at lactate

threshold intensity. All the athletes' lungfilling were below 65% of vital capacity, thus the improvement in V_T does not affect the running economy negatively.

During long duration exercise at relatively low work intensities, such as between 50% to 60% of VO_2 max for about 2 h, a gradual but measurable rise in breathing rate (15% to 40%) does occur. This is accompanied by a reduction in tidal volume of about 10%-15%. The decrease in tidal volume does not exactly compensate for the increased frequency, because V_E increases as well. This drift is not observed during the short-duration runs (Martin & Coe, 1997) and is therefore not noticeable in the test results. Thus the drift will be observed during the Comrades Marathon.

4.3.2 Minute ventilation (VE)

TABLE 9: Percentage change in maximum VE versus change in VE at lactate threshold intensity

VE						
Athlete #	**October max**	**May max**	**Maximum % change**	**October lactate threshold**	**May lactate threshold**	**Lactate threshold % change**
#1	134.5	131.6	-2.16%	101.4	106.9	5.42%
#2	109.7	113.4	3.37%	91	89.8	-1.32%
#3	114.4	111.4	-2.62%	96	111.4	16.04%
#4	143.8	134.2	-6.68%	91.7	111.8	21.92%
#5	105	117.3	11.71%	85.7	83	-3.15%
Average	**121.48**	**121.5**	**0.73%**	**93.16**	**100.58**	**7.78%**

Minute ventilation showed a small increase in maximal intensity (0.73%) and an increase of 7.78% at lactate threshold intensity. During strenuous exercise, the breathing rate increases between 35–45 breaths per minute, although rates as high as 60–70 have been measured in èlite athletes. In male endurance athletes, minute ventilation can increase to 160 L/min. Ventilation volumes of 200 L have been reported in research studies (McArdle et al., 1996). Even with such large V_E, tidal volumes for both trained and untrained individuals rarely exceed 60% of vital capacity. Endurance trained athletes demand a lower V_E than do

untrained athletes (Bailey et al., 1991). Athletes 1 and 4 have exercise induced asthma and they indicate the highest minute ventilation (134.5 and 143.8 L respectively). Their values did, however, improve during their training for Comrades. The other athletes indicate a relatively low V_E (below 120 L)

Wasserman et al. (1986) report V_E values of 15 liter per minute or 20 to 40% of the maximal voluntary ventilation in very fit individuals. A low breathing reserve is characteristic of patients with lung disease who are ventilatorily limited. CO_2 is a powerful ventilatory stimulant, and a small rise in the $P_a CO_2$ probably increases the V_E by 10%-30%. The level of V_E, with its removal of CO_2, thereby serves as the major determinant of arterial H^+ ion concentration during this submaximal long-term work (i.e. at workloads ranging from a long training run to marathon or ultradistance racing).

The average rise in V_E (7.78%) at lactate threshold intensity found in this case study can be a result of the higher speed (9.10%) achieved by the athletes during the last test before OBLA was reached. These changes in volume and rate dynamics are controlled automatically to optimise mechanical efficiency while maintaining normal blood O_2 and CO_2 concentrations. Thus, it is unwise for coaches or athletes to attempt voluntary regulation of breathing patterns (Martin & Coe, 1997).

4.3.3 Respiration rate (RR)

TABLE 10: Percentage change in maximum RR versus change in RR at lactate threshold intensity

RR						
Athlete #	October max	May max	Maximum % change	October lactate threshold	May lactate threshold	Lactate threshold % change
#1	47	45	-4.26%	32	33	3.13%
#2	51	48	-5.88%	50	45	-10.00%
#3	59	51	-13.56%	46	51	10.87%
#4	50	47	-6.00%	32	40	25.00%
#5	40	45	-12.50%	34	30	-11.76%
Average	49.4	47	-3.445%	38	39.8	3.45%

Excessively deep breaths, few in number, would be too energy costly. A large number of breaths, each small in volume, would not provide effective alveolar gas exchange. Breathing can be optimised with tidal volume never more than 60% to 65% of the vital capacity. Breathing rate values recorded in highly trained athletes were no greater than 55 per minute (Martin & Coe, 1997). None of the athletes reached a breathing rate of over 55 per minute except for athlete 3 during the first test. It is possible that he was feeling uncomfortable with the mask the first time. His respiration rate did, however, improve from 59 to 51 per minute. All the other athletes also indicated a decrease (-3.445%) in respiration rate at maximum intensity.

Only athletes 2 and 5 showed a decreased at lactate threshold intensity, while athletes 1, 3 and 4 indicated an increase. Carbon dioxide elimination increases during hyperventilation; for example, athlete 5 indicates a lower maximum heartrate (174 – 163 beats per minute), lower oxygen pulse and a higher RQ. The exercise induced asthma of athletes 2 and 5 also plays a role in their higher respiration rate.

Quite often, runners synchronise their breathing rate to their stride frequency. One practical implication of this breathing pattern is the usefulness of shortening stride and quickening cadence when climbing hills. The resulting increased breathing rate with increased stride frequency helps increase O_2 intake.

4.4 PERCENTAGE IMPROVEMENT IN THE VO_2 PARAMETERS AT LACTATE AND MAXIMAL INTENSITY

Figures 30 to 34 show the graph for each VO_2 parameter for each of the 5 athletes and it shows how the parameter has increased/decreased over time. On each graph the maximum as well as the lactate threshold is shown.

