ORIGINAL ARTICLE

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Time to exhaustion during severe intensity running: response following a single bout of interval training

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Abstract The primary aim of this study was to examine any change in performance caused by a fatiguing interval training session (TS). A secondary aim of this study was to examine the change in oxygen uptake (VO_2) during moderate and severe intensity running, and the relationship with the change in performance. Seven male runners [mean age 24 (SD 6) years, height 1.79 (SD 0.06) m, body mass 67.9 (SD 7.6) kg, maximal oxygen uptake (VO_{2max}) 4.14 (SD 0.49) 1 · min⁻¹] were studied. The VO_2 during moderate and severe intensity running and running performance were studied immediately prior to, 1 h following, and 72 h following TS. The TS was performed on a treadmill, and consisted of six bouts of 800 m at 1 km \cdot h⁻¹ below the velocity at \dot{VO}_{2max} $(v_{\dot{V}O_{2\text{max}}})$, with 3-min rest intervals. Performance was also assessed at 1 km · h⁻¹ below $v_{\dot{V}O_{2\text{max}}}$, in the form of time to exhaustion (t_{lim}) . The $\dot{V}O_2$ and heart rate (f_c) were assessed both during the severe intensity performance trial, and the moderate intensity run at 50% $v_{\dot{V}O_{2max}}$. Whilst a significant change was observed in running performance and the $\dot{V}O_2$ during both moderate and severe intensity running prior to and following TS, no relationship was observed between the magnitude of change in these variables. At 1 h following TS, t_{lim} had decreased by 24%, $\dot{V}O_2$ during moderate intensity running had increased by 2%, and the difference in $\dot{V}O_2$ between 2 min 45 s and the end of severe intensity running had increased by 91% compared with values recorded prior to TS. At 1 h following TS, f_c had also increased significantly during moderate intensity run-

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ning by 5% compared to the value recorded prior to TS. These findings demonstrated that TS resulted in a reduction in performance, and that the relationship between running performance and $\dot{V}O_2$ during running may be altered under conditions of prolonged fatigue.

Key words Oxygen uptake · Endurance · Running · Fatigue

Introduction

Time to exhaustion (t_{lim}) during severe intensity exercise has been used to assess endurance running performance previously (eg. Billat et al. 1994a, b). According to the critical power concept, it has been shown that t_{lim} during severe intensity exercise, which is defined as being above critical power, is generally less than 30 min (Hill 1993). During exercise to exhaustion on a cycle ergometer at constant load, it has been found that only exercise at power outputs above critical power results in the attainment of maximal oxygen uptake (VO_{2max}; Hill and Smith 1999). Conversely, Billat et al. (1998) have found that during running exercise at a constant velocity above critical velocity, oxygen uptake (VO_2) did not reach VO_{2max} in high-level distance runners. It has, however, been suggested that t_{lim} during severe intensity exercise may be related to the magnitude of the change in VO_2 between 3 min and the end of exercise (Poole et al. 1994a). Two recent studies have demonstrated that VO_2 during heavy intensity running (James and Doust 1999) and moderate intensity running (James and Doust 1998) is increased following an interval training session (TS). It would be interesting therefore to examine whether following a similar TS, changes in \dot{VO}_2 during moderate and severe intensity running are related to changes in $t_{\rm lim}$.

Many mechanisms have been proposed to explain the additional $\dot{V}O_2$ during running in a fatigued condition, including reduced neural input to the active muscles resulting in reduced force production, reduced tolerance

to stretch loads, reduced recoil characteristics, and depleted muscle glycogen stores (Sherman et al. 1983; 1984; Buckalew et al. 1985; Nicol et al. 1991a, b). A greater recruitment of type II muscle fibres, possibly in addition to, or instead of the type I fibres have been suggested to offer a further potential mechanism (Sejersted and Vollestad 1992). In the case of running exercise, it has been suggested that a changed fibre recruitment pattern may be a product of the damage caused by the repeated stretch-shortening cycles, glycogen depletion in selected fibres, or other "non-metabolic" fatigue (Hagerman et al. 1984; Vollestad et al. 1984; Green 1991).

Differences in the $\dot{V}O_2$ during running have been shown to have significant implications for performance, especially in subjects with similar values for $\dot{V}O_{2max}$ (Costill and Winrow 1970; Daniels 1974; Morgan et al. 1989a). However, in general, studies with both a crosssectional and longitudinal design have demonstrated equivocal findings with regard to the relationship between $\dot{V}O_2$ during constant velocity running and performance (Morgan and Craib 1992).

The $t_{\rm lim}$ at the velocity associated with $\dot{V}O_{2\rm max}$ $(v_{\dot{V}O_{2max}})$ appears to be a suitable measure of running performance, and is probably the best method currently available for the assessment of changes in endurance running performance following an intervention. The $t_{\rm lim}$ has been demonstrated to be a reliable measure during running at $v_{\dot{V}O_{2max}}$ (Billat et al. 1994b). The t_{lim} when measured during running at $v_{\dot{V}O_{2max}}$ has been shown to be closely related to velocity maintained during a half marathon competition, and the threshold blood lactate concentration when expressed as $\% \dot{V}O_{2max}$ (Billat et al. 1994a). Based on the usual duration of t_{lim} at $v_{VO_{2}\text{max}}$ (about 6 min 30 s), and the proposed physiological factors determining the t_{lim} at this intensity, it is somewhat surprising that t_{lim} is related to half marathon velocity and the threshold blood lactate concentrations, but not to $\dot{V}O_{2max}$, $v_{\dot{V}O_{2max}}$, running economy or velocity maintained during performance over 3000-m. However, results from previous studies have indicated that the measurement of t_{lim} at $v_{\dot{V}O_{2\text{max}}}$ in a laboratory setting is suitable for studying the effects of an intervention on performance in a group of endurance runners (Billat et al. 1994b).

This study aimed to establish whether t_{lim} was reduced following a bout of severe intensity interval running training, and whether a change in $\dot{V}O_2$ during moderate and severe intensity exercise was related to a change in t_{lim} .

Methods

Subjects

Procedure

All the subjects rested for 3 days prior to the start of the experiment, and no training was performed throughout the test period. The subjects were instructed to consume their normal high carbohydrate diets between test sessions. For 3 h prior to each test session the subjects refrained from eating, and consuming caffeine and alcohol. The subjects were instructed to arrive at each test session fully hydrated, and wearing identical footwear and similar clothing. The same individual warm-up routine was performed on each occasion. All testing took place between 1 p.m. and 5 p.m.

All running took place on a Woodway treadmill (Cardiosport Ltd., Salford, UK). The speed of the treadmill driving motor was monitored by sensors on the motor and was shown on the digital display. Since the treadmill belt was unable to slip due to the rack and pinion drive, it always rotated at the same speed as the motor, and therefore the treadmill belt was effectively self-calibrated at the speed displayed on the control console. Routine checks were made, in addition to the self-calibration, by visually counting the belt rotations during a recorded time. The belt length was accurately measured, and when multiplied by the number of revolutions and divided by the time for the known number of revolutions, the belt speed was found. The gradient of the treadmill was checked using a protractor and spirit level. The gradient was determined as the tangent of the angle of incline (i.e. opposite/adjacent).

On the first occasion the subjects visited the laboratory, age, height, mass, $\dot{V}O_{2max}$ and $v_{\dot{V}O_{2max}}$ were determined. The $\dot{V}O_{2max}$ was measured for treadmill exercise using an incremental velocity protocol (Jones and Doust 1996a). The duration of each increment was 1 min, with an increase of 1 km \cdot h⁻¹ each minute. All running was performed with the treadmill at a 1% gradient since this has been the gradient found to represent outdoor running best (Jones and Doust 1996b). The $v_{iO_{2max}}$ was defined as the velocity corresponding to the highest $\dot{V}O_{2max}$ value recorded during the incremental test. In the event of a plateau, the lowest associated velocity was recorded.

During exercise each subject wore a nose clip and a large, broad flanged rubber mouthpiece (Collins, Mass., USA) fitted to a lowresistance (inspired <3 cm H₂O and expired <1 cm H₂O at flow rates up to 350 l min⁻¹ ambient temperature and pressure, saturated) breathing valve (University of Brighton, England) of negligible volume (90 ml), consisting of lightweight perspex tubing (T-shape) into which was mounted two rubber flap one-way low resistance valves (Mine Safety Appliances Ltd.), connected to a 200-1 Douglas bag from the expired side via a 1-m length of lightweight Falconia tubing of 36-mm bore (Baxter Woodhouse and Taylor Ltd.). Expired gas was collected for a timed period of a whole number of breaths during the final 45 s of each minute. The expired gas was analysed for O₂ and CO₂ content, using a paramagnetic O₂ analyser (1100 series, Servomex, Crowborough, UK) and an infrared CO₂ analyser (1490 series, Servomex, Crowborough, UK). Each analyser was calibrated at two points, and checked for linearity using high precision gas mixtures and room air. Gas volume was measured using a dry gas meter (Harvard Apparatus Ltd., Edenbridge, UK) previously calibrated against a Tissot spirometer, and regularly checked for linearity throughout the complete collection volume range using a 7-1 calibration syringe (Hans Rudolf Inc., Kansas City, Mo., USA).

A standardised relative exercise load for each subject of 6×800^{-1} m intervals at 1 km \cdot h⁻¹ below $v_{\dot{V}O_{2max}}$, with a 3 min rest between each interval, was used as the interval training session (TS); this was designed to replicate a severe overload of the kind which well-trained runners would regularly perform. The heart rate (f_c) was recorded continuously using a telemetric system (Sport Tester, Polar Electro Oy, Kempele, Finland). The TS was performed on two occasions at a similar time of day (2–4 p.m.) by all the subjects.

Prior to and following both TS, a series of moderate constant intensity runs were performed (tests 1m, 2m, 3m, 4m, 5m). Also, a series of severe constant intensity runs were performed (tests 1s, 2s, 3s). The design of the study, which included the TS to be repeated, was to allow tests to be performed either 1 h or 72 h after TS. Due to the fatiguing nature of the severe intensity runs, it was not considered appropriate to perform tests at 1 h and 72 h following the same TS.

Seven well-trained male runners gave informed consent to take part in the study which had been approved by the Ethics Committee of Chelsea School, University of Brighton. The Subjects were all thoroughly habituated to laboratory test procedures.

Table 1 Anthropometric data
$(n = 7)$. $\dot{V}O_{2 \max}$ Maximal
oxygen consumption, $v_{VO_{2max}}$
running velocity at
maximal oxygen consumption

Age (years)		Height (m)		Mass (kg)		$\dot{V}O_{2 \max} (l \cdot \min^{-1})$		$v_{\dot{V}O_{2max}} \ (\mathrm{km} \cdot \mathrm{h}^{-1})$	
Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
24	6	1.79	0.10	67.9	7.6	4.14	0.49	19.4	1.3

The series of moderate intensity runs were at the velocity corresponding to 50% $v_{\dot{v}O_{2max}}$, which was chosen to ensure that all the subjects were exercising at an intensity well below that which would elicit a rise in blood lactate concentration above 2 mmol·1⁻¹ (James and Doust 1998). This requirement has been found necessary to provide steady-state gas exchange conditions (Barstow and Mole 1991). Each subject was weighed and then instructed to begin running on the treadmill ergometer for 15 min. From 10 min to 12 min and 13 min to 15 min f_c and cadence were recorded, along with duplicate collections of expired gas in whole numbers of breaths over a timed period which was always in excess of 1-min duration.

The series of severe intensity runs were designed to allow determination of t_{lim} , and were a continuation of certain moderate intensity tests, including test 1m, test 3m, and test 5m, respectively. Within 15 s the treadmill velocity was increased to 1 km \cdot h⁻¹ below $v_{\dot{V}O_{2max}}$, at which point the timing was initiated. Expired gas was collected for 30-s periods, and f_c and cadence were determined from 2 min 30 s until the exercise was terminated. Verbal encouragement was given, but the subjects were not told the time elapsed either during or following the test until the whole experiment had been completed, in order to minimise any effects of altered motivation due to a time goal. Timing was stopped as each subject's hands touched the frame of the treadmill. Following the test expired gas was analysed to determine the difference in VO_2 between the value after 2 min 30 s of severe intensity exercise and the value at the end of the test. The $\dot{V}O_2$ was recorded as representing the mid-point of the collection period (e.g. 2 min 45 s), and all calculations were based upon these times. Since the difference in VO_2 was observed over differing durations among subjects, the difference was expressed as a rate of increase in litres per minute per minute.

A velocity of 1 km \cdot h⁻¹ below $v_{\dot{V}O_{2max}}$ rather than $v_{\dot{V}O_{2max}}$, which has been widely used to determine t_{\lim} previously (Billat and Koralsztein 1996), was chosen in the light of the expected t_{\lim} . To determine a difference in $\dot{V}O_2$ between 2 min 30 s and the end of exercise, t_{\lim} had to be long enough to take at least two reliable samples of expired gas (i.e. >210 s) in all three severe intensity tests. The particular concern was the test in the fatigued condition at 1 h following the training session (i.e. test 2s). Accounting for our method of determination of $v_{\dot{V}O_{2max}}$, and our experience of t_{\lim} at this velocity under a variety of conditions, 1 km \cdot h⁻¹ below $v_{\dot{V}O_{2max}}$ was considered an appropriate velocity to ensure a duration of more than 210 s in all the subjects.

Test 1 m and test 1 s were performed following 3 days of rest. Test 2m and 4m were performed immediately prior to TS. Tests 3m and test 2s were performed 1 h following the first TS. Test 5m and test 3 s were performed 72 h following the second TS. Following the first TS, the subjects were weighed to determine any changes in body mass. The subjects were instructed to consume a volume of water calculated from the change in body mass during the overload to reflect the loss of fluid as sweat. This rehydration strategy has been previously shown to be successful through determination of changes in plasma volume (James and Doust 1998).

Statistical analysis

Repeated-measures analysis of variance with multiple comparison (Tukey) was used to determine the significance of differences between tests. A *P*-value of less than 0.05 was chosen prior to the study as the level at which a difference would be regarded as significant.

Results

The anthropometric details of the subject are given in Table 1. All but two subjects completed TS, with one subject failing to complete the final interval (400 m completed), and the other subject failing to complete the final three intervals in both TS (630, 660, 520 and 730, 630, 760 m completed, respectively). The reason for the failure to complete the TS was fatigue in both cases, so was not considered a limitation for the overall aim of the experiment.

The f_c response during the first and second TS is shown in Figs. 1 and 2 respectively. It is clear that the response was no different between the two TS, and the maximal f_c during each interval was approaching the maximal f_c recorded during the incremental test as shown by the line breaking the y axis in Figs. 1 and 2. The exercise intensity for both the training session and the run to exhaustion was severe, at a velocity which elicited about 95% $\dot{V}O_{2max}$ after 3 min of running.

Responses to the severe intensity runs are shown in Table 2. A significant difference was observed for t_{lim} between tests 1s and 2s, and tests 2s and 3s (P < 0.05). The t_{lim} recorded 1 h following the TS (test 2s) was 24% less than t_{lim} in the rested condition (test 1s) and 32% less than t_{lim} 72 h following TS (test 3s). The decrease in t_{lim} in test 2s was associated with a significantly greater VO_2 difference between 2 min 45 s and the end of exercise in test 2 compared with test 1 or 3 (P < 0.05). No

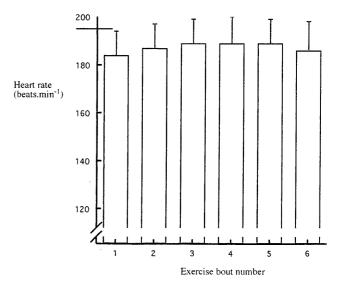


Fig. 1 Heart rate at the end of each exercise bout during the first training session (*line breaking axis* denotes mean maximum during the incremental test). Values are mean and standard deviation

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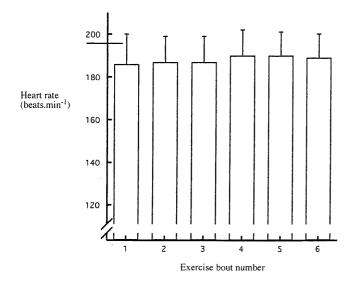


Fig. 2 Heart rate at the end of each exercise bout during the second training session (*line breaking axis* denotes mean maximum during the incremental test). Values are mean and standard deviation

relationship was observed between the change in t_{lim} and the change in $\dot{V}O_2$ between 2 min 45 s and the end of exercise during the severe intensity run (see Fig. 3).

Responses to the moderate intensity runs are shown in Table 3. A significant difference was observed for $\dot{V}O_2$ in test 3m compared with test 2m, and test 5m compared with test 4m (P < 0.05). At 1 h following the TS (test 3m), $\dot{V}O_2$ had increased by 2% above the value

Table 2 Responses to the severe intensity run to exhaustion following rest (*test 1s*), 1 h following (*test 2s*) and 72 h following (*test 3s*) the training session (*TS*). t_{lim} Time limit to exhaustion, 2 min 45 s $\dot{V}O_2$ oxygen consumption at 2 min 45 s, End $\dot{V}O_2$ oxygen consumption at exhaustion, $\dot{V}O_2$ diff difference between oxygen consumption at 2 min 45 s and exhaustion, $f_{\text{c,max}}$ heart rate at exhaustion

	Test 1s		Test 2s		Test 3s		
	Mean	SD	Mean	SD	Mean	SD	
$t_{\rm lim}$ (s)	318	59	243 ^a	52	359 ^b	130	
$2 \min_{(1 \cdot \min^{-1})} 45 \text{ s } \dot{V}O_2$	3.99	0.62	3.94	0.65	3.96	0.62	
End $\dot{V}O_2$ (1 · min ⁻¹)	4.21	0.61	4.19	0.62	4.17	0.59	
\dot{VO}_2 diff (1 · min ⁻²)	0.11	0.06	0.21 ^a	0.08	0.11 ^b	0.11	
$f_{c,max}$ (beats $\cdot min^{-1}$)	193	12	191	11	192	13	
Cadence (stride $\cdot \min^{-1}$)	91	6	91	5	91	6	

^a Difference between *test 1s* and *test 2s* (P < 0.05)

^b Difference between *test 2s* and *test 3s* (P < 0.05)

prior to TS (test 2m). At 72 h following the TS (test 5m), $\dot{V}O_2$ had decreased by 3% below the value prior to TS (test 4m). A significant difference was observed for f_c in test 3m compared with test 2m, and test 5m compared

Fig. 3 Relationship between change in time to exhaustion and change in oxygen uptake $(\dot{V}O_2)$ kinetics during severe exercise performed prior to and 1 h following the training session

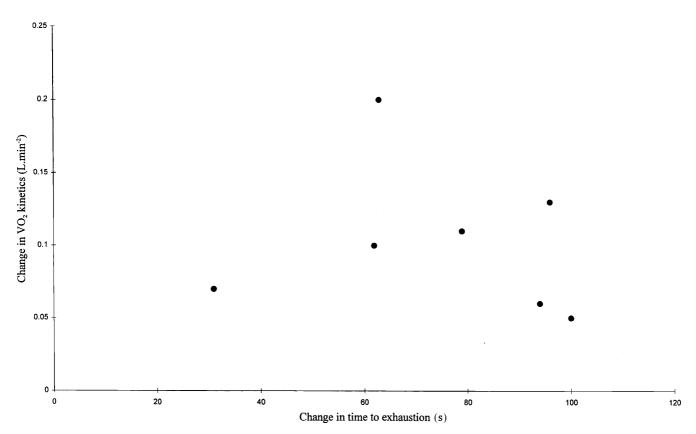


Table 3 Responses to the moderate intensity run following rest (*test 1m*), prior to (*test 2m* and *test 4m*), 1 h following (*test 3m*) and 72 h following (*test 5m*) both training sessions (*TS*). \dot{VO}_2 oxygen consumption, f_c heart rate

	Test 1m		Test 2m		Test 3m		Test 4m		Test 5m	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
$\dot{V}O_2 (l \cdot min^{-1})$ $f_c (beats \cdot min^{-1})$	2.21	0.23	2.19	0.26	2.23	^a 0.26	2.23	0.27	2,16 ^b	0.23
$f_{\rm c}$ (beats $\cdot \min^{-1}$)	141	17	136	13	143 ^a	14	137	15	119 ^b	48
Cadence (strides \cdot min ⁻¹)	81	5	80	4	81	4	80	4	80	4

^a Difference between *test 2m* and *test 3m* (P < 0.05)

^b Difference between *test 4m* and *test 5m* (P < 0.05)

with test 4m (P < 0.05). At 1 h following TS (test 3m), f_c had increased by 5% above the value prior to TS (test 2m). At 72 h following TS (test 5 m), f_c had decreased by 13% below the value prior to TS (test 4 m). No relationship was observed between the change in t_{lim} and the change in $\dot{V}O_2$ during the moderate intensity run (see Fig. 4).

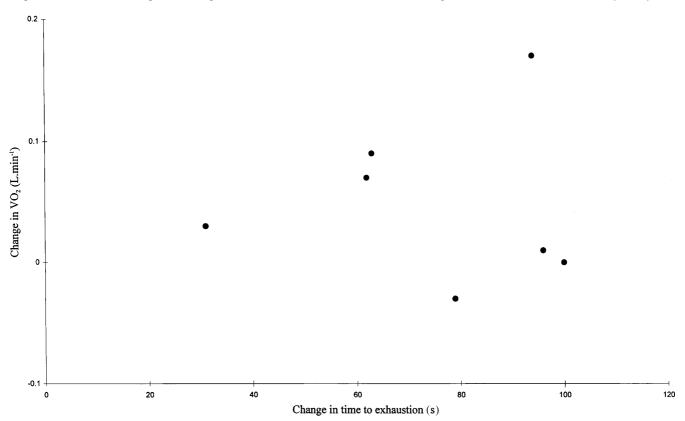
Although a significantly greater $\dot{V}O_2$ difference between 2 min 45 s and the end of running at severe intensity, and a significantly greater $\dot{V}O_2$ during running at moderate intensity was observed in test 2 compared with test 1, no relationship was found between these two measures (see Fig. 5). No significant differences were observed for cadence between any of the conditions in either the run at moderate intensity, or at severe intensity (P < 0.05).

Fig. 4 Relationship between change in time to exhaustion and change in oxygen uptake $(\dot{V}O_2)$ during moderate exercise performed prior to and 1 h following the training session

Discussion

Although a decrease in t_{lim} and an increase in the VO_2 response were observed during the trial at severe intensity following TS in the present study, no relationship existed between the change in these two variables. Likewise, although $\dot{V}O_2$ during exercise at moderate intensity was increased following TS, no relationship existed between this change and the decrease in t_{lim} .

Whilst the primary aim of the study was to examine the change in t_{lim} , in order to establish possible mechanisms, it was interesting to examine whether t_{lim} was changed in association with the $\dot{V}O_2$ response during running at a severe intensity. Although gas exchange has been shown not to reach a steady state during exercise at severe intensity (e.g. Barstow and Mole 1991), examination of the $\dot{V}O_2$ response at the same intensity as that used for the assessment of t_{lim} and TS may provide useful information. Poole et al. 1994a have suggested that t_{lim} during exercise at severe intensity may be



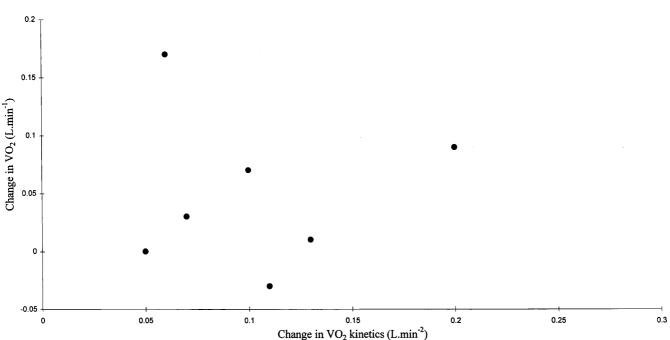


Fig. 5 Relationship between change in oxygen uptake $(\dot{V}O_2)$ kinetics during severe exercise and change in $\dot{V}O_2$ during moderate exercise performed prior to and following the training session

related to the magnitude of the change in $\dot{V}O_2$ between 3 min and the end of exercise. This suggestion is based on the notion that the quicker that $\dot{V}O_2$ reaches $\dot{V}O_{2max}$, the quicker fatigue will develop, since exercise at $\dot{V}O_{2max}$ can only be maintained for a finite time.

In the present study, the change in VO_2 between 2 min 45 s and the end of exercise was expressed as an acceleration and then examined in this regard, but no relationship was found between the change in t_{lim} and the change in the $\dot{V}O_2$ response following TS. Due to the off-line technique used to determine gas exchange in the present study, if subjects reached VO_{2max} prior to t_{lim} , it was not possible to account for this when calculating the acceleration in $\dot{V}O_2$. It is also possible that an increase in $\dot{V}O_2$ may be constrained as $\dot{V}O_2$ approaches $\dot{V}O_{2max}$, thereby slowing the attainment of \dot{VO}_{2max} . Since the slow component of $\dot{V}O_2$ kinetics has yet to be characterised, the extent of the limitation imposed by our gas analysis technique is unknown. Notwithstanding this limitation, the finding suggests that other factors, such as anaerobic capacity, may be important in determining t_{lim} at a severe intensity. It has been previously demonstrated that anaerobic capabilities may contribute significantly to distance running performance (Bulbulain et al. 1986).

Bernard et al. (1998) have recently demonstrated the importance of determining $\dot{V}O_2$ during running at velocities representative of performance velocities. This importance is greatest for athletes competing in events performed above the blood lactate threshold. Exercise above this threshold (heavy intensity) has been shown to elicit an excess $\dot{V}O_2$ above that predicted from the velocity- $\dot{V}O_2$ relationship derived from exercise below the threshold, which has been shown to have both an intensity and a time component (Whipp and Wassermann 1972). Bernard et al. (1998) have referred to $\dot{V}O_2$ during exercise above the threshold of blood lactate concentration as the "aerobic energy cost", and suggested that an increase in the aerobic energy cost occurs at heavy intensities. However, due to the timedependent nature of the development of the excess $\dot{V}O_2$, Bernard et al. (1998) have demonstrated that it is not satisfactory to determine the aerobic energy cost after 3 min of exercise.

