

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 $\text{VO}_2 \text{ max}$ (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 $\text{VO}_2 \text{ max}$** that an athlete can sustain for prolonged periods is an even better indicator than $\text{VO}_2 \text{ max}$ 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 $\text{VO}_2 \text{ max}$

Maximal oxygen consumption ($\text{VO}_2 \text{ max}$) 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_2max 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 (McDougall 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_2max 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_2max 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_2max requires the integration of a high level of ventilatory, cardiovascular, and neuromuscular functions (McArdle et al., 1996). This makes VO_2max a fundamental measure in exercise physiology. Martin & Coe (1997) find a high statistical correlation between aerobic power and competitive performance.

While VO_2max 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_2max 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 not 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 best 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 $\pm 3\%$, 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 $\text{F}_{\text{I}\text{O}_2}$ moderately does not diminish VO_2 max.
- VO_2 max may be alternatively be set 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_{O_2} 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 O₂ pathway contributes in an integrated way to determining VO₂ max, and a reduction the transport capacity of any of the steps will predictably reduce VO₂ max. For instance, a reduction in the inspired PO₂ at altitude will result in a decreased VO₂ max. A reduced hemoglobin level in anemia will result in a decreased VO₂ max. Also a reduction in cardiac output with cardioselective beta-blockade will result in a decreased VO₂ max.

2.3 OXYGEN CONSUMPTION PARAMETERS

In the scientific literature, and increase in VO₂ max is the most common method of demonstrating a training effect. In addition, VO₂ max is frequently used in the development of an exercise prescription. Given these applications of VO₂ max, there has been great interest in identifying the physiological factors that limit VO₂ 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-

atrial (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 hyperlacticaemia. 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.

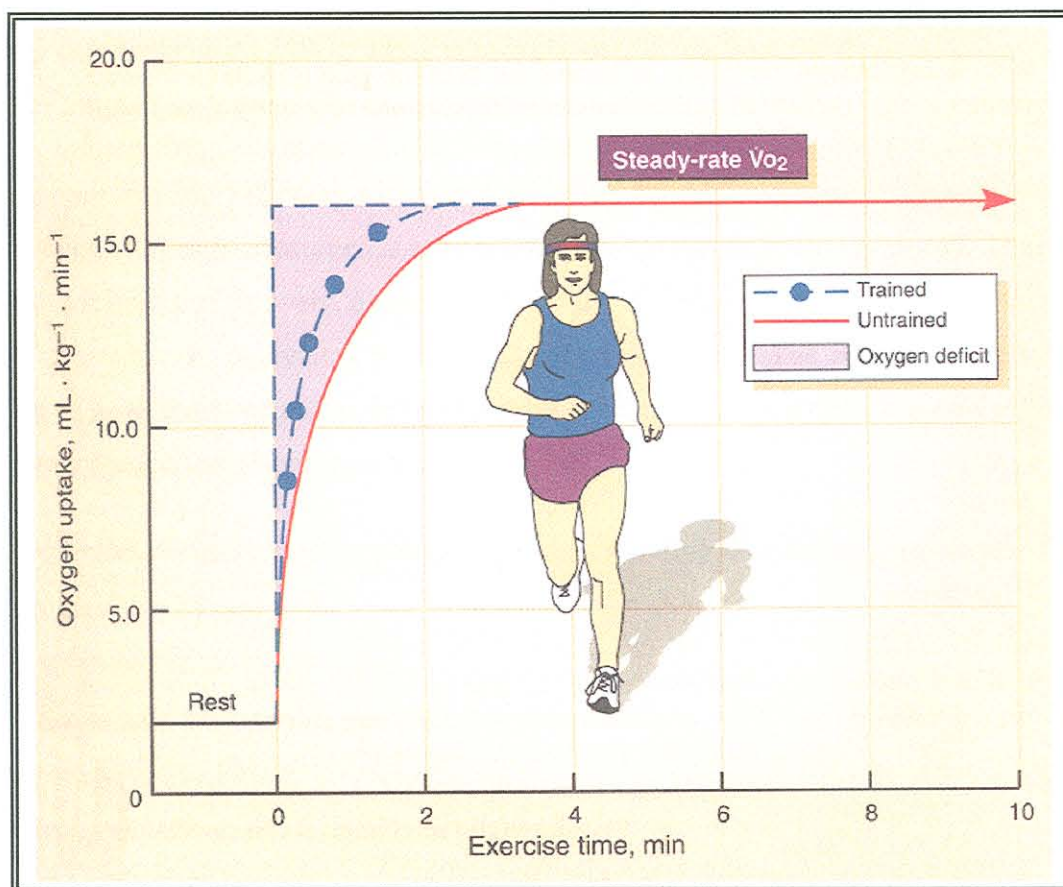


Figure 2: Time course of oxygen uptake during a continuous jog at a relatively slow pace for endurance-trained and untrained individuals who exercise at the same steady-rate VO_2 . The shaded area indicates the oxygen deficit or the quantity of oxygen that would have been consumed had the oxygen uptake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

2.3.3 Oxygen debt (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/V_{O_2})

The ratio of minute ventilation to oxygen uptake is termed the ventilatory equivalent and is symbolised V_E/V_{O_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/V_{O_2} max ratio increases without an accompanying increase in V_E/V_{CO_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 V_{O_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:

$$RQ = \text{CO}_2 \text{ produced} / \text{O}_2 \text{ consumed}$$

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 CO₂ 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 CO₂ 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 CO₂ 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 VO₂ at which there was a breakaway in V_E, an increase in V_E/VO₂ and an initial increase in the percentage of O₂ in the expired air, plotted against time. Martin & Coe (1997) refers to ventilatory threshold when the VCO₂ produced exceeds the VO₂ 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 arterial-mixed 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 to 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 individual's 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

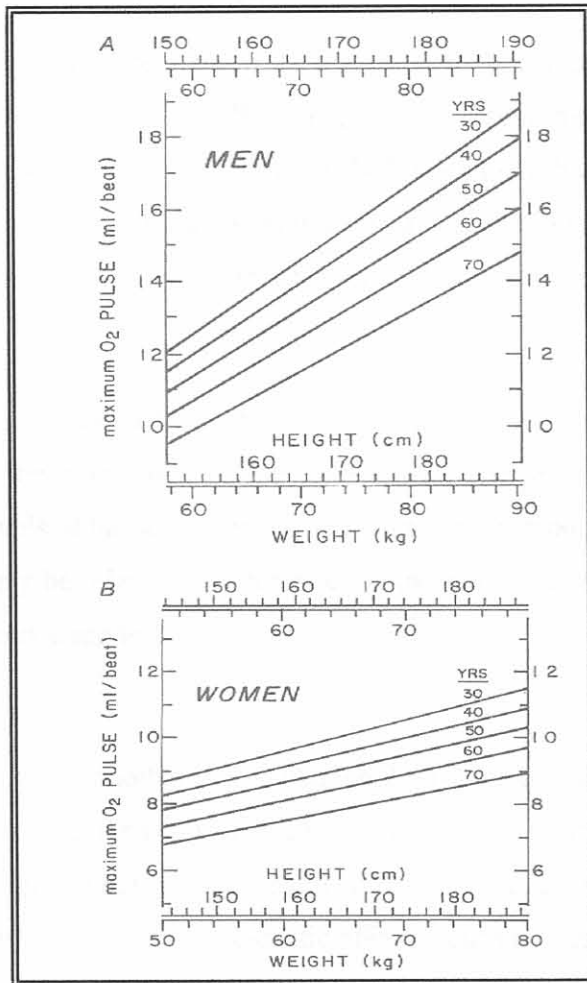


Figure 3: Mean maximum O_2 pulse for sedentary (A) men and (B) women. To use locate on the horizontal axis both the patient's weight and height. From the more leftward point draw a line vertically to the patient's age on the diagonal lines. From this point draw a horizontal line to the vertical axis 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 Rhyne’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).