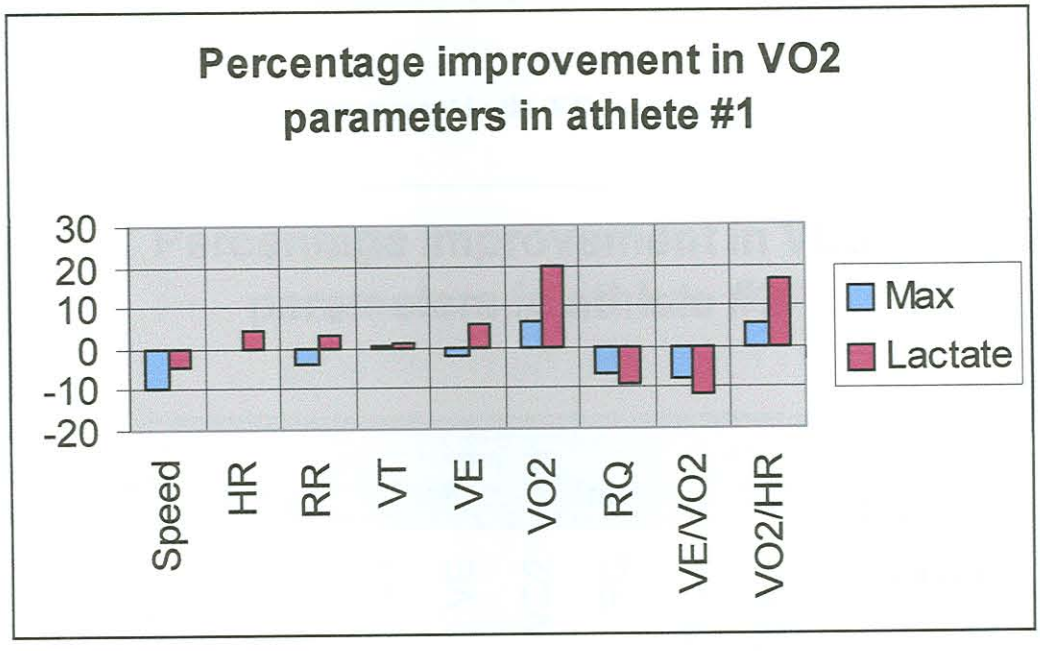


Figure 30: Percentage improvement in the VO_2 parameters in athlete 1

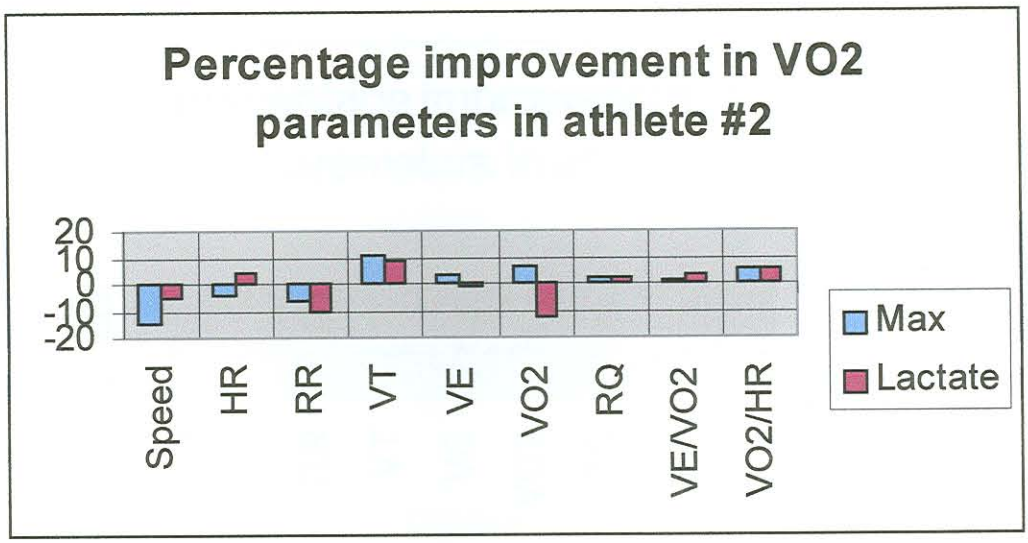


Figure 31: Percentage improvement in the VO_2 parameters in athlete 2

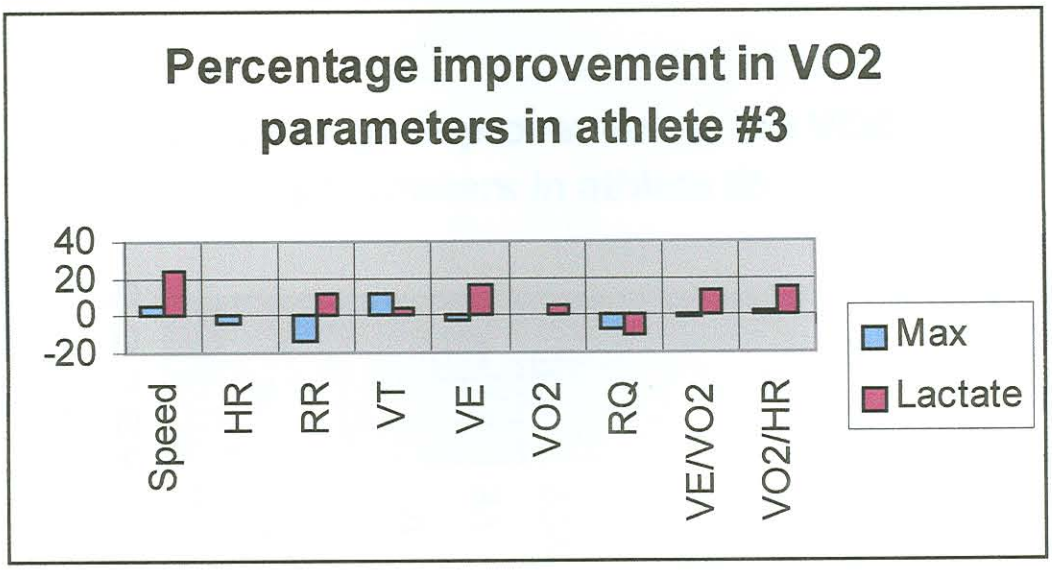


Figure 32: Percentage improvement in the VO_2 parameters in athlete 3

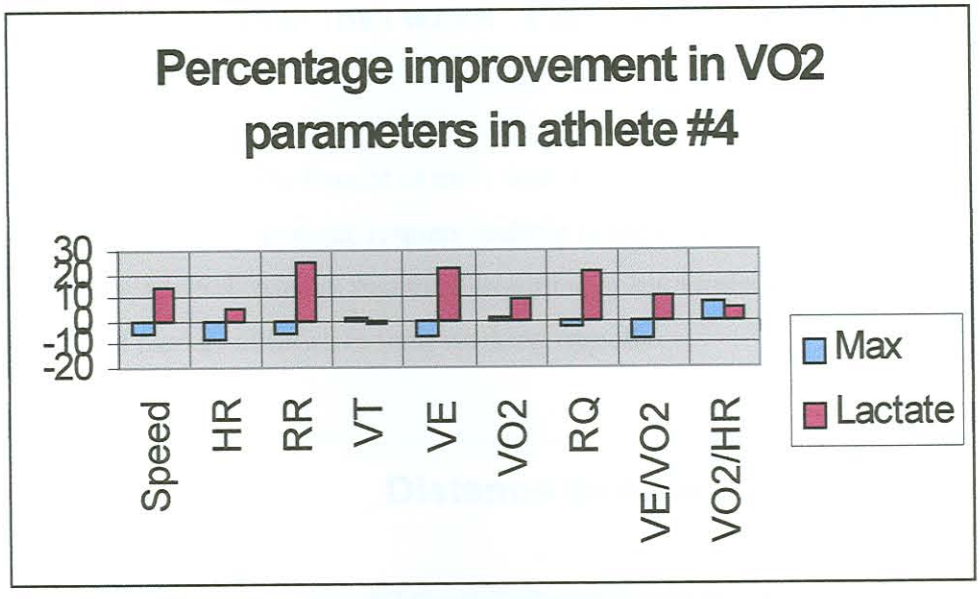


Figure 33: Percentage improvement in the VO_2 parameters in athlete 4

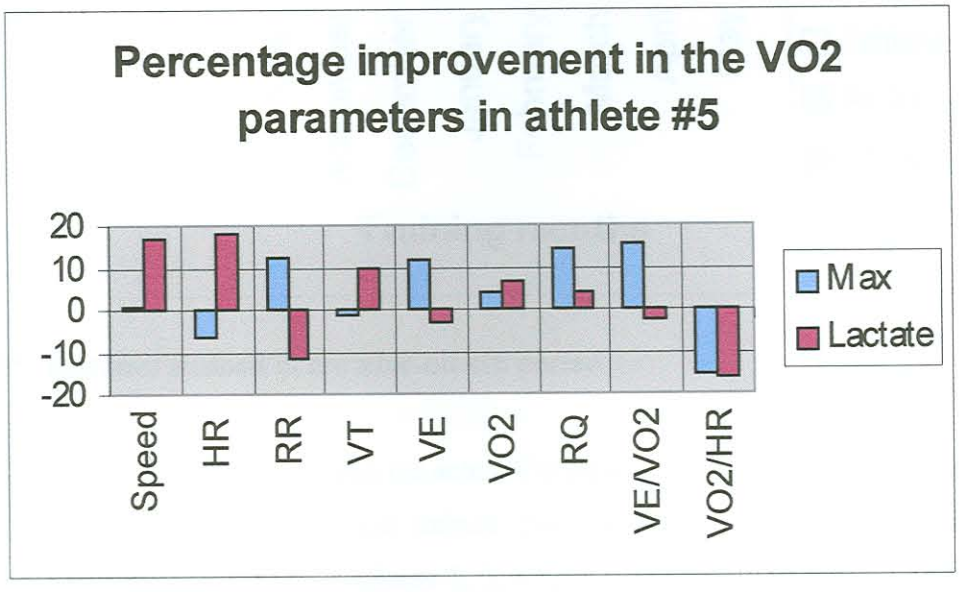


Figure 34: Percentage improvement in the VO_2 parameters in athlete 5

4.5 THE RELATIONSHIP BETWEEN THE DISTANCE TRAINED AND RUNNING TIMES

Martin & Coe (1997) question the benefit of more than 115 to 120 km a week at low intensity aerobic conditioning paces for distance runners seeking to improve their VO_2 max. Marathon runners are, however, special cases in that they require very high training volumes in order to stimulate greater fuel storage abilities in their working muscles.

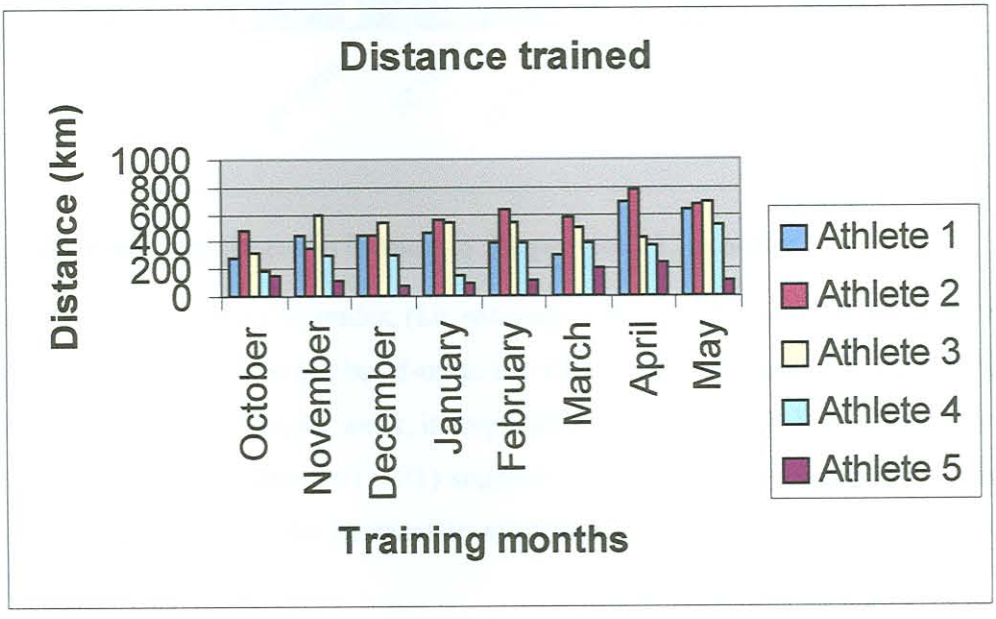


Figure 35: Distance trained in the nine-month period

A positive correlation were found between the actual Comrades time ($p<0.05$) and the distance trained. Athlete 2 has done the highest trained distance (4463km), and completes the Comrades in the fastest time (6h06). Athletes 1, 3, 4 and 5 followed him, in that order (see figure 35)

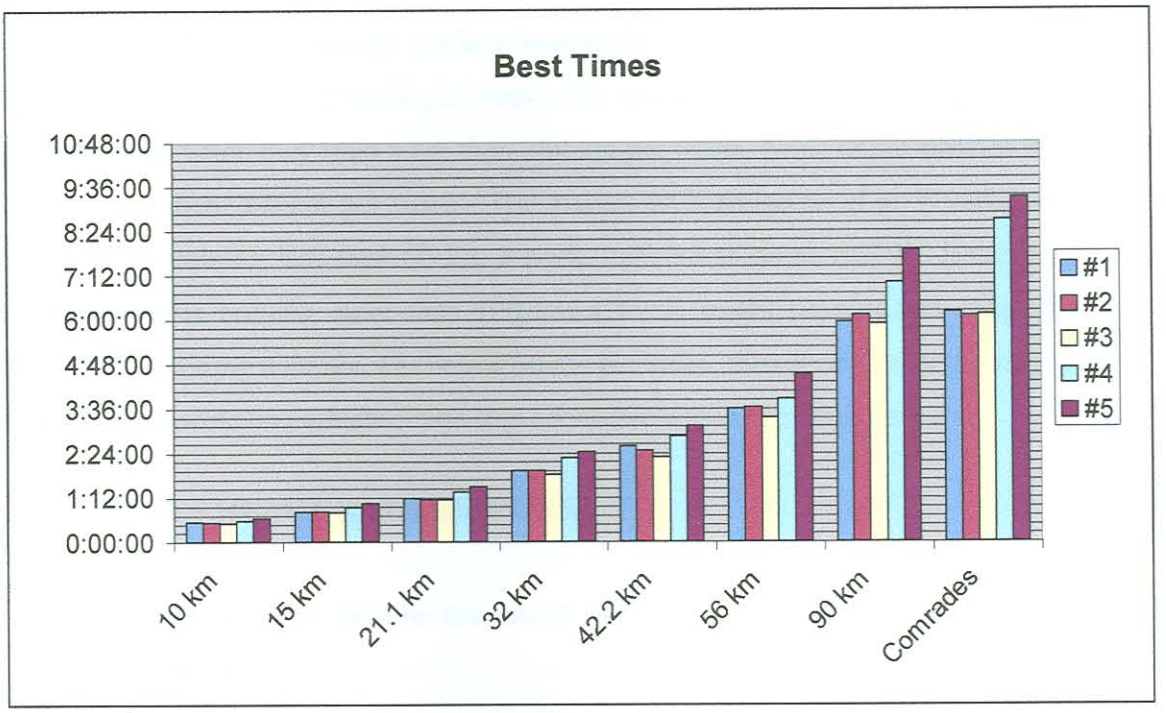


Figure 36 : Best running times performed in the previous 12 months

In the case of club running for the Comrades, (i.e. someone who would run 100 to 120km per week for a period of 4 to 6 weeks in the build-up to the Comrades), the base training for this athlete should be at least 50 to 60km per week, incorporating a long run of at least 18 to 20km. Evidence from Hickson & Rosenkoetter (1981) suggests that training frequency rather than training intensity is responsible for the increase in mitochondrial enzyme concentration and endurance exercise capacity.

