Effects of Recovery Mode on Performance, O_2 Uptake, and O_2 Deficit During High-Intensity Intermittent Exercise

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***Key words**: fatigue, oxygen deficit, anaerobic capacity, lactate*
***Mos-clés**: fatigue, déficit d'oxygène, capacité anaérobie, lactate*

Abstract/Résumé

The aim of this study was to determine the influence of activity performed during the recovery period on the aerobic and anaerobic energy yield, as well as on performance, during high-intensity intermittent exercise (HIT). Ten physical education students participated in the study. First they underwent an incremental exercise test to assess their maximal power output (Wmax) and $\dot{V}O_2$max. On subsequent days they performed three different HITs. Each HIT consisted of four cycling bouts until exhaustion at 110% Wmax. Recovery periods of 5 min were allowed between bouts. HITs differed in the kind of activity performed during the recovery periods: pedaling at 20% $\dot{V}O_2$max (HITA), stretching exercises, or lying supine. Performance was 3–4% and aerobic energy yield was 6–8% (both $p < 0.05$) higher during the HITA than during the other two kinds of HIT. The greater contribution of aerobic metabolism to the energy yield during the high-intensity exercise bouts with active recovery was due to faster $\dot{V}O_2$ kinetics ($p < 0.01$) and a higher $\dot{V}O_2$peak during the exercise bouts preceded by active recovery ($p < 0.05$). In contrast, the anaerobic energy yield (oxygen deficit and peak blood lactate concentrations) was similar in all HITs. Therefore, this study shows that active recovery facilitates performance by increasing aerobic contribution to the whole energy yield turnover during high-intensity intermittent exercise.

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Cette étude se propose d'analyser l'effet de l'activité effectuée durant la période de récupération sur l'approvisionnement d'énergie aérobie ainsi que anaérobie et sur la performance au cours d'un exercice intermittent de forte intensité (HIT). Dix étudiants en éducation physique participent à cette étude. Dans un premier temps, ils participent à une épreuve d'effort progressif pour déterminer leur puissance mécanique maximale (Wmax) et leur $\dot{V}O_2$ max. Au cours des jours suivants, ils participent à trois HIT différents, chacun consistant en quatre séances de pédalage jusqu'à épuisement, et ce, à une intensité correspondant à 110% de la Wmax. Entre chaque séance, une période de 5 min de récupération est permise, mais les activités autorisées diffèrent d'un HIT à l'autre: pédalage à 20% du $\dot{V}O_2$max (HITA), exercices d'étirement, repos couché. La performance est améliorée de 3 à 4% ($p < 0,05$), et l'approvisionnement aérobie d'énergie, de 6 à 8 % ($p < 0,05$) durant l'HITA comparativement aux deux autres modalités de récupération. La plus grande contribution du métabolisme aérobie à l'approvisionnement d'énergie au cours de l'exercice de forte intensité suivie d'une période de récupération active est due à une meilleure cinétique du $\dot{V}O_2$ ($p < 0,01$), et à une augmentation du $\dot{V}O_2$ de crête au cours des séances d'exercice précédées d'une récupération active ($p < 0,05$). Par contre, l'approvisionnement anaérobie d'énergie (déficit d'oxygène et concentrations de pointe de lactate sanguin) est semblable dans tous les HIT. En conclusion, cette étude indique que la récupération active améliore la performance en augmentant la contribution aérobie à l'approvisionnement global d'énergie au cours d'un exercice intermittent de forte intensité.

Introduction

Many sport activities are characterized by repeated maximal or nearly maximal bouts of short-duration exercise alternated with recovery periods during which exercise is either continued at a much lower intensity (active recovery) or is interrupted (passive recovery). This is referred to as high-intensity intermittent exercise (HIT). There is a general belief in the training field that active recovery with light exercise or just stretching allows for better performance during the next periods of high-intensity exercise than does passive recovery. However, experimental data are not conclusive. While some researchers have reported greater exercise capacity with active recovery (Thiriet et al., 1993; Weltman et al., 1977), others have not confirmed these results (Bangsbo et al., 1994; Dupont et al., 2003; Saltin et al., 1992; Thiriet et al., 1993; Weltman and Regan, 1983; Weltman et al., 1979). Part the of discrepancy may be due to differences in the duration of recovery and the intensity at which the exercise bouts were performed.

If active recovery enhances performance, it should facilitate additional aerobic (Saltin et al., 1992) and/or anaerobic energy release, compared for example with passive recovery. However, the effect of active recovery on energy release and fractional contribution of aerobic and anaerobic pathways remains unknown.

During high intensity exercise, ATP demand is extremely high and energy is supplied primarily via anaerobic pathways (Bangsbo et al., 1990; 1994; Calbet et al., 2003; Karlsson and Saltin, 1970). Consequently, lactate and inorganic phosphate accumulates, pH and phosphocreatine decrease, glycolitic rate-limiting enzymes are inhibited, while ATP turnover is attenuated and, subsequently, power output is reduced (Bangsbo et al., 1991; 1992). Approximately 50–60% of lactate produced during high intensity exercise is released from the active muscles (Bangsbo

et al., 1991; 1994). Muscle lactate accumulation in turn has been associated with fatigue (Bangsbo et al., 1994; Karlsson and Saltin, 1970; Spriet et al., 1989).

There is compelling evidence of a faster blood lactate elimination with active recovery (Newman et al., 1937), as well as when static stretching exercises are performed during the recovery periods (Young and Pitt, 1996). Some researchers have reported that lactate removal is faster at intensities of 60–70% of maximal oxygen uptake ($\dot{V}O_2max$) (Gisolfi et al., 1996; Hermansen and Stensvold, 1972). Others have observed that exercise intensities close to 30% are optimal to hasten lactate elimination after high intensity exercise (Belcastro and Bonen, 1975; Boileau et al., 1983; Dodd et al., 1984). But a faster removal of lactate does not imply a faster recovery of performance during repeated sprints (Balsom et al., 1992; Bogdanis et al., 1996b).

Hypothetically, active recovery could accelerate the reestablishment of anaerobic capacity since muscle blood flow is increased (Bangsbo et al., 1994), and greater O_2 availability could facilitate the recovery process (Harris et al., 1976; Sahlin et al., 1979). Changes in the anaerobic capacity during HIT can be estimated by measuring the accumulated O_2 deficit (AOD) in each bout (Bangsbo et al., 1990; Calbet et al., 1997; 2003; Hill et al., 2002; Medbo et al., 1988; Medbo and Tabata, 1989; Weber and Schneider 2001; Weyand et al., 1999). Several assumptions are made, however. The most critical is that there should be a way to estimate energy demand during supramaximal exercise from measurements obtained during submaximal exercise (Medbo et al., 1988; Medbo and Tabata, 1989; Saltin, 1990; Tabata et al., 1997).

Stretching exercises could also facilitate recovery during HIT. Stretching exercises such as passive movements performed by external forces without muscle contraction (Bangsbo et al., 2000) could enhance muscle blood flow during the recovery periods by inducing shear stress, which causes the release of nitric oxide (Koller and Bagi, 2002). It is likely that the stretch and subsequent static contraction causes a short phase of muscular ischemia (Wisnes and Kirkebo, 1976), which could induce a transient elevation of muscle blood flow at the end of the stretching exercise by eliciting postexercise hyperemia (Osada et al., 2003).

Therefore, the main aim of this study was to determine whether performance during high-intensity intermittent exercise on the cycle ergometer can be improved more with active recovery or recovery with static stretching exercises of the lower limb muscles than with passive recovery. Another aim was to determine the influence of the activity performed during the recovery periods on the aerobic and anaerobic energy yield during the subsequent phases of high intensity exercise.

Material and Methods

SUBJECTS

Ten healthy nonsmoking physical education students participated in this study. Their physical characteristics are reported in Table 1. All were physically active but none were participating in regular sport competitions. They were asked to follow similar diets and to avoid strenuous physical activities during the 48 hours prior to the experiments. This study was approved by the ethics committee at the

Table 1 Subjects' Physical Characteristics (mean ± *SD*)

Age (years)	24.1 ± 2.0
Body mass (kg)	70.3 ± 6.6
Height (cm)	174 ± 4.0
Body fat (%)	11.5 ± 3.4
Lean body mass (kg)	58.7 ± 5.0
Wmax (w)	336 ± 48
$\dot{V}O_2$max (ml·kg^{-1}·min^{-1})	58 ± 5

University of Las Palmas de Gran Canaria. The exact nature and purposes of the study were explained to each participant, and written consent was obtained.

EXPERIMENTAL PROTOCOL AND PROCEDURES

Subjects completed seven test sessions over a period of 6 weeks. They performed the first three sessions during the first week so as to familiarize themselves with the experimental protocol and to measure maximal oxygen uptake ($\dot{V}O_2$max) and maximal power output (Wmax). During one morning, body composition was determined by dual-energy x-ray absorptiometry (QDR-1500, Hologic Corp., Waltham, MA) as previously reported (Calbet et al., 2001). Approximately one week later, the slope of the relationship between $\dot{V}O_2$/power or cycling economy was determined so as to estimate $\dot{V}O_2$ demand during high intensity cycling (see below). During the last 4 weeks, the three main trials consisting of high-intensity intermittent exercise with different activity during the recovery periods were carried out in random order.

Familiarization. Familiarization consisted of a warm-up followed by submaximal and supramaximal cycling. The warm-up involved 10 min of stretching and then 7 min of continuous cycling split between seven submaximal intensities (80, 100, 120, 140, 120, 100, and 80 W). The session continued with an incremental exercise test to exhaustion with increments of 20 W·min^{-1}.

Power Output and $\dot{V}O_2$. All cycling tests were carried out on an electrically braked cycle ergometer (Ergometrics 900, Ergo-line, