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Oxygen uptake during high-intensity running: response following a single bout of interval training

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Abstract

Elevated oxygen uptake ($\dot{V}O_2$) has been previously observed during moderate intensity running following a bout of interval running training. To further investigate this phenomenon, the $\dot{V}O_2$ response to high-intensity exercise was examined following a bout of interval running. Well-trained endurance runners were split into an experimental group (maximum oxygen uptake, $\dot{V}O_2$ max 4.73 $\pm$ 0.39 L.min^{-1}) and a reliability group ($\dot{V}O_2$ max 4.77 $\pm$ 0.26 L.min^{-1}). The experimental group completed a training session (4 x 800 m @ 1 km.h^{-1} below speed at $\dot{V}O_2$ max, with 3 min rest between each 800 m interval). Five min prior to, and 1 h following the training session, subjects completed 6 min 30 s of constant speed, high-intensity running designed to elicit 40% Δ (test 1 and 2). The slow component of $\dot{V}O_2$ kinetics was quantified as the difference between $\dot{V}O_2$ from 6 min and $\dot{V}O_2$ from 3 min of exercise, i.e., $\Delta\dot{V}O_2$ (6-3). The $\Delta\dot{V}O_2$ (6-3) was the same in two identical conditions in the reliability group (mean $\pm$ s.d.: 0.30 $\pm$ 0.10 L.min^{-1} vs 0.32 $\pm$ 0.13 L.min^{-1}). In the experimental group, the magnitude of the slow component of $\dot{V}O_2$ kinetics was increased in test 2 compared with test 1 by 24.9% (0.27 $\pm$ 0.14 L.min^{-1} vs 0.34 $\pm$ 0.08 L.min^{-1}) ($p < 0.05$). The increase in $\Delta\dot{V}O_2$ (6-3) in the experimental group was observed in the absence of any significant change in body mass, core temperature or blood lactate concentration, either at the start or end of test 1 or 2. It is concluded that similar mechanisms may be responsible for the slow component of $\dot{V}O_2$ kinetics and the fatigue following the training session. It has been previously suggested that this mechanism may be primarily linked to changes within the active limb, with the recruitment of alternative and/or additional less efficient fibres.

Keywords: Oxygen uptake - running - training - fatigue

Introduction

Fatigue following long-duration running exercise has been observed previously (Sherman et al., 1984; Nicol et al., 1991a, b). The consequences of fatigue, which may last for several days following a bout of long-duration running exercise, have also been shown to influence the pulmonary $\dot{V}O_2$ during moderate-intensity running (Xu and Montgomery, 1995; James and Doust, 1998). James and Doust (1998) demonstrated an increased $\dot{V}O_2$ during moderate-intensity running at 50% $\dot{V}O_2$ max, one hour following an interval training session. Xu and Montgomery (1995) demonstrated increased $\dot{V}O_2$ during moderate-intensity running at 54% and 67% $\dot{V}O_2$ max, immediately following a 90 minute run performed at both 65% and 80% of $\dot{V}O_2$ max.

It is possible that a significant portion of the increased $\dot{V}O_2$ observed during moderate-intensity, long-duration running originates from a changed efficiency of the active muscles, since Morgan et al (1996) have suggested that 30 min of high-intensity running does not influence gait mechanics. Many mechanisms have been proposed 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; Sherman et al., 1984; Buckalew et al., 1985; Nicol et al., 1991a; Nicol et al., 1991b). A greater recruitment of type II muscle fibres, possibly in addition to, or instead of the type I fibres offers a further potential mechanism (Sejersted and Vollestad, 1992). In the case of running exercise, 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). Technical limitations have prevented systematic investigation of this issue in exercising humans, however.