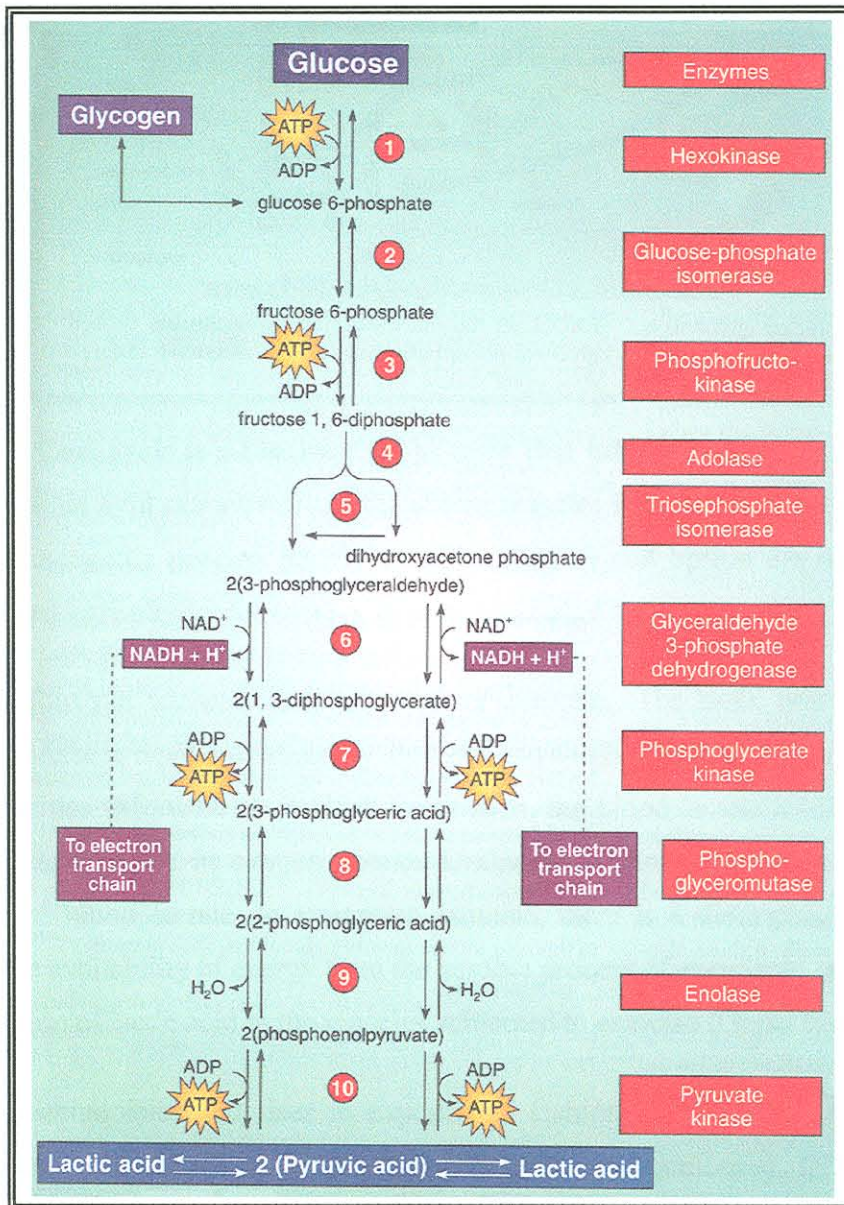


Figure 4: Glycolysis is a series of 10 enzymatically controlled chemical reactions that occur during the anaerobic breakdown of glucose to two molecules of pyruvate. Lactic acid is formed by the process of anaerobic glycolysis when the oxidation of NADH does not keep pace with its formation in glycolysis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

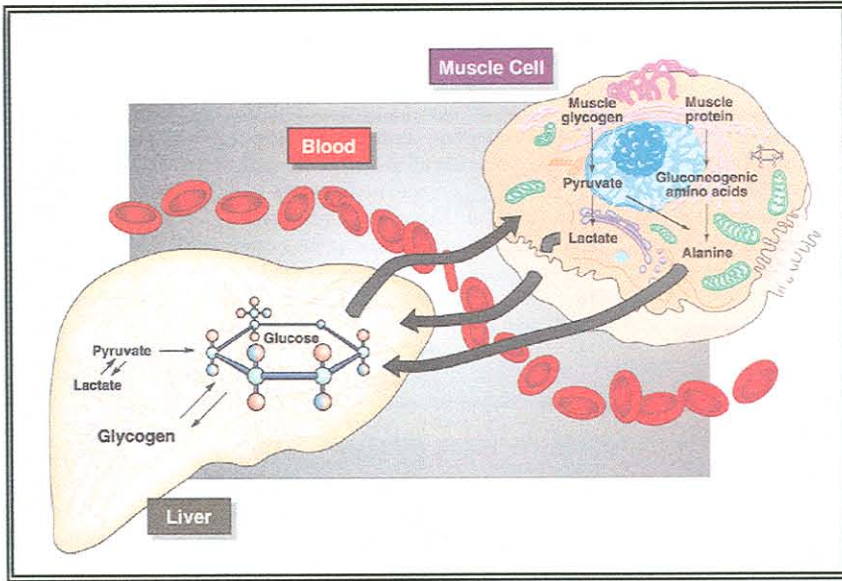


Figure 5: The Cori cycle is a biochemical process that takes place in the liver in which the lactic acid released from the active muscles is synthesised to glucose. This gluconeogenic process provides the body with an option for maintaining its limited carbohydrate reserves (McArdle et al., 1996).

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 it 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).