The difference between the true aerobic energy cost when a steady state is reached, and an inadequate determination of aerobic energy cost (i.e. prior to attainment of steady state or velocity too low), may be termed the aerobic energy deficit, or more simply the "oxygen cost deficit". The oxygen cost has been defined as the $\dot{V}O_2$ per body mass (kilograms) per unit distance (kilometres) expressed in millilitres, and is calculated as the quotient of exercising minus resting VO_2 (millilitres per kilogram per minute), and velocity (kilometres per minute); (Lacour et al. 1990, adapted from di Prampero 1986). In the case of the present study, we analysed our data to determine the oxygen cost deficit, which we defined as the difference between VO2 (millilitres per kilogram per kilometre), after 10 min of running at moderate intensity (50% $v_{\dot{V}O_{2max}}$) and the $\dot{V}O_2$ (millilitres per kilogram per kilometer) at exhaustion during running at severe intensity (about 95% $\dot{V}O_{2max}$), in the fatigued and non-fatigued conditions. However, we found no relationship between changes in oxygen cost deficit and performance changes between pre and post TS (see Fig. 6). This finding would suggest that the fatigue 1 h following TS, as suggested by the reduced $t_{\rm lim}$, is not simply related to altered oxygen cost deficit.

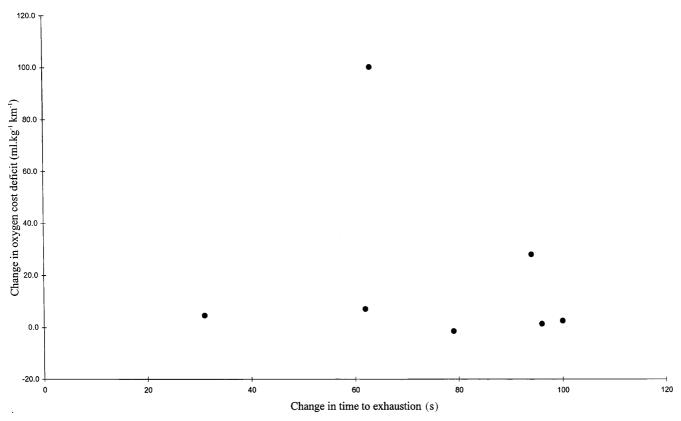


Fig. 6 Relationship between change in time to exhaustion and change in oxygen cost deficit during severe exercise performed prior to and 1 h following the training session. Oxygen cost deficit was calculated as the difference between the oxygen costs of the exercise at moderate and severe intensities

Although t_{lim} is itself a performance measure, t_{lim} at $v_{\dot{V}O_{2max}}$ has also been shown to correlate with other characteristics of endurance running such as the velocity maintained during a half marathon competition (Billat et al. 1994a). The results of studies examining the relationship between t_{lim} at $v_{VO_{2\text{max}}}$ and other physiological measures which are often related to endurance running performance have been equivocal (see Table 4). It is of interest that in one study anaerobic capacity has also been shown to account in part for differences in $t_{\rm lim}$ at $v_{VO_{2max}}$ (Hill and Rowell 1996). These different findings may reflect variations between the fitness level of the subjects, and methodological variations. The protocol for determination of $\dot{V}O_{2max}$, and particularly $v_{\dot{V}O_{2max}}$, may be particularly important. For example, the duration of each increment of a graded exercise test may influence the VO_2 -velocity relationship, since it is now widely acknowledged that with long increments an excess $\dot{V}O_2$ will develop for each increment above the anaerobic threshold (Hansen et al. 1988). The effect of the developing excess VO_2 during slow increment protocols will result in lower velocities for a given $\dot{V}O_2$, and hence a relatively reduced $v_{VO_{2max}}$. Although we exercised subjects at 1 km \cdot h⁻¹ below $v_{VO_{2max}}$ in the present study, the graded test to maximum consisted of rapid incre-ments (i.e. $1 \text{ km} \cdot \text{h}^{-1} \cdot \text{min}^{-1}$).

Whilst Billat et al. (1994b) have found that the determination of t_{lim} was repeatable in a group of sub-elite runners as a whole, the intra-subject variation was in the region of 10%. Intra-subject variation of this magnitude may have confounded our findings that no relationship existed between either the change in t_{lim} and $\dot{V}O_2$ during running at moderate intensity, or the change in t_{lim} and $\dot{V}O_2$ response to running at severe intensity.