A marathon runner, mindful of the effect that very long runs have on the body and the recovery time required, may choose not to train beyond 35 to 38km in preparation for a hard marathon, although some of the èlite athletes do train over distances of up to 45km. Serious Comrades runners invariably train over distances of up to 75km, but usually have just one or two runs, five or six 60-70km runs, and several shorter (40-50km) runs. Even the serious Comrades runner would not train "over distance" (i.e. he she would not, on a given day, exceed the total Comrades distance). Most of the long runs are at a pace slightly slower than race pace, while the very long training runs (over 60km) would be run significantly slower than race pace (Brink, 1999).

Noakes (1992) states that èlite runners perform best in the marathon and ultramarathon races when they train between 120 to 200 km per week, with an increasing likelihood that they will perform indifferently when they train more than 200 km per week. None of the athletes in this study trained more than 200 km per week. The studies of Scrimgeour et al. (1986), which report that athletes training less than 60 km a week indicate as much as 19% less running economy than athletes training more than 100 km a week, might support this suggestion. Most of athlete 5's training distance was below 60 km per week, thus according to Scrimgeour et al. (1986), it is possible that athlete 5 was less economical than the other runners.

4.6 COMRADES HEART RATE RESPONSE

4.6.1 Relationship between lactate threshold heart rate and the actual Comrades heart rate response

By simultaneously measuring maximum aerobic power and exercise heart rate in the laboratory, the scientist can estimate the relative intensity of exercise in the field on the basis of heart rate alone and indirectly determine maximum aerobic power (MacDougall et al., 1991). The 4 mmol/L value for OBLA implies the maximum exercise intensity that a person can sustain for a prolonged period. In reality, this maximum stable lactate level is probably quite variable among individuals (Noakes 1988; Orok et al., 1989; Mognoni et al., 1990). The higher the running speed at which the lactate concentration exceeds the 4 mmol/L threshold, the higher the aerobic capacity. Spurway (1992) calls 2 mmol/L the aerobic and 4 mmol/L the anaerobic threshold.

The relationship between lactate threshold and the actual heart rate response indicates that none of the athletes could complete a 90-km race at the OBLA. It has been found that the athletes could keep their heart rate above a certain percentage of the lactate threshold only for the duration of the race (30.3% above 95% of the lactate threshold, 58.3% above 90% of the lactate threshold and 77.3% above 85% of the lactate threshold) (Figure 37-41).

Optimal times in marathon and similar events are achieved by performing at 97 to 100% of lactate threshold (Hagberg, 1984) while events of the 5000-10 000 m type require running speed nearer OBLA (Davis, 1985). Blood lactate does not accumulate to very high levels

during exercise that lasts more than an hour. A good example of this is during marathon running. At the end of a marathon, a trained athlete's blood lactic acid is only two to three times that found at rest (Costill et al., 1967). However, Mognoni et al. (1990) conclude that the effect of blood and muscle lactate on resistance to fatigue decreases rapidly with time. The anaerobic threshold is very often determined in order to obtain the corresponding heart rate value which is used to set the exercise intensity in endurance training so that there is no, or very little, lactic acid accumulation. Thus, the exercise duration should also be kept in mind as lactate increases with prolonged exercise.

Although mean lactate values representing a maximal steady-state during continuous exercise were found to be close to 4 mmol/L, individual values varied from 3 – 5.5 mmol/L (McLellan et al., 1992). Stegman et al. (1981) recognise the extent of this individual variation in maximal lactate steady-state values and introduce the concept of the individual anaerobic threshold (IAT).

There is good evidence that both the lactate VO_2 and the lactate VO_2 (absolute) have a positive correlation with the percentage of time that the heart rate was above 90% and 95% of lactate threshold ($p<0.05$). No significant evidence was found that the corresponding maximum VO_2 had any relation to the percentage of time that the heart rate was above a certain level of lactate threshold ($p>0.05$). Powers et al. (1983) demonstrated that the oxygen uptake measured at the ventilatory threshold was a better predictor of distance running success than either VO_2 max or running economy (Schneider et al., 1991; Louanne et al., 1989).

The following figures indicate the actual heart rate of the athletes over time in the race. The two horizontal lines indicate lactate threshold levels of 85% and 90%.

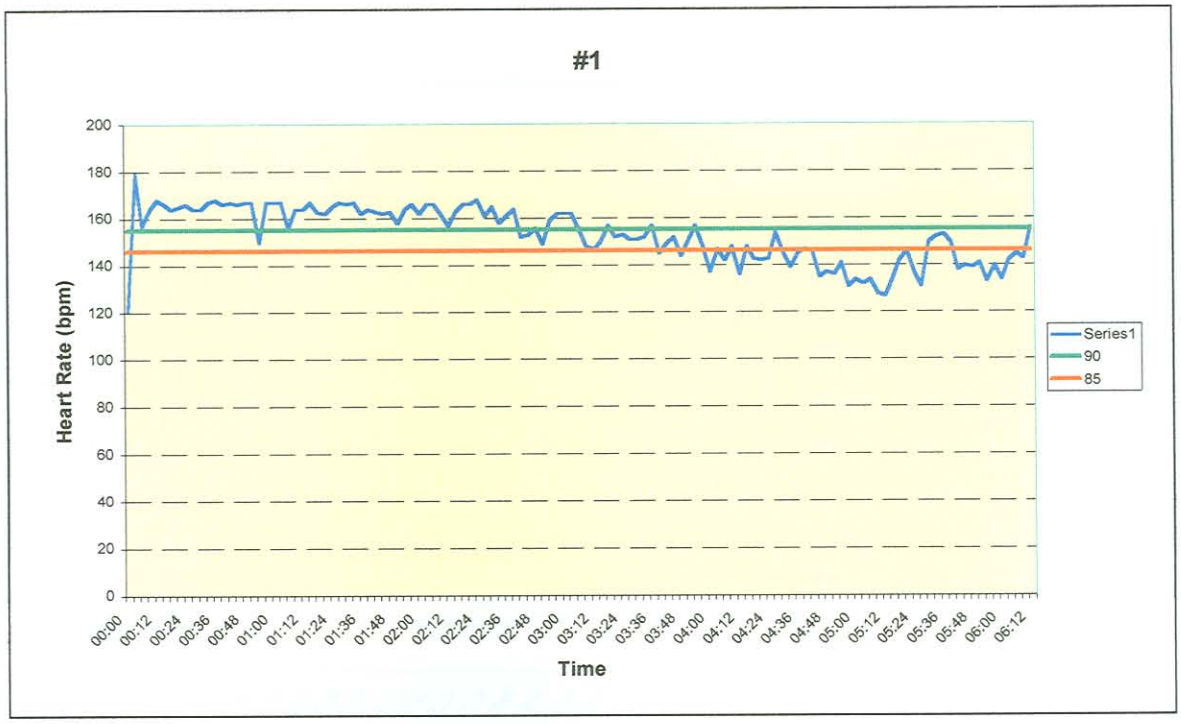


Figure 37: Heart rate response during the Comrades Marathon (athlete 1)

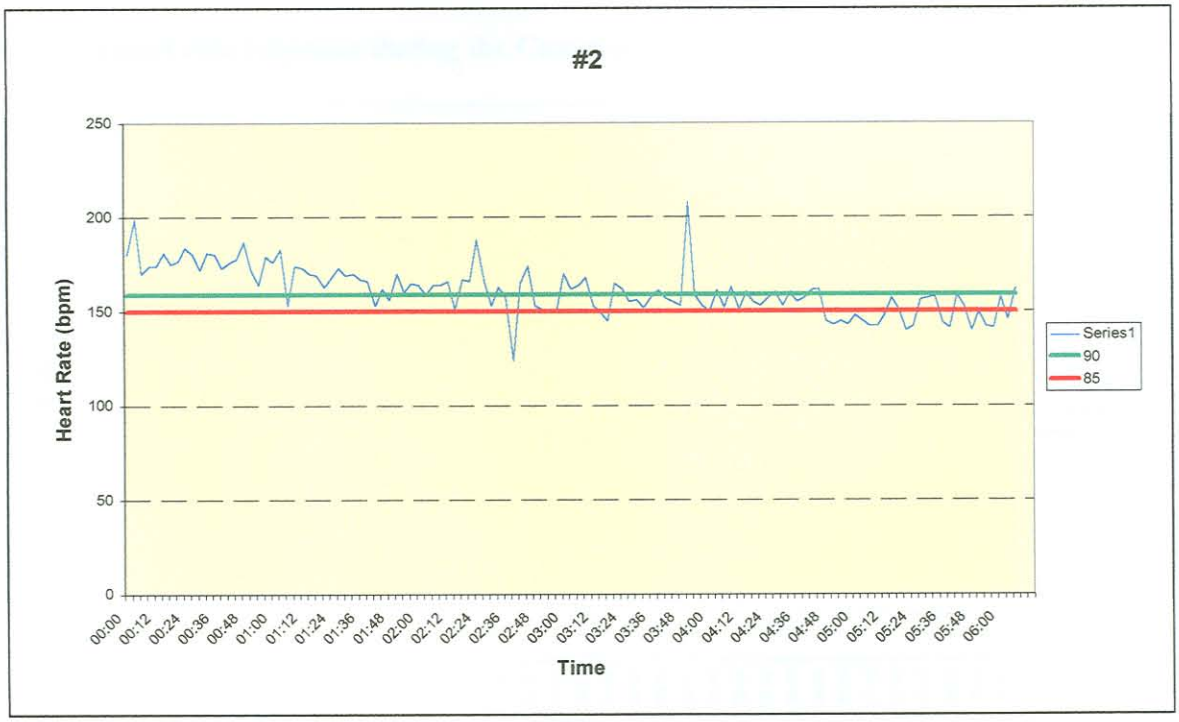


Figure 38: Heart rate response during the Comrades Marathon (athlete 2)

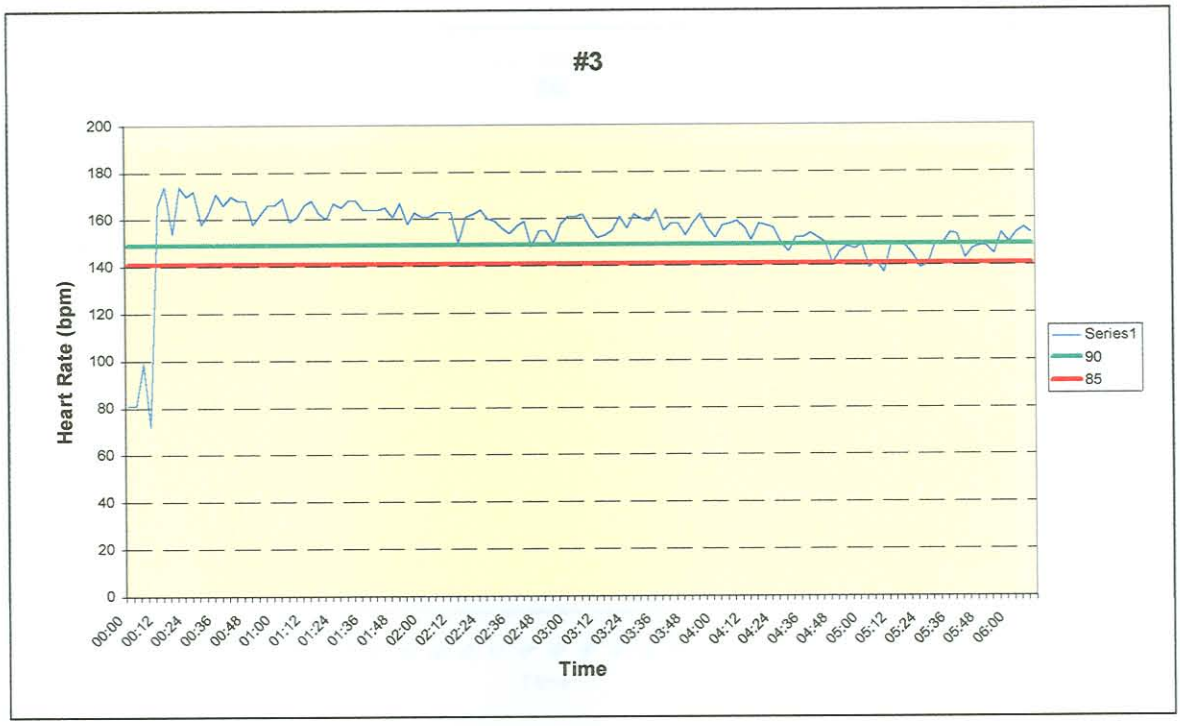


Figure 39: Heart rate response during the Comrades Marathon (athlete 3)

Figure 40: Heart rate response during the Comrades Marathon (athlete 4)

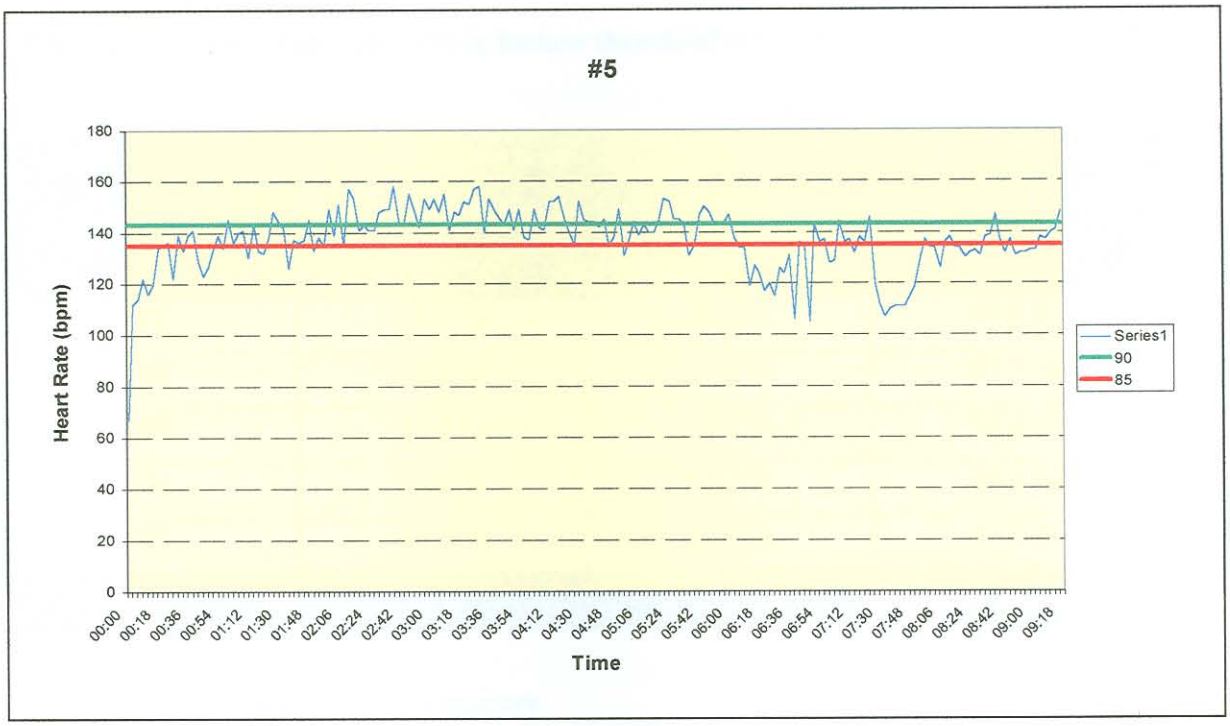


Figure 41: Heart rate response during the Comrades Marathon (athlete 5)

It has been found that the athletes could only keep their heart rate above certain lactate threshold levels for a percentage of the duration of the race. Below (Table 11 and Figure 42) is a summary of the five athletes' ability to keep their heart rate above the given level of lactate threshold.

TABLE 11: Percentage time above lactate threshold intensities of 95%, 90% and 85%

Athlete #	% time above a lactate threshold level of 95%	% time above a lactate threshold level of 90%	%time above a lactate threshold level of 85%
#1	28.8 %	68.8 %	69.6 %
#2	28.5 %	52.0 %	81.3 %
#3	49.6 %	79.2 %	92.0 %
#4	34.7 %	61.3 %	80.3 %
#5	10.2 %	29.9 %	63.1 %
Average	**30.3 %**	**58.3 %**	**77.3 %**

Figure 42 represents the above summary.

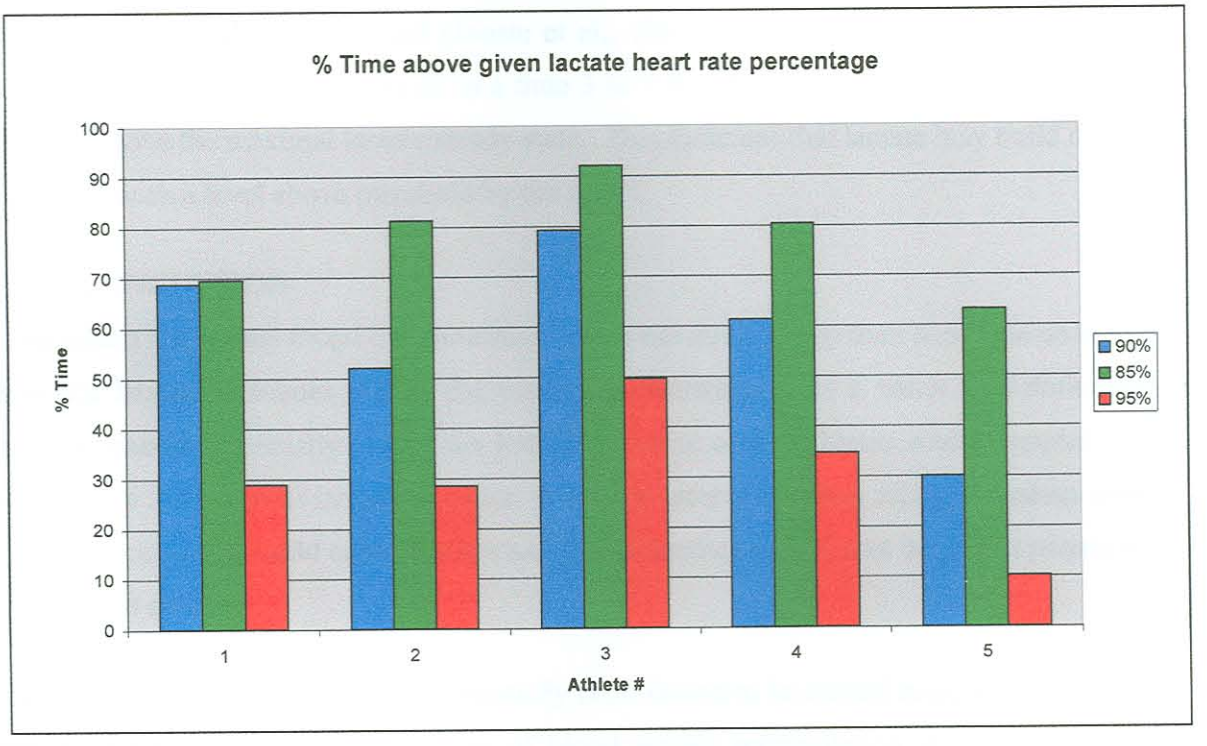


Figure 42: Percentage time above 95, 90 and 85% of lactate threshold heart rate

The relationships between the VO_2 parameters (both lactate and maximum), the actual Comrades time, distance trained and percentage time above a certain lactate threshold level (95%, 90%, 85%), have been tested using Spearman's rank correlation coefficient. It is interesting to see that there is substantial evidence that both the lactate VO_2 and the lactate VO_2 absolute parameters have a positive correlation with the percentage of time that the heart rate was above a certain level of lactate (90% and 95%), but that there was no significant evidence that the corresponding maximum parameters had any relation to the percentage of time that the heart rate was above a certain level of lactate (90% and 95%).

Anaerobic metabolism will probably occur among the Comrades athletes at the start, or while running up hills, but the lactic acid concentration in the blood immediately after a standard marathon (Costill, 1970) and the Comrades Marathon (Jooste et al., 1981) is fairly low. These facts emphasise the conviction that the Comrades Marathon is run mainly on aerobic energy. The pace of each athlete is thus limited largely by his lactic acid turning point and in such a way that this point is not exceeded (Jooste et al., 1981). Palmer et al. (1999) report that marathon runners finished their races in a time 3 to 7 min faster if they had been running at velocities above the maximal lactate steady-state. This indicates that lactate may build during the race to reach a level above threshold by the finish.

4.7 CONCLUSION

In conclusion it has been found that the athletes were not able to keep their heart rate up to just below the lactate threshold during the Comrades Marathon. As a result of deteriorated running economy, especially during the last 20km, none of the athletes could complete the race between 2-4 mmol/L lactate. It seems that more interval training and gymnasium work would be necessary to build enough of the stamina endurance which is an important parameter for Comrades athletes.

Endurance running performance has repeatedly been shown to be related more to submaximal effort measurements, such as the onset of blood lactate accumulation and the anaerobic threshold, than to VO_2 max (Maffulli et al., 1994). Thus, physiological parameters of importance are an improvement at lactate threshold intensity and not at maximum intensity because those parameters simulate the race intensity.

Although running is the world's largest participation sport, most runners have to train alone. Without the benefit of a coach, they have no one to make sure that they are using the most effective training methods; no one to show them how to achieve their maximum running potential. Sport Science can play a vital role in the success of ultra-marathons by helping the athletes to achieve their optimal fitness levels.

The results of the assessment become the basis for prescribing an optimal training programme that concentrates on identified areas of weakness. A testing programme provides feedback. Comparing the athletes' results on a given test item with those of their previous tests provides a basis for assessing the effectiveness of the intervening programme.

