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The Effect of Endurance Training on Parameters of Aerobic Fitness

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Abstract

Endurance exercise training results in profound adaptations of the cardiorespiratory and neuromuscular systems that enhance the delivery of oxygen from the atmosphere to the mitochondria and enable a tighter regulation of muscle metabolism. These adaptations effect an improvement in endurance performance that is manifest as a rightward shift in the 'velocity-time curve'. This shift enables athletes to exercise for longer at a given absolute exercise intensity, or to exercise at a higher exercise intensity for a given duration. There are 4 key parameters of aerobic fitness that affect the nature of the velocity-time curve that can be measured in the human athlete. These are the maximal oxygen uptake ($\dot{V}O_{2max}$), exercise economy, the lactate/ventilatory threshold and oxygen uptake kinetics. Other parameters that may help determine endurance performance, and that are related to the other 4 parameters, are the velocity at $\dot{V}O_{2max}$ (V-$\dot{V}O_{2max}$) and the maximal lactate steady state or critical power. This review considers the effect of endurance training on the key parameters of aerobic (endurance) fitness and attempts to relate these changes to the adaptations seen in the body's physiological systems with training. The importance of improvements in the aerobic fitness parameters to the enhancement of endurance performance is highlighted, as are the training methods that may be considered optimal for facilitating such improvements.

The performance of repeated bouts of exercise over a period of time causes numerous physiological changes that result in improved performance in that exercise activity. The magnitude of the training response depends on the duration of the exercise bouts, their intensity and the frequency with which they are performed,[1] along with the initial training status, genetic potential, age and gender of the individual. The specificity of the training stimulus is also important in terms of the type of training practised (endurance, strength or speed) and the exercise modality used.[2] Appropriate recovery periods are required to allow adaptation to the training load: an insufficient training stimulus and/or too much recovery can lead to lack of progress or detraining,[3] while too great a training overload with insufficient recovery can lead to overtraining.[4]

Endurance can be defined as the capacity to sustain a given velocity or power output for the longest possible time. Performance in endurance events is therefore heavily dependant upon the aerobic resynthesis of ATP; this requires an adequate delivery of oxygen from the atmosphere to cytochrome oxidase in the mitochondrial electron transport chain and the supply of fuels in the form of carbohydrates and lipids.[5,6] Endurance can be crudely described through the generation of individual ‘velocity-time curves’ which relate a series of velocities (or power outputs) to the time for which these velocities or power outputs can be sustained.[7,8] Endurance training causes adaptations in the pulmonary, cardiovascular and neuromuscular systems that improve the delivery of oxygen from the atmospheric air to the mitochondria and enhance the control of metabolism within the muscle cells. These adaptations shift the velocity-time curve to the right and therefore result in improved endurance exercise performance. This review will focus on the effect of endurance training on the 4 key parameters of aerobic (endurance) fitness identified by Whipp et al.:[9] the maximal oxygen uptake ($\dot{V}O_{2max}$), exercise economy, the lactate/ventilatory threshold and oxygen uptake kinetics. For the purposes of this review, endurance exercise will be considered to be continuous events of approximately 5 to 240 minutes duration completed at around 65 to 100% of the $\dot{V}O_{2max}$. Events of shorter duration require a significant contribution from anaerobic metabolic pathways,[10] while events of longer duration may be limited by psychological, nutritional, thermoregulatory or musculoskeletal factors rather than by ‘endurance fitness’, *per se*.

1. Maximal Oxygen Uptake ($\dot{V}O_{2max}$)

$\dot{V}O_{2max}$, which reflects an individual’s maximal rate of aerobic energy expenditure, has long been associated with success in endurance sports.[11,12] In whole-body exercise such as running, cycling and rowing, it is widely accepted that $\dot{V}O_{2max}$ is limited by the rate at which oxygen can be supplied to the muscles and not by the muscle’s ability to extract oxygen from the blood it receives.[13] The $\dot{V}O_{2max}$ appears to be strongly related to the maximal cardiac output (Q_{max}). The high Q_{max} and $\dot{V}O_{2max}$ values commonly found in elite athletes are, in turn, related to very high maximal stroke volumes since maximal heart rates tend to be similar to those of sedentary individuals.[14] Following training, exercising muscle may require less blood flow for the same submaximal exercise intensity because of an increase in the arterio-venous oxygen difference.[15] The increased stroke volume resulting from increases in left ventricular size, myocardial contractility and end-diastolic volume with training, along with a decreased sensitivity to catecholamines, leads to a reduced heart rate during submaximal exercise.[16] During maximal exercise, the greater cardiac output, along with an increased extraction of oxygen by the exercising muscle, results in a greater $\dot{V}O_{2max}$.[16,17] In addition, the oxygen carrying capacity of the blood is increased following endurance training owing to an increased total blood haemoglobin content. There is also an increase in red cell 2,3-diphosphoglycerate which offsets the reduced haemoglobin concentration consequent to the relatively larger increase in plasma volume compared to red cell mass.[18] The lower [Hb] following training may be advantageous in that the reduced blood viscosity may reduce the resistance of the vasculature to blood flow.