It has also been demonstrated that a proportion of the slow component of $\dot{V}O_2$ kinetics during prolonged high-intensity, constant-load cycling exercise primarily originates in the exercising limb (Poole et al., 1991). Barstow et al. (1996) have demonstrated an inverse relationship between the amplitude of the slow component of $\dot{V}O_2$ kinetics expressed relative to the overall increase in $\dot{V}O_2$, and the % type I fibres in cycling exercise. It has also been demonstrated that a relationship exists between % type I fibres and muscular efficiency during both cycling and novel knee extension exercise (Coyle et al., 1992). Fatigue during isometric exercise in humans has been associated with increased $\dot{V}O_2$ in the active limb, and this increased $\dot{V}O_2$ closely mapped altered fibre recruitment patterns determined through intramuscular EMG recordings and the glycogen depletion method (Sejersted and Vollestad, 1992). Attempts to manipulate the contribution of different fibre types to power production during cycling exercise through alterations in cadence have produced equivocal findings. It has been observed that an increase in cycling cadence results in an increased magnitude of the slow component of $\dot{V}O_2$ kinetics in one study (Gaesser et al., 1992), and a tendency for a decreased magnitude of the slow component of $\dot{V}O_2$ kinetics in another (Barstow et al., 1996). During graded incremental cycling exercise, alterations in cadence had no systematic effect on the development of a slow component of $\dot{V}O_2$ kinetics for work rates above the lactate threshold (Zoladz et al., 1995).

Since the responses following prior running exercise are not well known, the present study examined the effect of a bout of fatiguing running exercise on the $\dot{V}O_2$ during constant-speed, high-intensity running.

Methods

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 familiar with laboratory testing

procedures and treadmill running. Subjects were divided into an experimental and a reliability group, for which details are given in table 1.

All subjects rested for 3 days prior to testing. The subjects were instructed not to eat, or consume caffeine or alcohol in the 3 h prior to the test, and to consume their normal diet between testing sessions. For each subject, all testing took place at the same time of day, and subjects followed their individual warm-up routines. Subjects were instructed to present to each testing session in a rested and fully hydrated state, wearing identical footwear and clothing.

In the experimental group, test 1 was performed immediately preceding the training session and test 2 was performed 1 hour following the training session. Following the training session, the subjects were weighed to determine any changes in body mass. Then subjects were instructed to consume a volume of water determined by the loss of body mass during the training session, which was thought to primarily constitute sweat losses during the training session. This rehydration strategy has been previously shown to be successful through determination of plasma volume changes (James and Doust, 1998).

The reliability group performed test 1 and 2 within a week under rested conditions, with no training performed in the three days prior to both tests. Within each subject, the tests were performed at a similar time of day.

On the first occasion subjects visited the laboratory, age, height, and body mass were recorded. A maximal incremental test with the treadmill set at a 1% gradient was performed to determine $\dot{V}O_2$ max and ventilatory threshold (T_{vent}). Increment duration was one minute, with an increase in speed of 1.0 km.h^{-1} each minute until volitional exhaustion. The primary criterion used to determine whether $\dot{V}O_2$ max was attained, was a plateau in $\dot{V}O_2$ with an increase in speed. Since not all

subjects demonstrated a clear plateau, $\dot{V}O_2$ max was also used to refer to the highest $\dot{V}O_2$ recorded during the test, as long as other criteria were fulfilled (i.e., R > 1.05, maximum fc > 90% of age-adjusted estimated maximum). The speed at $\dot{V}O_2$ max (v-$\dot{V}O_2$ max) was taken as that which elicited the highest $\dot{V}O_2$ during the continuous incremental test. When a plateau in $\dot{V}O_2$ was observed with increasing speed, the first point on the plateau was taken as v-$\dot{V}O_2$ max.

The criterion for determination of T_{vent} was a sudden and sustained increase in respiratory exchange ratio ($\dot{V}CO_2/\dot{V}O_2$), in addition to the ventilatory equivalent for oxygen ($\dot{V}_E/\dot{V}O_2$). The T_{vent} was determined by three experienced independent reviewers without prior knowledge of the subjects under review. In all cases, at least two reviewers agreed, and the third reviewer never differed by more than 1.0 km.h^{-1} for the speed at T_{vent} (v-T_{vent}).

The training session was intended to represent a severe overload which most runners in serious training would regularly perform (Martin and Coe, 1991), and consisted of four intervals of 800 m at 1 km.h^{-1} below v-$\dot{V}O_2$ max. The treadmill was maintained at a 1% gradient since this was the gradient which has been found to best represent the energetics of outdoor running (Jones and Doust, 1996). The rest duration between each interval was 3 mins, during which time subject's opted to walk around the laboratory. Throughout the training session, heart rate (f_c) was recorded for each subject using telemetry (Sports Tester, Polar Electro Oy, Kempele, Finland).