CHAPTER 5

CONCLUSIONS AND RECOMMENDATIONS

Laboratory tests are widely used by athletes of many different endurance sports in an effort to guide training, optimise race performance and monitor recovery. The science of sport is particularly exciting and challenging. The extent to which physiological factors are "trainable" as opposed to genetically determined is a topic of considerable debate (Bouchard et al., 1992). Sport Science can play an important role in the success of ultra marathon runners by helping the athletes to achieve their optimal fitness levels. Science has given us a unique insight into the anatomy of one of the most difficult races in the world, the Comrades Marathon.

Many physiological factors are related to successful endurance performance. Maximal oxygen uptake is generally considered to be a useful indicator of successful performance in endurance activities when the subjects are heterogeneous in terms of VO_2 max (Costill et al., 1973; Farrel et al., 1979; Daniels, 1985; Vago et al., 1987; Noakes, 1988; Schneider & Pollack, 1991; Hawley, 1995).

It would appear, however, that the fraction of VO_2 max that an athlete can sustain for prolonged periods is an even better indicator than VO_2 max alone (Costill et al., 1973; Sjodin et al., 1982; Hawley, 1995). Recently, both sports physiologists (Noakes, 1988; Hawley, 1995) and coaches have recognised the importance of peak sustained power output as a predictor of endurance performance.

Other variables include fatigue resistance (Noakes, 1988; Hawley, 1995; Holtzhausen et al., 1996), anaerobic threshold (Vago et al., 1987; McLellan & Jacobs, 1989; Louanne et al., 1989; Keith et al., 1992; Hirokoba et al., 1992; Urhausen et al., 1993; Burke et al., 1994; Hawley, 1995), economy of motion (Hawley, 1995; Brisswalter et al., 1996; McArdle et al., 1996) and fuel utilisation (Vago et al., 1987; Hawley, 1995).

The purpose of the study was to assess experimentally the physiological status of a Comrades Marathon athlete and to examine the effect of training on the physiological parameters. The

testing programme indicates the athletes' strengths and weaknesses in relation to marathon running and provides baseline data for an individual training programme prescription.

Five male marathon athletes volunteered to take part in the study. All the subjects were training for the 1998 Comrades Marathon. They had been following a training programme for a minimum of four years. The first testing occurred eight months before the Comrades; thereafter, another five tests were undertaken. Following an anthropometric evaluation, a maximal incremental treadmill test was undertaken to ascertain the VO_2 max and endurance fitness of the athletes. The physiological parameters measured were oxygen consumption (VO_2 max), maximal heartrate (HR), lactate threshold, ventilatory threshold, respiratory exchange ratio (R), and oxygen pulse. Each subject's best running times for distances of 10, 21, 42, 50, 56 and 90km had been recorded during the previous year. Training distances were also recorded during the testing period. Each subject ran the Comrades Marathon with a Polar Vantage NV to determine race intensity, heartrate response during the race, percentage below the lactate threshold and percentage above the lactate threshold.

It has to be stated that the results obtained from the tests cannot be used to make conclusions about the population (all marathon athletes), because the sample is not big enough or random enough. Therefore, it is a **case study** of five Comrades Marathon athletes, who were training for the 1998 Comrades Marathon.

The anthropometric data did not change much during the training months. The most substantial change could be seen in the fat percentage of the athletes (2.45% decrease). Since energy cost of running is a function of body mass, one way to maximise performance is to reduce excess body fat. Previous studies indicated that lower fat-free body weight is one of the variables primarily characterising the faster endurance runners (Costill, 1967; Housh et al., 1988). With every 1% increase in percentage fat, the VO_2 max is reduced by slightly more than 1% (Londeree, 1986). All of the athletes did, however, show a decrease in LBM (3.37%). Greater body mass in the trunk area appears to be advantageous in terms of running economy. Conversely, those individuals who possess greater percentages of their body mass in the arms and legs may be able to obtain higher VO_2 max values because a greater proportion of their lean muscle mass is active during running (Bailey et al., 1991). Several

studies reported a high relationship between VO_2 max, body composition and endurance performance (Noakes, 1990; Brandon et al., 1995; Brisswalter et al., 1996). Berg et al. (1998) found a strong linear relationship between VO_2 max (L/min) and gross body mass for ectomorphs and mesomorphs, while these two variables are unrelated to endomorphs.

The statistics indicated that some of the VO_2 max parameters changed during the eight-month time period. It has been found, however, that some of the maximum parameters did not change to a great extent (VE/VO_2 = 0.01%, VO_2/HR = 1.3%, VO_2 max = 3.54%, VO_2 absolute = 0.98%, RQ = 0.58%, VT = 4.31%, VE = 0.73%); and speed and heartrate showed a decrease at the maximal exercise intensity (speed = -4.94%, heartrate = -4.37%). There was a greater improvement in parameters measured at threshold level (VE/VO_2 = 1.45%, VO_2/HR = 5.43%, VO_2 max = 5.73%, VO_2 absolute = 6.62%, RQ = 1.70%, VT = 4.19%, VE = 7.78%, speed = 9.10% and heartrate = 6.34%).

Two important physiological variables are important in evaluating distance-running abilities. One is the velocity at anaerobic threshold – the pace at which blood lactate just starts to rise substantially. Marathon pace is slightly slower than this. Once VO_2 max has been raised to as high a level as possible without inordinate additional training volumes, anaerobic development will make the additional difference between being optimally fit and marginally fit. The study of Noakes et al. (1990) shows that the physiological variables determining success at distances from 10-90km are not different, at least in marathon and ultramarathon specialists. This suggests that with appropriate training for longer distance events, the fastest 10km runners will also be the fastest marathon and ultramarathon runners. Athletes 1, 2 and 3 showed a very good speed at the lactate threshold (17.5-, 17.0- and 18.0 km/hr) in May before the Comrades Marathon. This correlates with the Comrades Marathon times that they achieved: 6h12 for athlete 1, 6h06 for athlete 2 and 6h08 for athlete 3. Athletes 4 and 5 indicate lower threshold speeds (16- and 14 km/hr). Their respective times achieved (8h41 and 9h18) were also much lower than those of athletes 1, 2 and 3.

Results indicated a decrease in all the athletes' (1–5) heart rate at maximal intensity and an increase in heart rate at lactate threshold intensity, except for athlete 1 who showed no difference at maximal intensity. Thus, training prescriptions based on heart rate at designated

metabolic markers with subsequent heart rate monitoring will enable coaches and athletes to monitor training intensity accurately. The ability, then, to sustain a high heart rate during prolonged exercise could be hypothesised to be necessary for a fast pace and fast finish time. According to this statement, athletes 1-5 improved their threshold heart rates, which is a very important improvement for Comrades Marathon athletes.

Only athlete 1 indicated a decrease in VE/VO_2 at both lactate threshold (-11.74%) and maximal intensity (-8.03%). Athletes 3 and 4 showed an increase at lactate threshold intensity (13.81% and 10.12%). Athlete 5 showed an improvement only at the lactate threshold intensity (-2.27%). In healthy young adults, this ratio is usually maintained at approximately 25 L during submaximal exercise up to approximately 55% of the oxygen uptake. Thus, a decrease in VE/VO_2 at ventilatory threshold indicates a better oxygen extraction potential, and is therefore advantageous for marathon athletes. Thus, although athletes 3 and 4 indicated a increase in VE/VO_2, al the athletes' (1–5) ventilatory equivalent are still below 30L, which is ideal for marathon runners.