The magnitude of the increase in $\dot{V}O_{2max}$ resulting from endurance training depends on a number of factors, notably the initial fitness status of the individual, the duration of the training programme and the intensity, duration and frequency of the individual training sessions.[1] Since most studies of endurance training have shown some increase in $\dot{V}O_{2max}$ with time, the optimal exercise volume and intensity for developing this parameter is not known. However, there is some evidence from the literature to suggest that a high intensity of training (approximately 80 to 100% of $\dot{V}O_{2max}$) may be of crucial importance provided that the minimal training volume for a particular event is covered.[1,19] In a recent study,[20] we examined the influence of 6 weeks of endurance training on parameters of aerobic fitness in 16 physical education students. Despite the relatively modest training programme (3 to 5 sessions per week of 20 to 30 minutes duration at a running speed close to the lactate threshold), we found that $\dot{V}O_{2max}$ increased by approximately 10% (from 47.9 ± 8.4 to 52.2 ± 2.7 mg/kg/min). Other groups[21-27] have also shown a 5 to 10% improvement in $\dot{V}O_{2max}$ with short term endurance training programmes. Hickson et al.[28] reported that $\dot{V}O_{2max}$ increased by 23% over 9 weeks of endurance training, but the majority of this increase (14%) occurred after only 3 weeks. This rapid increase in $\dot{V}O_{2max}$ and the similarly rapid reduction in submaximal exercise heart rate have been partly attributed to an early hypervolaemia which will increase stroke volume during exercise and also afford an increased tolerance to heat stress.[29,30] There is some evidence that during longer term training programmes, $\dot{V}O_{2max}$ will eventually stabilise, with subsequent improvements in performance resulting from continued improvements in submaximal factors such as exercise economy and lactate threshold.[2,31-33]

2. Exercise Economy

Exercise economy has been defined as the oxygen uptake required at a given absolute exercise intensity. There is considerable interindividual variability in the oxygen cost of submaximal exercise, even in individuals of similar aerobic fitness (defined as $\dot{V}O_{2max}$) or similar performance capability.[34-36] For example, Horowitz et al.[37] demonstrated that elite cyclists exercising at the same power output required different rates of oxygen uptake. Interestingly, the more efficient cyclists had a greater percentage of type I fibres in the vastus lateralis, suggesting that the pattern of motor unit recruitment during exercise may be important in the determination of economy. In a classic study, Conley and Krahenbuhl[34] reported that 10km race performance was closely related to running economy in a group of well-trained volunteers who had similarly high $\dot{V}O_{2max}$ values. Better exercise economy (i.e. lower $\dot{V}O_2$ for a given absolute running speed or power output) can be considered to be advantageous to endurance performance because it will result in the utilisation of a lower percentage of the $\dot{V}O_{2max}$ for any particular exercise intensity. It has been suggested that the relatively low $\dot{V}O_{2max}$ scores that have been reported in some elite endurance athletes can be compensated for by exceptional exercise economy.[38,39] Indeed, an inverse relationship between $\dot{V}O_{2max}$ and running economy has been reported in samples of well-trained runners.[40,41]

