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**Oxygen uptake during moderate intensity running: response following a single
bout of interval training**

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Abstract

Eight male endurance runners (mean \pm s.d.: age 25 ± 6 years; height 1.79 ± 0.06 m; body mass 70.5 ± 6.0 kg; % body fat 12.5 ± 3.2 ; VO_{2max} 62.9 ± 1.7 ml.kg⁻¹.min⁻¹) performed an interval training session, preceded immediately by test 1, followed after 1 hour by test 2, and followed after 72 hours by test 3. The training session was six 800 m intervals at 1 km.h⁻¹ below velocity achieved at maximal oxygen uptake (VO_{2max}) with 3 min recovery between each interval. Tests 1, 2, and 3 were identical, and included collection of expired gas, and measurement of ventilatory frequency (f_v), heart rate (f_c), rate of perceived exertion (RPE), and blood lactate concentration ($[La^-]_B$) during the final 5 min of 15 min of running at 50% of the velocity achieved at VO_{2max} (50% v- VO_{2max}). Oxygen uptake (VO_2), ventilation (V_E), and respiratory exchange ratio (R) were subsequently determined from duplicate expired gas collections. Body mass and plasma volume changes were measured preceding, and immediately following the training session, and before tests 1, 2, and 3. Subjects ingested water immediately following the training session, the volume of which was determined from the loss of body mass during the session. Repeated measures analysis of variance with multiple comparison (Tukey) was used to test differences between results.

No significant differences in body mass or plasma volume existed between the three test stages, indicating that the differences recorded for the measured parameters could not be attributed to changes in body mass or plasma volume between tests, and that rehydration after the interval training session was successful. A significant ($p < 0.05$) increase was found from test 1 to test 2 (mean \pm s.d.) for VO_2 (2.128 ± 0.147 to 2.200 ± 0.140 l.min⁻¹), f_c (125 ± 17 to 132 ± 16 b.min⁻¹), and RPE (9 ± 2 to 11 ± 2). A significant ($p < 0.05$) decrease was found for submaximal R (0.89 ± 0.03 to 0.85 ± 0.04). These results suggest that alterations in VO_2 during moderate-intensity, constant-velocity running do occur following heavy-intensity endurance running training, and that this is due to factors in

addition to changed substrate metabolism towards greater fat utilisation, which could explain only 31% of the increase in VO_2 .

Keywords: Oxygen uptake - running - training - fatigue

Introduction

Currently, there is no consensus of opinion regarding the effect of running exercise on oxygen uptake (VO_2) during subsequent moderate-intensity, constant-velocity running. This may partly reflect the differences between the relevant studies. Xu and Montgomery (1995) have demonstrated that VO_2 is increased immediately following a bout of prolonged moderate-intensity running exercise, whereas Morgan et al. (1990; 1996) failed to demonstrate any change in VO_2 1, 2, or 4 days following a bout of heavy-intensity exercise.

The interest shown in this area is warranted, since factors affecting VO_2 during moderate-intensity, constant-velocity running are not completely understood (Morgan et al. 1989). Also, small differences in VO_2 during moderate-intensity running between individuals have been associated with differences in performance capability (Morgan et al. 1989). The effect of prior exercise on pulmonary gas exchange variables is also of interest due to the implications for the reliability of measurement of these variables.

The purpose of this study was to examine VO_2 during moderate-intensity running, prior to and following a heavy-intensity, interval training session performed under controlled laboratory conditions. The interval training session was designed to induce fatigue, whereby performance capability would be severely reduced following the training session. The VO_2 response was examined 1 hour following the completion of the training session, and three days (72 h) later. The timing of the test 1 hour following the training session was aimed to avoid any effect of dehydration, change in body mass, or acute metabolic effects of the training session, but prior to recovery from the training session, and at a similar time of day to test 1. By 72 hours it was anticipated that the recovery process would be complete and any fatigue would be alleviated.

Methods

Eight well-trained male runners gave informed consent to take part in the study which had been approved by the local ethics committee. The subjects were all thoroughly habituated to laboratory testing procedures.

All subjects rested for 3 days prior to the start of the experiment, and no training was performed throughout the testing period. On each testing occasion subjects refrained from eating or consuming caffeine or alcohol 3 h prior to testing, wore the same running shoes and clothing, and they performed the same individual warm-up routine. Subjects were instructed to arrive at the laboratory in a rested and fully hydrated state. Tests took place at the same time of day for each subject, and subjects consumed their normal diet throughout the 3 day testing period.

On a preliminary occasion age, height, mass, sum of skin folds ($\sum\text{SkF}$) at four sites (biceps, triceps, subscapular, suprailiac), lung function measures (vital capacity, VC; forced vital capacity, FVC; forced expired volume in 1 second, FEV₁), and maximal oxygen uptake (VO_{2max}) were determined. Maximal oxygen uptake was measured for treadmill exercise (Woodway) using an incremental velocity protocol (Jones and Doust 1996). Increment duration was one minute, with an increase of 0.5 km.h⁻¹ each minute.

During exercise subjects wore a nose clip and a large, broad flanged rubber mouthpiece (Collins, Mass, USA) fitted to a low-resistance (Inspired < 3 cmH₂O and expired < 1 cmH₂O) breathing valve (University of Brighton, England) of negligible volume (90 ml), consisting of lightweight perspex tubing (T-shape) into which is mounted two rubber flap one-way low resistance valves (Mine Safety Appliances Ltd.), connected to a 200 L Douglas bag from the expired side via a 1 m length of light weight Falconia tubing of 1.5" bore (Baxter Woodhouse and Taylor Ltd.). Expired gas was collected for a timed

period of whole number of breaths during the final 45 seconds of each minute. The expired gas was analysed for O₂ and CO₂ content, using a paramagnetic O₂ analyser (1100 series, Servomex, Crowborough, U.K.) and an infrared CO₂ analyser (1490 series, Servomex, Crowborough, U.K.). 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, 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).

A standardised relative workload for each subject of 6 x 800 m intervals at 1 km.h⁻¹ below the velocity reached at VO_{2max} during the incremental protocol, with 3 min rest between each interval, was used for the interval training session; this was designed to replicate a severe overload which well-trained runners would regularly perform. Heart rate (f_c) was recorded continuously using a telemetric system (Sport Tester, Polar Electro Oy, Kempele, Finland). The training session was performed at a similar time of day (2 - 5 pm) by all subjects. Furthermore, since test 1, test 2 and test 3 were performed at predetermined times before, immediately, and 3 days following the training session respectively, all measurements were made at a similar time of day, both within and across subjects.

The protocols for test 1, 2 and 3 were identical. Prior to exercise, capillary blood samples were taken for haematocrit (Hct) and haemoglobin concentration ([Hb]) to subsequently determine plasma volume changes according to Dill and Costill (1974). Haemoglobin concentration was determined using a beta - Hb photometer transformer (Hemocue, A B Helsingborg, Sweden). Haematocrit was determined using a Micro - Hct reader (Hawksley and Sons Ltd., Lancing, West Sussex, U.K.).

Each subject ran for 15 min at a predetermined velocity corresponding to 50% of that at which they first achieved VO_{2max} on the incremental protocol. During minutes 10-12 and 13-15 the subject's expired air was collected, and ventilatory frequency (f_v), f_c and rating of perceived exertion (RPE) according to Borg (1982) were recorded. Immediately following each gas collection period the subject was instructed to stand astride the treadmill for approximately 10 - 15 seconds while a fingertip capillary blood sample was taken. The 25 μ l sample of capillary blood was subsequently analysed enzymatically in duplicate for blood lactate concentration $[La^-]_B$ (P-GM7, Analox Instruments Ltd., London, England).

Test 1 was performed immediately preceding the training session, test 2 was performed 1 hour following the training session and test 3 was performed 3 days (72 hours) following the training session. Hct and [Hb] was measured to determine changes in plasma volume (PV) prior to test 1, immediately following the training session, preceding test 2 and preceding test 3. At these four points the subjects were also weighed to determine any changes in body mass. Following the training session the subjects were instructed to consume a volume of water equal to the change in body mass during the training session.

Repeated measures analysis of variance with multiple comparison (Tukey) was used to determine the significance of differences between test 1, 2, and 3. Based on the power of the experiment, p-value of less than 0.05 was regarded as significant.

Results

Subject anthropometric details are given in table 1. All subjects completed the six intervals forming the training session. The f_c at the end of each interval ranged from 173 ± 12 (mean \pm s.d.) for the first interval to 183 ± 11 for the sixth interval, corresponding to

93% and 98% respectively of the maximum f_c recorded during the incremental test (figure 2). The average f_c during the six intervals corresponded to 97% of the average maximal heart rate recorded during the incremental test.

Physiological measurements obtained in test 1, 2 and 3 are shown in table 2. A significant difference ($p < 0.05$) existed between tests 1 and 2, and tests 2 and 3 for VO_2 , f_c , R, and RPE. The mean average increase for VO_2 from test 1 to test 2 was 3.4%, with some subjects showing an increase of up to 7.9%. The mean increase in f_c from test 1 to test 2 was 5.6%, with some subjects showing an increase of up to 11.8%. Interestingly, the subjects who demonstrated the greatest increase in f_c were the same subjects who demonstrated the greatest increase in VO_2 (figure 1). This relationship was demonstrated by a correlation coefficient of 0.912 between the increase in VO_2 and f_c between test 1 and 2. The mean decrease from test 1 to 2 for R was 4.5%, with an decrease of 9.0% for one subject. It is important that these changes are examined in relation to the value at 72 h, when each subject should be fully recovered, and therefore provide their own control. The mean average difference between test 1 and 3 for VO_2 , f_c , and R was 0.15%, 0.8%, and 1.1% (table 2).

Although not significantly different ($p < 0.05$), $[\text{La}^-]_B$, f_v and V_E tended to increase between test 1 and 2, and then decrease between test 2 and 3. No significant differences ($p < 0.05$) existed prior to tests 1, 2, and 3 for mass or plasma volume, although a significant change did occur immediately following the training session (table 3).

Discussion

The results of this study demonstrate that VO_2 during moderate-intensity, constant-velocity running is significantly increased following an endurance interval training session. Although this is only a small mean change (3.5%), we have previously

demonstrated that in our laboratory the precision of measurement of VO_2 during different exercise bouts of exercise of this intensity is within 2.1% (James and Doust, 1997).

The significant change in VO_2 was observed at a velocity which elicited 50% of the velocity at each subject's $\text{VO}_{2\text{max}}$. This equated to an average velocity of $9.5 \pm 0.5 \text{ km.h}^{-1}$ ($2.6 \pm 0.1 \text{ m.s}^{-1}$). Previous studies which have examined the effect of a fatiguing bout of running on subsequent VO_2 during constant-velocity running have all used faster constant velocities which were 12 km.h^{-1} (Morgan et al., 1990), a mean of 19.0 km.h^{-1} (Morgan et al., 1996), and 11.3 and 13.7 km.h^{-1} (Xu and Montgomery, 1995). It was noted by Morgan et al. (1994), that at lower velocities, such as the velocity used in the present study, there appeared to be more variation in VO_2 , i.e., less reliability with repeated measurements, which may partly explain the choice of the higher velocities in other studies. The choice of higher velocities may also be due to the similarity between these velocities and the velocity at which the subjects race and train. However, in such studies, the use of velocities which exceed lactic acidosis threshold (LAT) (Wasserman et al., 1973) may be unavoidably including the effects of the slow component of VO_2 kinetics (cf. Whipp, 1994). In the present study, the low $[\text{La}^-]_{\text{B}}$ responses during the three tests provides evidence that the velocity used (i.e., 50% $v\text{-VO}_{2\text{max}}$) was below the velocity associated with each subjects LAT (see table 2). Additionally, as a practical measure, lower velocity tests would be desirable if proved successful in detecting the effect of an intervention. From a practical perspective, it is also of interest that a strong relationship exists between the increase in VO_2 and the increase in f_c following the interval training session.