Test 1 and 2 consisted of constant-velocity high-intensity running which was calculated to elicit a $\dot{V}O_2$ which corresponded to 40% of delta. In this case, delta (Δ) represents the difference between $\dot{V}O_2$ at T_{vent} and $\dot{V}O_2$ max. To determine the velocity which corresponded to 40% Δ for each subject, a linear regression of $\dot{V}O_2$ from running velocity during the maximal incremental test was used.

Linearity was demonstrated by a coefficient of determination (R^2) for the regression lines of 98.4% ± 1.4% (mean ± s.d.) (see figure 1). It is recognised that the relationship between $\dot{V}O_2$ and velocity increasingly deviates from linearity at intensities above T_{vent}, particularly as increment duration increases. The consequence for the present study is that the velocity predicted from the linear model actually elicited a $\dot{V}O_2$ of 51% Δ, rather than 40% Δ as predicted.

Initially, a rectal probe was inserted (10 cm into the rectum) for the determination of rectal temperature prior to and following each test. Following the individual warm-up of approximately 5 min, body mass was determined for each subject. A free-flowing capillary blood sample was then taken from each subject for subsequent analysis for determination of whole blood lactate concentration ($[La^-]_B$).

Each subject then began running for 6 min 30 s at the predetermined velocity. From 3 min to 6 min 30 s expired air was continually collected. The duration of each collection was timed, and generally lasted for ~40 s. The expired gas was subsequently analysed for determination of $\dot{V}O_2$, $\dot{V}CO_2$, respiratory exchange ratio (R) and expired minute ventilation ($\dot{V}_E$). Within 1 min following the final gas collection period a second post-exercise fingertip capillary blood sample was taken for subsequent determination of $[La^-]_B$.

Since the slow component of $\dot{V}O_2$ kinetics is operationally defined as the difference between the $\dot{V}O_2$ at 3 and 6 minutes of high intensity exercise (Whipp and Wasserman, 1972), $\dot{V}O_2$ values immediately after 3 minutes and 6 minutes of exercise were used for determination ($\Delta\dot{V}O_2$ (6-3)). A typical $\dot{V}O_2$ response to the high intensity exercise is shown in figure 2.

All testing was performed on a treadmill (Woodway ELG2, Cardiokinetics, Salford, U.K.), for which the speed was regularly checked. The number of belt revolutions were counted over a recorded time, and in combination with measurement of belt length, belt speed was determined. However, since the treadmill belt is unable to slip, and the speed of the motor driving the belt is the speed that appears on the display, any changes in speed would be evident on the display.

During test 1, test 2, and the continuous incremental test, subjects wore a nose clip and a large, broad flanged rubber mouthpiece (Collins, Mass, USA) fitted to a low-resistance (inspired < 3 cmH_2O @ 360 $L.min^{-1}$ and expired < 1 cmH_2O @ 360 $L.min^{-1}$) breathing valve of small volume (90 ml) (University of Brighton, England). The breathing valve was connected to a 200 L Douglas bag from the expired side via a 1 m length of light weight Falconia tubing of 4.0 cm bore (Baxter Woodhouse and Taylor Ltd.). Expired gas was collected for a timed period. During the incremental test expired gas was collected during the final 40 seconds of each stage, and during test 1 and 2 expired gas was collected continuously from 3 minutes. The expired gas was subsequently analysed for mixed expired O_2 fractions (F_EO_2) and mixed expired CO_2 fractions (F_ECO_2), using a paramagnetic O_2 analyser (1100 series, Servomex, Crowborough, U.K.) and an infrared CO_2 analyser (1490 series, Servomex, Crowborough, U.K.) respectively. Each analyser was calibrated at two points, and checked for linearity using high precision gas mixtures (B.O.C.) and room air. Gas volume was measured using a dry gas meter (Harvard Apparatus Ltd., Edenbridge, U.K.) previously calibrated against a Tissot spirometer, and regularly checked for linearity throughout the complete collection volume range using a 7 L calibration syringe (Hans Rudolf Inc., Kansas City, Mo., USA).