Although trained athletes are known to have better exercise economy than untrained individuals,[39] studies that have examined the effect of endurance training on exercise economy have produced equivocal results.[42-45] This may be because such training studies (typically of 6 to 12 weeks duration) are too short to produce a measurable improvement in economy, especially in individuals who are already trained. It may be speculated that good exercise economy is somehow related to the total volume of endurance training performed, since the best economy values are often found in older or more experienced athletes, or those who complete a large weekly training mileage.[33,40,42] Furthermore, athletes' most economical velocities or power outputs tend to be those at which they habitually train (unpublished data). This may indicate that athletes should train over a wide variety of speeds if they wish to lower the slope of the $\dot{V}O_2$-exercise intensity relationship. Only a few studies have tracked changes in exercise economy over a prolonged period of training.[33,40,46,47] In one such study that measured changes in a number of physiological variables over a 5-year period in an elite female distance runner,[33] it was reported that running economy improved appreciably with each year of training. For example, the $\dot{V}O_2$ at a running speed of 16.0 km/h decreased from 53.0 ml/kg/min in 1992 to 47.6 ml/kg/min in 1995. However, improvements in running economy can sometimes be observed even with short term training programmes.[26,27,48] In a recent study, we found that 6 weeks of endurance running training caused a significant improvement in running economy in 16 recreationally active individuals (fig. 1),[48] with the $\dot{V}O_2$ at a representative running speed of 12.0 km/h decreasing from approximately 39 ml/kg/min to approximately 36 ml/kg/min. Franch et al.[26] also reported that the running economy of trained volunteers could be reduced significantly following 6 weeks of high intensity distance running or long-interval training, and found that the

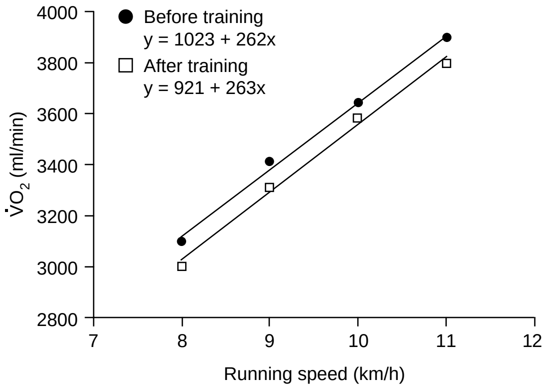

Fig. 1. The effect of 6 weeks of endurance training on submaximal oxygen uptake ($\dot{V}O_2$) [running economy]. The data represent the mean response of 16 individuals (from Jones et al.,[48] with permission).

reduction in submaximal $\dot{V}O_2$ was significantly correlated with the reduction in minute ventilation ($\dot{V}_E$).

Running economy has been associated with anthropometric (including segmental mass distribution), physiological and metabolic, and biomechanical and technical factors.[49] Improvements in exercise economy with endurance training may result from improved muscle oxidative capacity and associated changes in motor unit recruitment patterns,[50] reductions in exercise ventilation and heart rate for the same exercise intensity,[26] and improved technique.[51] These improvements may be partly offset by an increased utilisation of fat as exercise substrate following training due to the greater amount of oxygen that is required for the resynthesis of ATP from fat metabolism compared to carbohydrate metabolism. Of interest is the possibility that exercise economy is related to muscle elasticity. It has been speculated that running economy might be related to 'fluency' of movement and that it might therefore be improved by flexibility training.[52,53] However, recent observations from our laboratory suggest that the oxygen cost of running at 16.0 km/h is negatively related to lower limb flexibility (estimated with the sit-and-reach test) in 26 international-standard male distance runners, i.e. 'stiffer' runners were more economical.[54] Similar results can be found in the literature.[55,56] One explanation for these results is that stiffer muscles and tendons are better able to store elastic energy during the eccentric phase of stretch-shortening activities and that this stored energy can be released during the concentric phase of the action, thus lowering the oxygen cost of the exercise.[57] Alternatively, inflexibility in the trunk and hip may stabilise the pelvis during the stance phase and limit the requirement for stabilising muscular activity.[56]

It has been suggested that increasing maximal leg strength through resistance training may improve economy and endurance performance by reducing the proportion of the maximal force required for each contraction (e.g. pedal thrust) and hence delaying the recruitment of type II motor units.[58] However, traditional resistance training programmes which involve lifting moderate to high loads at relatively slow movement speeds have, with some exceptions,[58,59] been shown to be ineffective in improving endurance performance.[60,61] However, of great interest is a recent study which demonstrated that 'explosive strength training', involving sprinting and jumping exercises and weight training using high to maximal movement speeds and low loads (0 to 40% of the 1-repetition maximum), can improve both running economy and 5km race performance.[62] The authors suggested that the improved neuromuscular control resulting from the training could have improved running economy by allowing a tighter regulation of muscle stiffness and better utilisation of muscle elasticity. It is also possible that strength training using maximal velocity contractions may improve economy by allowing for a better recruitment of motor units or a reduced co-contraction of antagonistic muscle groups.[63] One other study has demonstrated a similar effect of explosive strength training on the economy of cross-country skiers.[64] Clearly, additional research is required to confirm and extend these findings.

3. Interaction Between $\dot{V}O_{2max}$ and Economy

The locomotory velocity associated with $\dot{V}O_{2max}$ (V-$\dot{V}O_{2max}$), which is a function of individual $\dot{V}O_{2max}$ and exercise economy characteristics and which can be calculated by solving the regression equation describing the relationship between $\dot{V}O_2$ and submaximal exercise intensity for $\dot{V}O_{2max}$, has been shown to be an important determinant of endurance exercise performance.[65-68] Morgan et al.[66] reported that the running speed at $\dot{V}O_{2max}$ strongly predicted 10km running performance in a group of well trained male runners with homogeneous $\dot{V}O_{2max}$ values (approximately 65 ml/kg/min). Jones and Doust[69] presented a comprehensive battery of physiological tests to 13 trained runners with a wide range of $\dot{V}O_{2max}$ values (53 to 67 ml/kg/min), and reported that V-$\dot{V}O_{2max}$ correlated more strongly with 8km running performance (r = 0.93) than any of the other measures, including $\dot{V}O_{2max}$ (r = 0.69) and running economy (r = –0.16). Although they are closely related, the V-$\dot{V}O_{2max}$ should not be confused with the maximal velocity reached in a fast incremental treadmill test ($\dot{V}_{max}$).[70] Although some studies have shown that the $\dot{V}_{max}$ correlates highly with endurance exercise performance,[70,71] $\dot{V}_{max}$ is influenced not just by $\dot{V}O_{2max}$ and exercise economy factors but also by anaerobic capability, muscle power and neuromuscular skill in exercising at high speeds.

Several studies have shown an increased V-$\dot{V}O_{2max}$ following endurance training. Jones[33] reported that V-$\dot{V}O_{2max}$ increased from 19.0 to 20.4 km/h over a 5-year period in an elite female distance runner. This improvement in V-$\dot{V}O_{2max}$ was the result of an improved running economy because $\dot{V}O_{2max}$ fell slightly over the same period of time. The V-$\dot{V}O_{2max}$ is similar to the velocity that can be sustained during distance running races of 3000m (approximately 8 minutes in the elite athlete),[33] and so this parameter may be especially important for success in middle-distance events. Billat et al.[27] reported that only 4 weeks of normal training caused a significant improvement in running economy and V-$\dot{V}O_{2max}$ (from 20.5 to 21.1 km/h), with no significant change in $\dot{V}O_{2max}$ (from 71.2 to 72.7 ml/kg/min), in 8 trained males. Berthoin et al.[72] reported that V-$\dot{V}O_{2max}$ was significantly improved only with high intensity training in adolescent volunteers. In another study, Jones et al.[48] found that 6 weeks of endurance training increased V-$\dot{V}O_{2max}$ from 15.3 to 16.6 km/h in 16 volunteers, with the increased V-$\dot{V}O_{2max}$ resulting from significant improvements in both $\dot{V}O_{2max}$ and running economy. The V-$\dot{V}O_{2max}$ appears to be an important and sensitive measure of endurance fitness and can be usefully measured during longitudinal work with endurance athletes.[33,66] An improvement in the V-$\dot{V}O_{2max}$ with training will mean that certain percentages of the $\dot{V}O_{2max}$ will be associated with higher speeds after training. This may be important in the improvement of endurance race performance because athletes tend to operate at quite similar percentages of $\dot{V}O_{2max}$ for a given duration of exercise.[5,6,73] However, while the V-$\dot{V}O_{2max}$ construct is practically useful, great care should be taken in its measurement. This is because $\dot{V}O_{2max}$ may be achieved during constant-load exercise over a wide range of submaximal exercise intensities above the 'critical power' because of the upward drift in oxygen uptake with time (see section 5).[74-76] Therefore, for the accurate determination of V-$\dot{V}O_{2max}$ there is a requirement both for a valid measure of $\dot{V}O_{2max}$ and for exercise economy to be measured at several moderate intensities below the lactate threshold.