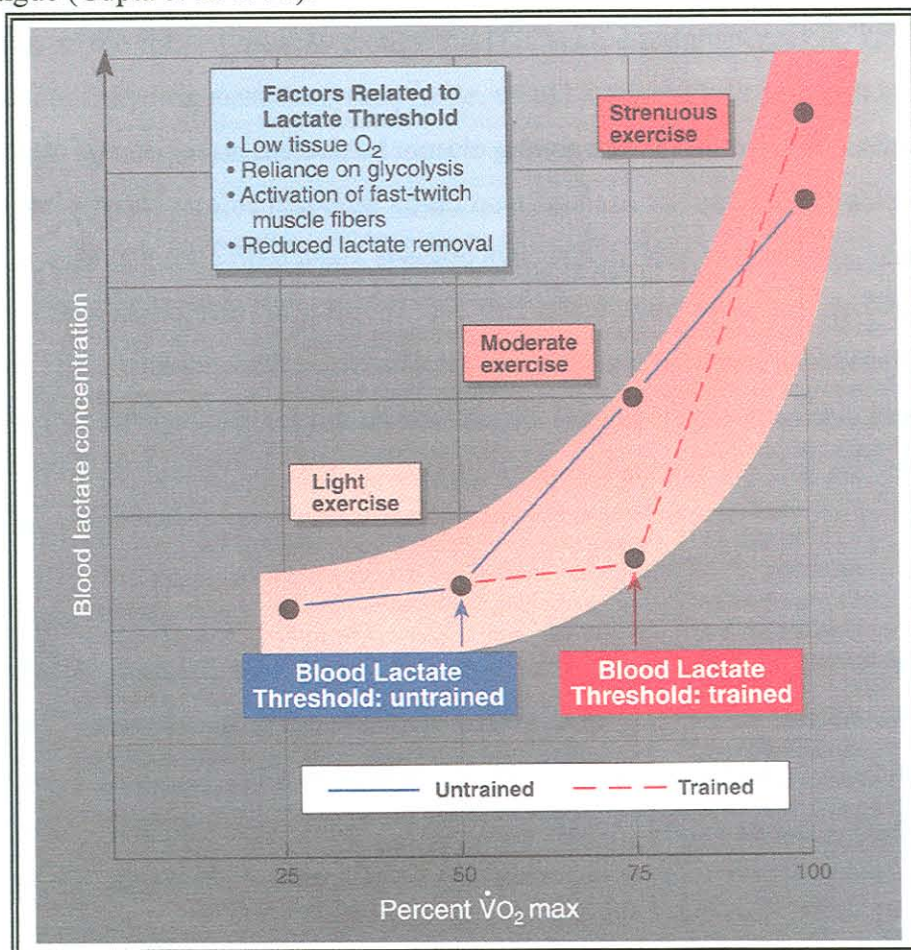


Figure 6: Blood lactate concentration at different levels of exercise expressed as a percentage of maximal oxygen uptake for trained and untrained subjects (McArdle et al., 1996).

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).

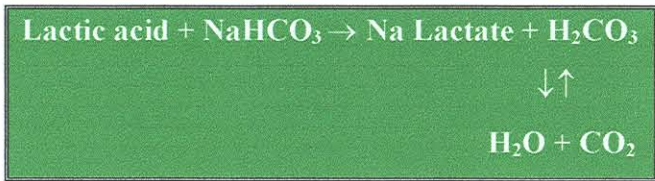
Research into ventilation and the maximal lactate steady-state (MLSS) indicates that ventilation exhibits a threshold phenomenon and that the breathing frequency of 32 breaths/min is the rate most associated with the maximal lactate steady-state (Palmer et al., 1999). Palmer et al. (1999) also reported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



The excess, non-metabolic carbon dioxide released in this reaction stimulates pulmonary ventilation, and CO₂ 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/VO₂ 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 VO₂ max before the onset of blood lactate accumulation occurs (Louanne et al., 1989). Schneider & Pollack, (1991) has defined the anaerobic threshold as the VO₂ 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 VO₂ 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

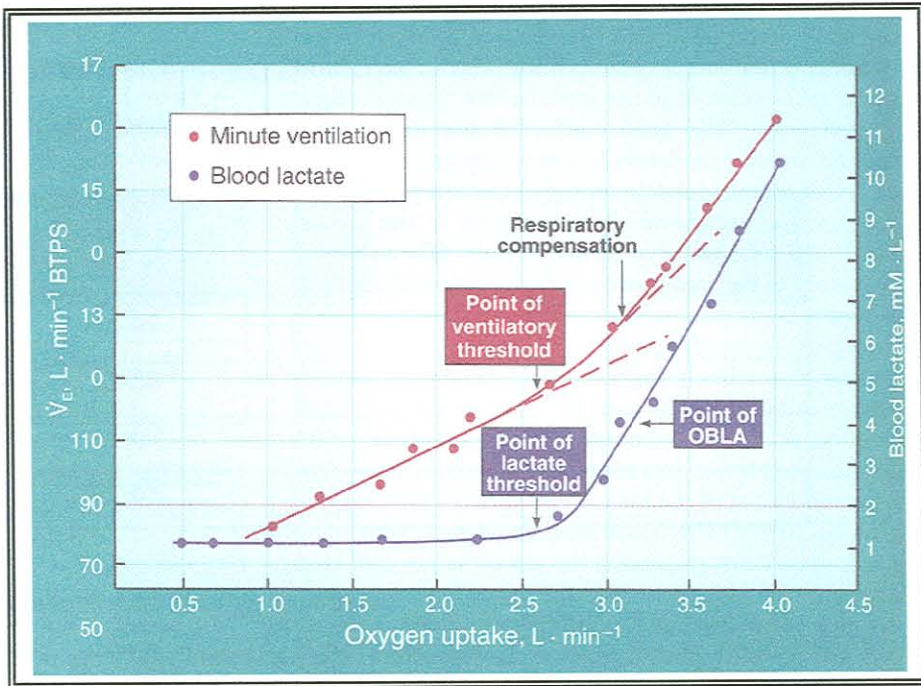


Figure 7: Pulmonary ventilation, blood lactate, and oxygen uptake during graded exercise to maximum (McArdle et al, 1996).

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 CO₂ 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.