The reduced performance at 1 h following TS compared with performance after rest or 72 h following TS may have been due to a variety of factors. It is unlikely, however, that factors such as dehydration or increased core body temperature were responsible taking into

Table 4 Comparison of time to exhaustion (t_{lim}) and relationship with other physiological characteristics in endurance runners $\dot{V}O_{2max}$ Maximal oxygen uptake, $v_{\dot{V}O_{2max}}$ velocity at $\dot{V}O_{2max}$, T_{an} anaerobic threshold

Study	$t_{\rm lim}$	Correlation coefficient				
		$\dot{V}O_{2\text{max}}$	$v_{\dot{V}\mathrm{O}_{2\mathrm{max}}}$	T _{an}		
This study ^a Billat et al. $(1994b)^b$ Billat et al. $(1994d)^c$ Billat et al. $(1995)^c$ Billat et al. $(1994e)^b$ Billat et al. $(1994c)^c$ Billat et al. $(1994a)^b$ Hill et al. $(1996)^d$	318 404 360 321 404 325 371 290	-0.793* 0.138 -0.347* -0.200 0.170 -0.502*	-0.621 0.241 -0.362* -0.538* 0.320 -0.691*	-0.343 0.671* 0.378* -0.050 0.580* 0.629* 0.660*		

* Denotes significant correlation (P < 0.05)

^a Well-trained, ^b subelite, ^c elite, ^d well-trained woman Note: all studies used $v_{i/O_{2max}}$ except the present study account our findings following an identical TS in previous studies (James and Doust 1998, 1999). In those studies, by 1 h following TS, plasma volume and rectal temperature had both returned to the values recorded prior to TS. Furthermore, it was demonstrated in the present study that cadence was unchanged 1 h following TS. Although this finding has been demonstrated previously, the initial measurement was not made until 1 day following the training session (Morgan et al. 1990, 1996). In contrast, Xu and Montgomery (1995) have found that immediately following prolonged exercise at moderate intensity, cadence is slightly, but significantly altered. However, it should be noted that that change was observed immediately following the training session.

It may be speculated that changes in other kinematic and kinetic variables may have occurred following the training session, which were not evident when examining cadence. For example, recently Candau et al. (1998) have demonstrated increased variability in step frequency with fatigue during running. The relationship demonstrated by Candau et al. (1998) between step variability and VO_2 during running is of particular interest.

Several physiological changes have been associated with an increased $\dot{V}O_2$ during moderate intensity running, and the changed $\dot{V}O_2$ response to severe intensity running, which may act to reduce performance. Of importance are studies by Poole et al. (1991, 1992) who have suggested that pulmonary $\dot{V}O_2$ closely reflects $\dot{V}O_2$ measured over the working limb during exercise at both moderate and severe intensities. Therefore, factors that may contribute to a changed $\dot{V}O_2$ in the exercising limbs should be considered as possible candidates for altered $t_{\rm lim}$. With regard to running at moderate intensity, due to the steady-state response, it is possible to derive an energy equivalent. Following exactly the same overload as that used in this study, we have observed that 31% of the change in $\dot{V}O_2$ was due to increased metabolism of fat (James and Doust 1998). In the present study, 10% of the change in VO_2 was due to increased metabolism of fat, as calculated from pulmonary gas exchange variables. It is possible that t_{lim} was influenced by substrate availability, and an increased metabolism of fat in the moderate intensity run 1 h following TS indicated a depletion of muscle glycogen stores. A previous study has demonstrated the importance of muscle glycogen stores prior to exercise for short duration performance (Maughan and Poole 1981).

With regard to exercise of severe intensity, whilst a close relationship between blood lactate concentration and the $\dot{V}O_2$ response to running at severe intensity has been observed, it has been suggested that blood lactate concentration is simply correlated with, but does not cause the $\dot{V}O_2$ response. A dissociation between the two has been demonstrated via training and infusion of adrenaline during exercise in humans which increases blood lactate concentration and decreases pH (Gaesser 1994) and infusion of lactate into exercising dog muscle (Poole et al. 1994b). Factors which are associated with

lactate production in the exercising limb have also been investigated. Although evidence is not direct, changes in muscle fibre recruitment has been suggested as a likely explanation for a significant part of the $\dot{V}O_2$ response to severe exercise (Poole et al. 1994a). The suggestion that a progressively greater recruitment of type II fibres may contribute to the $\dot{V}O_2$ response to exercise at a severe intensity may also partly explain the reduced performance following an interval training session.

In conclusion, the significant reduction in performance during running at a severe intensity following the training session was associated with an increase in $\dot{V}O_2$ during running at moderate intensity, and a changed $\dot{V}O_2$ response to running at severe intensity. However, no relationship existed between the magnitude of the change in performance and the $\dot{V}O_2$ response during running at either moderate or severe intensity.

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