It has been suggested that the V-$\dot{V}O_{2max}$ might represent an optimal training stimulus for improvements in endurance fitness.[77-81] Hill and Rowell[81] contend that training at V-$\dot{V}O_{2max}$ is important because V-$\dot{V}O_{2max}$ is the lowest speed that will elicit $\dot{V}O_{2max}$ and it is necessary to train at $\dot{V}O_{2max}$ to improve it. A concept that is closely related to the V-$\dot{V}O_{2max}$ is the time for which exercise at V-$\dot{V}O_{2max}$ can be sustained (T_{max}).[82] It has been shown that training at 100% V-$\dot{V}O_{2max}$ allows exercise at $\dot{V}O_{2max}$ to be sustained for the longest possible time (approximately 4 to 8 minutes).[82] Hill and Rowell[81] demonstrated that if interval or repetition sessions are constructed with the goal of allowing the longest possible training time at V-$\dot{V}O_{2max}$, then each repetition needed to be longer than 60% of T_{max}. Recently, it was shown that a 4-week training programme which included 2 interval training sessions per week (6 repetitions at V-$\dot{V}O_{2max}$ intensity for an exercise duration of 60 to 75% of the pre-training T_{max}) resulted in significant improvements in

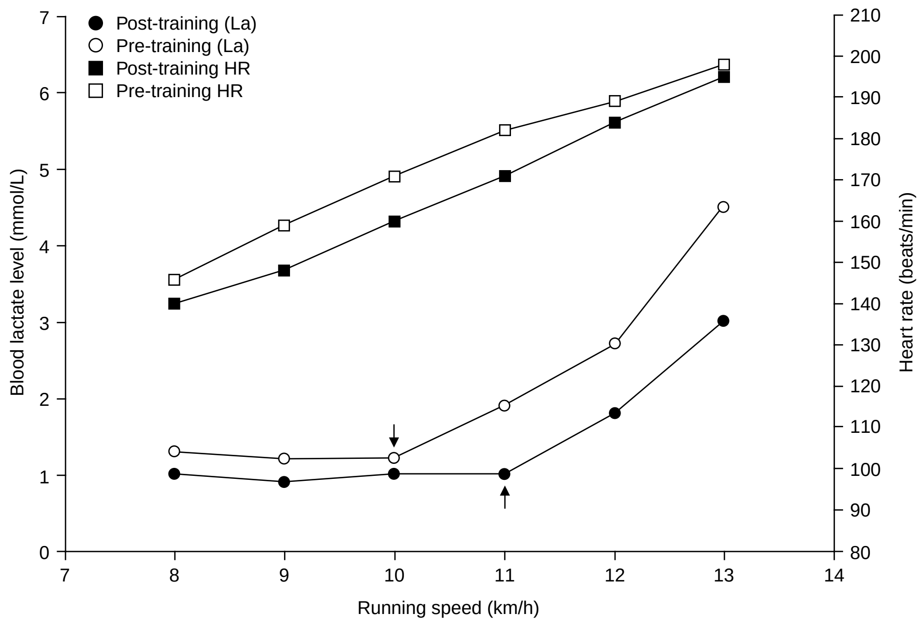

Fig. 2. The effect of 6 weeks of endurance training on blood lactate levels and heart rate response to incremental exercise in a typical individual. The vertical arrows denote the lactate threshold determined before and after training (from Carter et al.,[20] with permission).

$\dot{V}O_{2max}$, V-$\dot{V}O_{2max}$, T_{max} and 3000m performance in trained runners.[83] Unfortunately, this study did not have a control group, and additional studies are needed to confirm the value of using V-$\dot{V}O_{2max}$ to set training intensity and T_{max} to set training duration when the goal is to improve the V-$\dot{V}O_{2max}$.

4. Lactate/Ventilatory Threshold

The exercise intensity corresponding to the increase in blood lactate above resting levels (lactate threshold; LT) and the associated changes in gas exchange (ventilatory threshold; VT) are powerful predictors of endurance performance.[35,69,84-88] Numerous studies also testify to the sensitivity of the LT and VT to endurance training (fig. 2).[20,89-93] A rightward shift of the LT/VT to a higher power output or running speed is characteristic of successful endurance training programmes.[94] This adaptation allows a higher absolute (running speed or power output) and relative (% $\dot{V}O_{2max}$) exercise intensity to be sustained without the accumulation of blood lactate after training. Endurance training is also associated with a reduction in the degree of lactacidaemia for any given absolute or relative exercise intensity. This causes the power output or running speed corresponding to arbitrary 'blood lactate reference values' such as 4 mmol/L blood lactate to increase following a period of endurance training.[20,93,95-98] Exercise above the LT is associated with a nonlinear increase in metabolic, respiratory and perceptual stress.[99,100] Furthermore, exercise above the LT is associated with more rapid fatigue, either through the effects of metabolic acidosis on contractile function[101] or through an accelerated depletion of muscle glycogen.[102] Therefore, an improvement in the LT/VT with training is a clear marker of an enhanced endurance capacity. However, it should be noted that the LT/VT is typically found at 50 to 80% $\dot{V}O_{2max}$ even in highly trained individuals, and it therefore occurs at a lower exercise intensity than is maintained by endurance athletes during most forms of endurance competition. The maximal lactate steady state (MLSS), which is the highest exercise intensity at which blood lactate does not accumulate over time (see section 5), may be of more importance to success in these events.