Blood lactate concentration was determined from a 25 µl sample of capillary blood collected in a heparinised capillary tube. The sample was analysed enzymatically in duplicate for $[La^-]_B$ (P-GM7, Analox Instruments Ltd., London, England), which was calibrated prior to use with an 8 mM standard.

A paired student t test was used to test for differences between test 1 and 2 in both the reliability and the experimental group. Differences were regarded as significant for a p-value of less than 0.05. Where a direction of change could be specified, a one tailed test was performed.

Results

The anthropometric characteristics of both the reliability and experimental group were similar, with little variation within each group (table 1). In both groups, the subjects were well-trained runners, demonstrated by a $\dot{V}O_2$ max of 4.77 l.min^{-1} and 4.73 l.min^{-1}, and a v-$\dot{V}O_2$ max of 19.6 km.h^{-1} and 20.0 km.h^{-1} respectively. The $\dot{V}O_2$ at T_{vent} was 79% and 77% of $\dot{V}O_2$ max respectively.

The mean heart rate response during the interval training session for the experimental group is given in figure 3. Over the four 800 m bouts of running, heart rate reached 181, 182, 184, and 183 beats.min^{-1} respectively. These values compare with a mean maximum heart rate measured during the continuous incremental test of 184 beats.min^{-1}.

The measurement of $\Delta\dot{V}O_2$ (6-3) was shown to be repeatable in the two tests performed by the reliability group, and the mean difference in $\Delta\dot{V}O_2$ (6-3) was 0.022 L.min^{-1} (7.0%) (table 2). This was in comparison to a significant difference of 0.076 L.min^{-1} (24.9%) in $\Delta\dot{V}O_2$ (6-3) between the two tests in the experimental group ($p < 0.05$) (table 2).

With regard to the actual values after 3 min and 6 min of high-intensity exercise, no significant differences were seen in either the reliability or experimental groups (table 2). However, there was a tendency for the 6 min value in the experimental group to increase between test 1 and 2 (4.51 l.min^{-1} and 4.59 l.min^{-1}). These results demonstrated that variation existed between subjects in the way in which the difference in $\Delta\dot{V}O_2$ (6-3) between test 1 and 2 was achieved.

These results were observed without any significant changes in body mass (table 3), core temperature (table 4), or $[La^-]_B$ (table 5) between test 1 and 2 for either group ($p > 0.05$). It was, however, apparent that $[La^-]_B$ had not quite returned to normal pre-test values by 1 hour following the interval training session. Despite the slightly increased pre-test value in test 2, the post-test $[La^-]_B$ was similar to that observed following test 1 in the experimental group.

Discussion

Most studies examining the kinetics of the $\dot{V}O_2$ response to high intensity exercise have used cycle ergometry to provide the exercise stress. A few studies have examined the kinetics of the $\dot{V}O_2$ response during running exercise. In 1970, Nagle and colleagues measured a 0.36 l.min^{-1} increase in $\dot{V}O_2$ between minutes 5 and 30 of treadmill running at 82-89% $\dot{V}O_2$ max, and an increase of 0.20 l.min^{-1} at 74-79% $\dot{V}O_2$ max. More recently, Sloniger et al (1996) found that during running at a speed calculated to elicit 99% $\dot{V}O_2$ max, an increase in $\dot{V}O_2$ of 0.70 $\pm$ 0.15 l.min^{-1} occurred between the third minute and the end of exercise (range 7.3 - 13.5 min). Also, Billat and Koralsztein (1996) found that during running at 90% and 100% of v-$\dot{V}O_2$ max, $\dot{V}O_2$ between 2 and 5 minutes increased by 0.23 $\pm$ 0.14 l.min^{-1} and 0.22 $\pm$ 0.19 l.min^{-1} respectively. Interestingly in this study, after 5 min of exercise at 90% v-$\dot{V}O_2$ max, $\dot{V}O_2$ failed to increase further. However, after 5 min at 100% v-$\dot{V}O_2$ max, $\dot{V}O_2$ continued to increase to the $\dot{V}O_2$ max value measured during the graded incremental test. The slow component of $\dot{V}O_2$

kinetics during running, taken as the difference between $\dot{V}O_2$ after 3 and 6 min of exercise, was measured during two identical trials in the reliability group in the present study, and on both occasions a similar value was recorded (0.30 $\pm$ 0.10 $l.min^{-1}$ and 0.32 $\pm$ 0.13 $l.min^{-1}$).