Our results support the findings of other studies which have used other types of fatiguing overload. Xu and Montgomery (1995) found that VO_2 measured at 11.3 and 13.7 km.h^{-1} (3.1 and 3.8 m.s^{-1}) was altered immediately following 90 min of running at 65% and 80%

of $\text{VO}_{2\text{max}}$ on a level outdoor running track. It was noted, however, that the subjects lost 1.3 kg and 1.4 kg of body mass respectively during each 90 min run, despite 688 and 806 ml of fluid ingestion. Although it has been previously reported that reductions in body mass reduce VO_2 when expressed in an absolute form during running at a constant velocity (Thorstensson, 1986), Xu and Montgomery still demonstrated an increase in VO_2 when expressed in an absolute form (i.e., $\text{L}\cdot\text{min}^{-1}$). Since this loss of body mass would have largely been sweat, which would have been derived primarily from the plasma volume, it is important to establish what the likely effect of a loss of plasma volume would be on VO_2 during constant velocity running. Saltin (1964) demonstrated that with thermal, metabolic, or thermal and metabolic dehydration of 2.72 kg (3.8% body mass), no change was evident for VO_2 at either 45% or 74% of $\text{VO}_{2\text{max}}$, despite a significant increase in heart rate. With reference to the present study, it was considered important to allow time for rehydration and restoration of plasma volume to take place prior to the assessment of VO_2 to ensure maintenance of body mass between conditions, and to control for any effects on heart rate. Since VO_2 was measured 1 h following the training session, it was important to ascertain whether the subjects had sufficient time in which to rehydrate. It is clear that no significant differences ($p < 0.05$) existed for mass or plasma volume between tests 1, 2, and 3 (table 3). It is therefore unlikely that any differences in VO_2 could be attributed to hydration status in the present study. Further support is provided by the relationship between the increase in VO_2 and the increase in f_c between test 1 and 2.

Morgan et al. (1990; 1996) performed two studies to examine the effect of 30 min of high-intensity running (89% and 90% $\text{VO}_{2\text{max}}$) on VO_2 during constant-load running (12 $\text{km}\cdot\text{h}^{-1}$ and 19.1 $\text{km}\cdot\text{h}^{-1}$) assessed at 1, 2, and 4 days following the 30 min run. In neither study did VO_2 change during the 10 minute constant-load runs. However, a fundamental protocol difference between these two studies and the present study may account for the

different findings. In the present study, VO_2 was determined after one hour when it was anticipated that significant fatigue effects due to the training session would be apparent, whereas Morgan et al. (1990; 1996), performed the first test following the 30 min run after one day of recovery. After one day it may be the case that significant recovery has taken place, particularly in moderate to well-trained runners who are familiar with this type of fatiguing overload. Although we did not measure VO_2 after one day in this study, we have observed in a separate study that after one day VO_2 had returned back to pre-training session values following an identical interval training session to that used in the present study (James and Doust, 1997).

The significant change in VO_2 was accompanied by a significant decrease (4.5%) in R. For these clearly sub-LAT tests, as VO_2 and VCO_2 both had ample time to reach a new steady state (Whipp and Wasserman, 1972), R is likely to represent the metabolic respiratory quotient. A change in substrate oxidation towards a greater utilisation of fat cannot explain all of the change in VO_2 .

When the average R value from all subjects in test 1 and 2 was considered, the decreased metabolic efficiency in test 2 as a result of the training session accounted for 31% of the increased VO_2 (i.e., 22 $\text{ml}\cdot\text{min}^{-1}$ out of the 71 $\text{ml}\cdot\text{min}^{-1}$ of O_2). This value was calculated by examining the effect of a change in R, of the magnitude of change observed between test 1 and 2, on the amount of energy produced for each litre of O_2 consumed (i.e., at an R-value of 0.89 and 0.85, 20.557 kJ and 20.352 kJ are produced for each litre of O_2 consumed) (Lusk, 1926). Since it is 1.010 times less efficient to have an R value of 0.85 as opposed to 0.89, the necessary increase in VO_2 was calculated by multiplying the initial VO_2 value measured in test 1 by 1.010 (i.e., $1.010 \times 2.129 = 2.151 \text{ L}\cdot\text{min}^{-1}$). The resulting VO_2 value would be that observed in test 2 if 100% of the change in VO_2 was

due to the change in R. Clearly, the substrate related increase in VO_2 in test 2 only explained 30% of the observed increase in VO_2 .

Another possible mechanism for the remainder of the increase in VO_2 may reside within the active muscle mass. Increases in muscle temperature within the physiological range likely to occur (3°C) can affect muscle energetics (Willis and Jackman, 1994). It is known that muscle temperature increases rapidly at the onset of submaximal exercise and then starts to level off by 10 mins (Saltin et al., 1968). Additionally, changes in core body temperature of 2 - 3°C have been demonstrated to have no discernible effect on exercise VO_2 (Rowell, 1974). It is, therefore, unlikely that temperature changes influence the results of this experiment since our measurements were made after 10 min of constant-load, moderate-intensity running. Similarly, significant elevations in circulating catecholamines are not expected at such low velocities.