Mader[103] proposed that the precision with which training loads can be applied may be improved through individual consideration of the LT. Several authors have hypothesised that the LT represents the optimal intensity for improvement of endurance fitness.[103,104] Training at the LT should provide a high quality aerobic training stimulus without the accumulation of lactate that would compromise training duration.[105,106] Anecdotally, endurance athletes and coaches feel that training at LT through the inclusion of a regular 'threshold' or 'tempo' training session is a critical component of a balanced training programme.[107] The effect of training intensity on improvements in the LT/VT has recently been reviewed.[108] In general, it appears that training at intensities close to or slightly above the existing LT/VT is important in eliciting significant improvements in this parameter.[20,92,93,109-111] For example, it was reported that increasing training intensity through the use of fartlek training on 3 days per week,[110] or adding a 20 minute run at LT speed to the weekly training programme,[109] caused an improvement in the LT with no change in $\dot{V}O_{2max}$ in runners. Henritze et al.[92] reported that training at intensities above the LT may be even more effective for improving the LT, while Keith et al.[111] have shown that continuous training at the LT or intermittent training above and below the LT are equally effective in improving LT. Collectively, these studies indicate that exercise training at an appropriately high intensity might be most effective in stimulating improvements in LT and performance.

The reduction in blood lactate for the same absolute and relative exercise intensities following endurance training may result from a reduction in the rate of lactate production (possibly consequent to a lower rate of muscle glycogen utilisation or to speeded oxygen uptake kinetics that may increase initial O_2 availability/utilisation),[112,113] or from an increase in the ability to exchange and remove lactate from the blood.[114-116] Elite endurance athletes have a predominance of type I ('slow-twitch') muscle fibres in the trained musculature when compared to their sedentary peers.[117] This is of interest because of the strong relationship that is known to exist between the percentage of type I muscle fibres and the LT.[118-120] Endurance training causes a selective hypertrophy of the type I fibres and it is possible that a transformation of muscle fibre types from type IIb to type IIa,[23,121] and even from type IIa to type I[122,123] can eventually occur. There is also evidence that endurance training can cause an increased expression of slow myosin in type II fibres which reduces the maximal shortening speed in these fibres.[124] Conversely, detraining and micro-gravity lead to a reduction in the expression of slow myosin in muscle fibres.[125] The increased capillarity of skeletal muscle with endurance training[121,126] has the effect of increasing both the maximal muscle blood flow capacity and the surface area available for exchange of gases, substrates and metabolites between blood and muscle. The longer mean transit time for red blood cells to pass through the muscle capillary bed will increase the time available for diffusion of oxygen from the red blood cell and increase the potential for widening the arterial-venous oxygen difference during exercise.

Endurance training results in numerous adaptations within skeletal muscle that may be significant for exercise performance, including increases in sodium-potassium pump concentration,[127] lactate transport capacity[128,129] and possibly myoglobin concentration.[130] Endurance training also results in a marked increase in the oxidative capacity of skeletal muscle. This is due to an increase in the size and the number of mitochondria per unit area and an increase in the concentration of the enzymes of the Krebs cycle, electron transport chain and malate-aspartate shuttle.[23,131,132] These adaptations help maintain cellular phosphorylation potential, improve the sensitivity of respiratory control and increase the capacity for aerobic ATP resynthesis during exercise in both type I and type II muscle.[133,134] Muscle respiratory capacity is highly correlated with LT and these enzymatic adaptations may be important in allowing an athlete to exercise at a high percentage of $\dot{V}O_{2max}$ for prolonged periods.[118,119] It is possible that a greater oxidative enzyme complement in type I muscle fibres might delay the point at which the type II muscle fibres are recruited during exercise.[135] Furthermore, an increase in the oxidative potential of the type II fibres might reduce their reliance on anaerobic glycolysis for ATP production.[133] Animal studies suggest that low intensity training (approximately 50% $\dot{V}O_{2max}$) may be sufficient to maximise the increase in mitochondria in type I muscle, but that much higher intensities are needed to cause significant increases in mitochondrial volume in type II muscle.[130,136]