Whilst the results recorded for the reliability group in the present study do not differ markedly from the results of previous studies which used running as a mode of exercise, the use of 40 second samples of expired gas collected in Douglas bags contrasts with the breath-by-breath gas analysis technique utilised in the previous studies. A limitation of the single, relatively long sample after 3 minutes and 6 minutes of exercise is provided by the possibilities for errors. A single collection of expired gas *after* 3 and 6 minutes is clearly less reliable than several collections *at* 3 and 6 minutes. However, the use of a reliability group provided evidence that the technique used in the present study gave similar results on two occasions within the group.

A significant increase in $\Delta\dot{V}O_2$ (6-3) in the experimental group was demonstrated as a consequence of the running exercise which was performed as part of both the training session and test 1. This finding was observed in the absence of a significant change in body mass, core temperature, or blood lactate concentration prior to, or following each test. A slightly higher $[La^-]_B$ prior to test 2 in contrast to test 1 was not accompanied by a higher $[La^-]_B$ following test 2 in contrast to test 1.

Poole et al (1991) have demonstrated that ~86% of the increase in pumonary $\dot{V}O_2$ beyond the third minute of exercise could be accounted for by the increase in leg $\dot{V}O_2$ during severe-intensity cycling exercise. Energy consuming processes which originate outside the active limb are therefore thought to be a small contributing factor to increasing $\dot{V}O_2$ after 3 minutes of high-intensity exercise. Gaesser et al

(1994) have shown that increased levels of circulating plasma epinephrine concentrations (420 ± 130 pg.ml^{-1} to 2190 ± 410 pg.ml^{-1}) at 10 minutes of high-intensity exercise have no effect on the slow phase of $\dot{V}O_2$ kinetics between 10 and 20 minutes. Also, following six weeks of endurance training, Womack et al (1995) found an attenuated increase in $\dot{V}O_2$ between 10 and 20 minutes of high-intensity cycling exercise at the same absolute power. The reduced $\dot{V}O_2$ slow component was also observed when plasma epinephrine concentrations were increased to pre-training levels in the post-training condition.

Although the magnitude of the increase in $\dot{V}O_2$ after 3 minutes of high-intensity exercise has been shown to be highly correlated with the rise in blood lactate concentration, Stringer et al (1994) have postulated that it is not lactate *per se*, but the associated acidosis that causes the increase in $\dot{V}O_2$, due to the rightward shift of the oxyhaemoglobin dissociation curve which raises capillary PO_2. An increase in temperature would elicit a similar shift in the oxyhaemoglobin dissociation curve. In the present study whole body core temperature was not changed throughout exercise, or in absolute terms at the beginning and end of exercise in test 2 compared with test 1 in either group. It is, therefore, unlikely that changes in whole body temperature contributed significantly to the change in the $\Delta\dot{V}O_2$ (6-3) between test 1 and 2 in the experimental group. In any case, the effect of temperature on the increase in $\dot{V}O_2$ beyond three minutes of high-intensity exercise is not well established, since Poole et al (1988) have also found that a rising core temperature (0.81 ± 0.16 °C) during 24 minutes of high-intensity cycling can accompany a steady $\dot{V}O_2$. Conversely, during a similar duration of severe-intensity cycling (17.7 ± 1.2 min) a similar magnitude of increase in temperature (0.98 ± 0.30 °C) accompanied a large increase in $\dot{V}O_2$ after 3 minutes.