All the athletes showed an increase in the oxygen pulse at lactate threshold and maximal intensity, except for athlete 5. Athlete 5 indicated a lower maximum heartrate (174 – 163 beats per minute) and his training was not very high in distance trained. The athletes' average improvement in lactate threshold is 5.43% compared to the 1.3% at maximal intensity. All the athletes' (1–5) oxygen pulse is within the ideal range of 20 –25 mlO_2/HR.

A very positive improvement in the VO_2 is shown at lactate threshold (5.73%) and maximal intensity (3.54%). Powers et al. (1983) demonstrated that the oxygen uptake measured at the ventilatory threshold was a better predictor of distance running success than either VO_2 max or running economy (Louanne et al., 1989; Schneider et al, 1991). In this context, Yoshida et al. (1992) documented that endurance training induces an improvement of mitochondrial respiratory function resulting in a reduced production of lactate during heavy exercise. According to Yoshida et al. (1992), the positive improvement in the VO_2 max (5.73%) and speed (16.67%) at lactate threshold intensity can be related to their endurance training.

A decrease in the RQ values (at lactate threshold intensity) in marathon athletes is advantageous; thus the decrease in both lactate threshold (-1.7%) and maximal intensity (-

0.58%) is a positive improvement. As a result of high volumes of endurance training and less speed work it is possible to have a decrease in the lactate tolerance and therefore a decrease in RQ values. The athletes speed at lactate threshold had, however, improved; this is an indication of the improvement of their aerobic system.

Lower ventilation, particularly over a prolonged effort (e.g. the Comrades Marathon), would mean, on a ratio basis, less oxygen to the respiratory muscles and more to the working skeletal muscles (Fox et al., 1993). Thus manipulation of the amount of ventilatory work necessary at a given running velocity could alter overall running economy (Bailey et al., 1991). However, tidal volume increased in both maximal (4.31%) and lactate threshold intensity (4.19%). The increase indicates a better percentage lungfilling with exercise at maximum and at lactate threshold intensity. All the athletes' lungfilling was below 65% of vital capacity, thus the improvement in V_T does not affect the running economy negatively.

Minute ventilation showed a small increase in maximal intensity (0.73%) and an increase of 7.78% at lactate threshold intensity. Endurance trained athletes demand a lower V_E than do untrained athletes (Bailey et al., 1991). Athletes 1 and 4 have exercise induced asthma and they indicate the highest minute ventilation (134.5 and 143.8 L respectively). Their values did ,however, improve during their training for the Comrades. The other athletes indicate a relative low V_E (below 120 L). The level of V_E, with its removal of CO_2, thereby serves as the major determinant of arterial H^+ ion concentration during this submaximal long-term work (i.e. at workloads ranging from a long training run to marathon or ultradistance racing). These changes in volume and rate dynamics are controlled automatically to optimise mechanical efficiency while maintaining normal blood O_2 and CO_2 concentrations. Thus, it is unwise for coaches or athletes to attempt voluntary regulation of breathing patterns (Martin & Coe, 1997).

A positive correlation was found between the actual Comrades time ($p<0.05$) and the distance trained. Athlete 2 had the highest training distance (4463km), and completed the Comrades in the fastest time (6h06). Athletes 3, 1, 4 and 5 followed him, in that order. Noakes (1992) states that èlite runners perform best in the marathon and ultramarathon races when they train between 120 to 200 km per week, with an increasing likelihood that they will perform

indifferently when they train more than 200 km per week. None of these athletes trained more than 200 km per week. The studies of Scrimgeour et al. (1986), which report that athletes training less than 60 km a week have as much as 19% less running economy than athletes training more than 100 km a week, might support this suggestion. Most of athlete 5's training distance was below 60 km per week, thus, according to Scrimgeour et al. (1986) it is possible that athlete 5 was less economical than the other runners.

The relationship between the lactate threshold and the actual heartrate response indicated that none of the athletes could complete a 90km race at the lactate threshold intensity. It has been found that the athletes could only keep their heart rate above a certain percentage of the lactate threshold only for the duration of the race (30.3% above 95% of the lactate threshold, 58.3% above 90% of the lactate threshold and 77.3% above 85% of the lactate threshold). As a result of deteriorated running economy, especially during the last 20km, none of the athletes could complete the race between 2-4-mmol/L lactate. Optimal times in marathon and similar events are achieved by performing at 97 to 100% of lactate threshold (Hagberg, 1984). There is good evidence that both the lactate VO_2 and the lactate VO_2 (absolute) has a positive correlation with the percentage of time that the heart rate was above 90% and 95% of lactate threshold ($p<0.05$). No significant evidence was found that the corresponding maximum VO_2 has any relation to the percentage of time that the heart rate was above a certain level of lactate threshold ($p>0.05$). Powers et al. (1983) demonstrated that the oxygen uptake measured at the ventilatory threshold was a better predictor of distance running success than either VO_2 max or running economy (Louanne et al., 1989; Schneider et al, 1991).

Athletes 1, 2 and 3 were in the top 5% of the 1998 Comrades Marathon, and athletes 4 and 5 in the top 19% and 31% respectively. Therefore all the athletes were successful Comrades athletes. A positive correlation was found between the actual Comrades time ($p<0.05$) and the distance trained. Athlete 2 had done the highest trained distance (4463km), and completed the Comrades in the fastest time (6h06). Athletes 3, 1, 4 and 5 followed him, in that order.

It seems that more interval training and gymnasium work would be necessary to build enough of the stamina endurance which is an important parameter for Comrades athletes. Laboratory testing can help the athlete to optimise his running potential. Physiological parameters of

importance are an improvement at lactate threshold intensity and not at maximum intensity because those parameters simulate the race intensity.

The following recommendations are made to expand the knowledge of the physiological parameters of importance for ultra marathon athletes:

- Further adequate tests of experimental modifications of training load need to be made using athletically relevant outcome measures rather than laboratory variables, which, because laboratory conditions feel unnatural to the athlete, can impair the accuracy of the tests. Field testing, which simulates the natural environment for the athlete, should be undertaken as well.

- Effects of different treadmill protocols on peak sustained power output and OBLA. McConnel's (1988) findings suggest that a continuous protocol with workstage durations of 1 minute or less may be the most efficient in terms of testing time for obtaining VO_2 max in runners and may be perceived as being less difficult by the runner.

- The measurement of lactate levels during and at the end of the Comrades Marathon. At the end of a marathon, trained athletes' blood lactic acid is only two to three times that found at rest (Costill et al., 1967).

- An examination of the difference between black and white Comrades athletes with similar VO_2 max values. Bosch et al. (1990) show that subèlite black runners matched with white runners for best 42 km marathon time had slightly lower VO_2 max values than white runners, but compensated for this by sustaining a significantly higher % VO_2 max during the marathon races.

- An examination of a gymnasium programme specially designed for Comrades athletes on the eccentric strength and recovery time when completing the down run. The impact on a downhill can increase as much as eight times the athlete's bodyweight (McGee, 1998). Therefore, the importance of eccentric contraction exercises to improve muscle endurance and decreased risk of injury has to be taken into account in the development of a gymnasium programme.

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