The greater capacity of the Krebs cycle to accept pyruvate following training may be important in reducing the production of lactate by mass action at the onset of exercise and during high intensity exercise.[137] However, the greater capillarity of trained muscle also allows for a greater uptake of free fatty acids from the blood and the increased activity of the enzymes involved in lipid metabolism increase the capacity for mitochondrial B-oxidation.[138] It has been shown that there is a reduction in the rate of glycogen depletion,[139,140] a decreased production and oxidation of blood-borne glucose[141,142] and an increased storage and rate of utilisation of intramuscular triacylglycerol following training.[143,144] The greater use of lipid during submaximal exercise, which can be documented in the lower respiratory exchange ratios found for the same absolute and relative exercise intensity following training, reduces the contribution of carbohydrate to ATP resynthesis and is therefore important in sparing muscle glycogen.[138] This adaptation, along with evidence that endurance training increases the storage of muscle glycogen,[145,146] is an important adaptation to endurance training because a depletion of muscle glycogen stores have been linked to fatigue during endurance exercise.[147]

The hormonal response to exercise appears to change rather quickly following the onset of endurance training.[141,148] For example, the catecholamine response appears to be substantially blunted for the same exercise intensity after only a few days of training.[142,148] Since adrenaline is a major effector of lactate production through its modulation of muscle glycogenolysis, this may partly account for the reduction in muscle glycogen utilisation seen with endurance training.[149] The reduced sympathetic nervous system activity may also contribute to the reduction in heart rate observed for the same exercise intensity following training.[16]

5. Oxygen Uptake Kinetics

At the onset of 'moderate' exercise (that is, exercise that is below the LT) pulmonary oxygen uptake increases mono-exponentially to achieve a new steady state within 2 to 3 minutes. For constant-intensity exercise in this domain, the oxygen deficit that is incurred at the onset of exercise may cause blood lactate to rise transiently before it returns to resting levels as exercise proceeds. On the other hand, the imposition of an exercise challenge that is just above the LT causes blood lactate to rise until it attains a steady state level that is higher than the resting concentration. In this exercise domain, pulmonary $\dot{V}O_2$ will also attain a delayed steady state but the $\dot{V}O_2$ that is achieved may be higher than would be predicted based upon the relationship between $\dot{V}O_2$ and exercise intensity for moderate exercise.[150] The MLSS can be defined as the highest running speed or power output at which blood lactate remains stable or increases only minimally ($<$ 1.0 mmol/L) between 10 and 30 minutes of exercise.[69,151] The MLSS therefore demarcates the highest exercise intensity at which a balance exists between the appearance of lactate in the blood and the removal of lactate from the blood during long term exercise, and is perhaps the 'gold standard' measure of endurance exercise capacity. In theory, the MLSS is the same as the concept of 'critical power' (CP)[74,76,152] or 'critical velocity'[153,154] that is represented by the asymptote of the hyperbolic relationship between exercise intensity and time to exhaustion. Submaximal exercise above the CP/MLSS is associated with an inexorable increase in blood lactate, pulmonary ventilation, and $\dot{V}O_2$ with time, and depending on the exercise intensity, $\dot{V}O_2$ may even rise to attain $\dot{V}O_{2max}$.[74,76,155] This 'drift' in

$\dot{V}O_2$ during constant-load exercise to values that are greater than might be expected has been termed the $\dot{V}O_2$ slow component. While the mechanisms responsible for this apparent metabolic inefficiency during high intensity submaximal exercise are not fully understood,[155,156] exercise that elicits a $\dot{V}O_2$ slow component is poorly tolerated by volunteers.[157] Therefore, training programmes that attenuate the $\dot{V}O_2$ slow component or that extend the range of exercise intensities over which the slow component does not develop will improve endurance exercise performance.

Several studies have evaluated the effects of endurance training on $\dot{V}O_2$ kinetics during cycle exercise. In general, the steady state $\dot{V}O_2$ for the same moderate intensity exercise has not been found to change following a period of endurance training,[89,158] although the primary exponential increase in $\dot{V}O_2$ at the onset of exercise may be speeded.[158,159] In cross-sectional studies, the $\dot{V}O_2$ on-kinetic adjustment to the same absolute or relative exercise intensity has been reported to be faster in individuals with higher $\dot{V}O_{2max}$ values.[158,160] Faster $\dot{V}O_2$ kinetics at exercise onset, resulting in a more rapid attainment of the requisite steady state oxygen uptake, might be important in reducing the initial oxygen deficit and limiting the early increase in blood lactate. A speeded $\dot{V}O_2$ on-kinetic response may facilitate the rapid establishment of an intracellular environment that allows tighter metabolic control later in exercise.[161,162] Whether the primary mechanism for any speeding of the initial $\dot{V}O_2$ response to exercise is related to increased O_2 delivery to muscle or to a reduced inertia of the intracellular oxidative machinery consequent to an increased muscle mitochondrial density is debated.[159,163] Endurance training increases the CP,[164-166] and reduces the magnitude of the $\dot{V}O_2$ slow component (defined as the increase in $\dot{V}O_2$ between 3 and 6 minutes of exercise) for the same absolute power output.[164,167,168] Recent work in our laboratory has shown that 6 weeks of endurance running training results in a significant increase in the running speed at the MLSS, [20] and a significant reduction in the amplitude of the $\dot{V}O_2$ slow component (from 321 to 217 ml/min on average) for the same absolute treadmill running speed (unpublished observations; fig. 3). Although the reductions in blood lactate levels, ventilation, heart rate and plasma catecholamine levels that accompany endurance training (see section 4) might partly explain the reduced O_2 cost of heavy submaximal exercise after training, it appears that intramuscular changes and possibly alterations in motor unit recruitment patterns might be more important.[156,169] Of interest in this respect is the suggestion that the relative contribution of the $\dot{V}O_2$ slow component to the total $\dot{V}O_2$ response to heavy exercise is negatively related to aerobic fitness (as $\dot{V}O_{2max}$) and/or the proportion of type I fibres in the working muscles.[156]

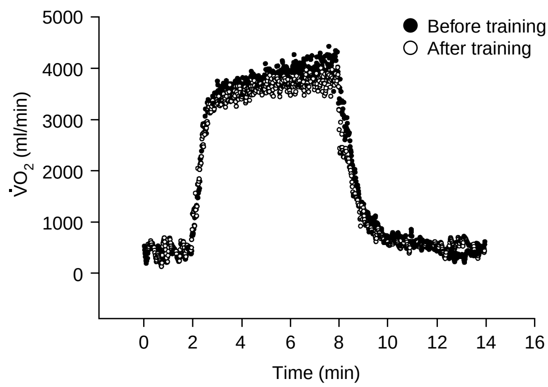

Fig. 3. The effect of 6 weeks of endurance training on the oxygen uptake response to a constant-load heavy exercise challenge in a typical individual. Note the marked reduction in the oxygen uptake ($\dot{V}O_2$) slow component (unpublished data).

6. Conclusion

Endurance exercise training results in numerous adaptations to the neuromuscular, metabolic, cardiovascular, respiratory and endocrine systems. These adaptations are reflected in improvements in the key parameters of aerobic fitness, namely the $\dot{V}O_{2max}$, exercise economy, the lactate/ventilatory threshold and the CP which will influence the oxygen uptake kinetics. An improvement in one or more of these parameters will result in an improvement in endurance exercise performance consequent to a rightward shift at various points on the velocity-time curve. The latter will allow an athlete to exercise for longer at the same exercise intensity or to sustain a higher speed for a given exercise duration. Although the aerobic parameters reviewed above are important determinants of endurance exercise performance, it should be borne in mind that competitive performance also depends upon psychological factors, race tactics and the prevailing environmental conditions. In addition, an athlete's ability to generate ATP anaerobically can be important in sprint finishes between athletes whose aerobic capabilities are similar.[170,171] Fukuba and Whipp[172] have recently suggested that an athlete's anaerobic work capacity (a derivative of the concept and computation of critical power) can determine his or her ability to initiate or respond to sections of a race that are faster than the athlete's best average velocity for the distance.

While the parameters of aerobic fitness are interrelated,[69] the specific emphasis placed on the training of each of these will depend upon an individual's personal physiological 'strengths' and 'weaknesses' (which may be assessed in the sports physiology laboratory), and the duration of the event being trained for. For example, a 3000m runner may place special importance on the development of the $\dot{V}O_{2max}$ and anaerobic capacity, while a marathon runner may focus on training to improve running economy and the running speed at lactate threshold. Presently, little is known about the most effective training practices for specifically improving the key parameters of aerobic fitness, or for altering different points on the velocity-time curve in order to effect a shift to the right of the velocity-time relationship. Exploration of the effect of various combinations of training volume, intensity and frequency on these determinants of endurance performance remains a fruitful area for future research.

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