It is possible that a slowed $\dot{V}O_2$ kinetics prior to 3 minutes of running may be responsible for the increase in $\Delta\dot{V}O_2$ (6-3) in the present study. However, Gerbino et al. (1996) have demonstrated speeded $\dot{V}O_2$ fast kinetics during high-intensity exercise 6 min following a prior bout of high intensity exercise, which was attributed to the vasodilatory effect of the lactic acidosis associated with the prior high-intensity exercise bout. Associated with the speeded $\dot{V}O_2$ fast kinetics was a decreased $\Delta\dot{V}O_2$ (6-3) after the high-intensity exercise. Gerbino et al. (1996) suggested that the fast component of $\dot{V}O_2$ kinetics during high-intensity exercise are normally dictated by the muscle perfusion response to exercise, and so a prior bout of high-intensity exercise which will increase perfusion to the active musculature will speed the $\dot{V}O_2$ fast kinetics. In the present study, it is possible that the prior exercise elicited some effect on the fast component of $\dot{V}O_2$ kinetics in test 2 in the experimental group, since the $[La^-]_B$ was slightly increased prior to the start of test 2. However, the observation that the 3 minute $\dot{V}O_2$ value was not different in the two tests in the experimental group would suggest that the fast component of $\dot{V}O_2$ kinetics in test 2 was similar to that in test 1.

In an attempt to manipulate the contribution of different muscle fibre populations in the exercising limb to the slow component of $\dot{V}O_2$ kinetics, Gaesser et al (1992) measured $\dot{V}O_2$ at 3 min and at 18 min of high-intensity cycling exercise in subjects pedalling at 50 and 100 revs.min^{-1}. The change in $\dot{V}O_2$ between 3 min and 18 min was doubled in subjects pedaling at the faster cadence (0.67 $\pm$ 0.11 l.min^{-1} vs 0.38 $\pm$ 0.07 l.min^{-1}). In contrast to this finding, Barstow et al. (1996) have found no significant effect of manipulation of pedal cadence on the change in $\dot{V}O_2$ between 3 min and 8 min of high-intensity cycling at cadences ranging from 45 to 90 revs.min^{-1}, although a trend of decreasing $\dot{V}O_2$ change for increasing cadence was observed. Also in the study by Barstow et al. (1996), it was demonstrated that an inverse relationship exists between the amplitude of the slow component of $\dot{V}O_2$ kinetics expressed relative to the overall increase in $\dot{V}O_2$, and

the % type I fibres. During moderate-intensity exercise it has also been demonstrated that a relationship exists between % type I fibres and muscular efficiency during both cycling and novel knee extension exercise (Coyle et al., 1992). Some evidence seems to exist that a progressive recruitment of additional, possibly less efficient muscle fibres may be responsible for the increasing $\dot{V}O_2$ during high-intensity exercise. Shinohara and Moritani (1992) found that between minutes 4 and 7 of high-intensity cycling exercise the integrated electromyogram of the active muscles was positively correlated with the rise in pulmonary $\dot{V}O_2$. An increased recruitment of less efficient fibres has also been proposed as a factor contributing to the increased metabolic rate during prolonged fatiguing exercise (Vollestad et al., 1990), and it is conceivable that the findings following the training session in the present study are largely due to progressively greater fibre recruitment. Fatigue during isometric exercise in humans has been associated with increased $\dot{V}O_2$ in the active limb, and this increased $\dot{V}O_2$ closely mapped altered fibre recruitment patterns determined through intramuscular EMG recordings and the glycogen depletion method (Sejersted and Vollestad, 1992).

In conclusion, it is demonstrated that the slow component of $\dot{V}O_2$ kinetics can be determined reliably during running. Additionally, a significant increase in the slow component of $\dot{V}O_2$ kinetics was observed 1 hour following an interval training session. The increase in $\Delta\dot{V}O_2$ (6-3) following the training session was observed in the absence of a significant change in body mass, core temperature, and blood lactate concentration. It is possible that a changed muscle fibre recruitment pattern towards a progressively greater recruitment of less efficient type II fibres may occur following the training session. Further studies are needed to determine the response following a running training session. As well as providing an explanation for the prolonged fatigue following this mode of exercise, such studies may increase knowledge about the mechanism for the slow component of $\dot{V}O_2$ kinetics during high intensity exercise.

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These experiments comply with the current laws of the United Kingdom.

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Table 1: Group anthropometric characteristics.

	Age (yrs)	Mass (kg)	$\dot{V}O_2$ max (L.min^{-1})	v-$\dot{V}O_2$ max (km.h^{-1})	T_{vent} (L.min^{-1})	v-T_{vent} (km.h^{-1})
Reliability Group						
x	28.0	71.0	4.77	19.6	3.78	15.9
$\pm$ s.d	8.3	4.3	0.26	0.6	0.36	1.3
Experimental Group						
x	29.8	71.2	4.73	20.0	3.65	16.0
$\pm$ s.d	6.3	7.3	0.39	0.7	0.41	1.2

Table 2: Oxygen uptake at 3 and 6 minutes, and difference between 3 and 6 minute values in test 1 and 2 for the reliability and experimental group

	Test 1	Test 2
Difference in $\dot{V}O_2$ between 3 and 6 minutes (L.min^{-1})		
Reliability group	0.30 (0.10)	0.32 (0.13)
Experimental group	0.27 (0.14)	0.34 (0.08)#
$\dot{V}O_2$ at 3 min (L.min^{-1})		
Reliability group	4.26 (0.24)	4.24 (0.25)
Experimental group	4.24 (0.28)	4.25 (0.24)
$\dot{V}O_2$ at 6 min (L.min^{-1})		
Reliability group	4.55 (0.32)	4.56 (0.34)
Experimental group	4.51 (0.39)	4.59 (0.31)

Values are expressed as mean (standard deviation). # indicates significantly different value from test 1 ($p < 0.05$).

Table 3: Body mass prior to and following each test, and difference between pre-and post-test values for the reliability and experimental group

	Test 1	Test 2
Difference between pre- and post-test body mass for each test (kg)		
Reliability group	0.4 (0.3)	0.2 (0.1)
Experimental group	0.2 (0.1)	0.2 (0.1)
Pre-test body mass (kg)		
Reliability group	72.1 (2.0)	72.4 (2.3)
Experimental group	71.9 (6.3)	72.0 (6.3)
Post-test body mass (kg)		
Reliability group	71.8 (1.9)	72.1 (2.3)
Experimental group	71.7 (6.2)	71.7 (6.3)

Values are expressed as mean (standard deviation). No significant differences between tests 1 and 2 for either group ($p > 0.05$).

Table 4: Core temperature prior to and following each test, and difference between pre- and post-test values for the reliability and experimental group

	Test 1	Test 2
Difference between pre- and post-test core temperature for each test (°C)		
Reliability group	0.8 (0.3)	0.8 (0.3)
Experimental group	0.8 (0.3)	0.7 (0.2)
Pre-test core temperature (°C)		
Reliability group	37.4 (0.4)	37.4 (0.5)
Experimental group	37.2 (0.4)	37.0 (0.1)
Post-test core temperature (°C)		
Reliability group	38.1 (0.5)	38.2 (0.3)
Experimental group	38.0 (0.3)	37.6 (0.4)

Values are expressed as mean (standard deviation). No significant differences between tests 1 and 2 for either group ($p > 0.05$).

Table 5: Blood lactate concentration prior to and following each test, and difference between pre-and post-test values for the reliability and experimental group

	Test 1	Test 2
Difference between pre- and post-test $[La^-]_B$ for each test (mM)		
Reliability group	5.2 (1.6)	5.3 (1.9)
Experimental group	5.4 (1.7)	4.4 (1.7)
Pre-test $[La^-]_B$ (mM)		
Reliability group	0.8 (0.3)	1.0 (0.2)
Experimental group	0.9 (0.3)	1.8 (1.0)
Post-test $[La^-]_B$ (mM)		
Reliability group	6.0 (1.6)	6.5 (1.6)
Experimental group	6.5 (1.4)	6.4 (1.7)

Values are expressed as mean (standard deviation). No significant differences between tests 1 and 2 for either group ($p > 0.05$).

Figure 1:

Oxygen uptake for the incremental test for one subject

Figure 2:

Oxygen uptake after 3 minutes of constant-speed high-intensity running in one subject

Figure 3:

Final heart rate during each 800 meter bout of running during the training session for all subjects