Although kinetic and kinematic variables were not measured in the present study, changes in such factors have been reported following endurance running, including overstriding (Buckalew et al., 1985) and a loss of elasticity of the active muscles (Nicol et al., 1991a; 1991b).

Although not demonstrated during running exercise, increases in limb VO_2 during a fatiguing isometric exercise bout in the absence of a significant alteration in the concentration of muscle glycogen, lactate and high energy phosphates have been shown (Vøllestad et al., 1990). The proposed mechanism for this increased limb VO_2 , and one which may be of importance for this study, is a change in muscle fibre recruitment towards greater recruitment of type II fibres which are known to be less efficient (Sejersted and Vøllestad, 1992; Willis and Jackman, 1994).

Since VO_2 at rest was not measured in this study, it is not possible to discount the effect of an increased resting VO_2 on the exercising VO_2 . It has been demonstrated by Bahr et al. (1991) that following a severe physical overload lasting 3 to 4 days, with limited food intake and sleep, resting VO_2 was elevated. However, in that study the increase in resting VO_2 only accounted for 12% of the increase in exercising VO_2 .

It is also well established that an increased resting metabolic rate is related to increased core temperature. Although not in this study, in another study by our group (James and Doust, 1997), it was demonstrated that core temperature had returned to starting levels after 1 hour following the same overload as that employed in this study. Clearly, it is only possible to speculate about the potential contribution of an elevated resting metabolic rate on exercising VO_2 .

To our knowledge, the present study is the first to monitor changes in VO_2 during constant-velocity, moderate-intensity running following intense interval training. The results from this study demonstrate that VO_2 is increased following an interval training session and returns to normal within three days of passive recovery. Approximately 31% of the change in VO_2 may be explained by an altered substrate utilisation. The remainder of the change may reflect changes in muscle mechanics, fibre recruitment patterns or resting metabolic rate. Further investigations are needed to resolve this issue.

Acknowledgements

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

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 1: 1: Anthropometric data.

Age (yrs)	Height (m)	Mass (kg)	$\sum SkF$ (mm)	VO _{2max} (ml.kg ⁻¹ .min ⁻¹)
25	1.79	70.5	30.7	62.9
±6	±0.06	±6.0	±9.2	±1.7

Values are expressed as mean ± s.d., n = 8.

Table 2: Responses obtained prior to (test 1), 1 h following (test 2), and 72 h following (test 3) the training session (TS).

Measure	test 1		test 2		test 3
f_c (b.min ⁻¹)	125 ± 17	a	132 ± 16	b	124 ± 13
R	0.89 ± 0.03	a	0.85 ± 0.04	b	0.88 ± 0.03
VO ₂ (l.min ⁻¹)	2.128 ± 0.147	a	2.200 ± 0.140	b	2.125 ± 0.105
V _E (l.min ⁻¹)	43.5 ± 5.0		43.7 ± 5.3		41.6 ± 4.5
f_v (br.min ⁻¹)	29 ± 8		30 ± 9		27 ± 7
[La ⁻] _B (mmol.L ⁻¹)	1.0 ± 0.5		1.2 ± 0.5		1.0 ± 0.4
RPE	9 ± 2	a	11 ± 2	b	9 ± 1

Results expressed as mean ± s.d.

(^a indicates test 1-test 2 differences and ^b indicates test 2-test 3 differences ($p < 0.05$)).

Table 3: Body mass and plasma volume obtained prior to test 1 (pre-test 1), immediately following the training session (post-TS), prior to test 2 (pre-test 2) and prior to test 3 (pre-test 3).

Measure	pre-test 1	post-TS	pre-test 2	pre-test 3
Mass (kg)	70.8±6.4	70.0±6.5	70.8±6.3	71.0±6.7
Plasma volume change (%)	0.0±0.0	-10.0±6.1	+2.3±5.8	-1.1±11.2

Results expressed as mean ± s.d.

Legends

Figure 1: Relationship between change in oxygen uptake and heart rate 1 hour following the training session compared to values immediately prior to the training session.

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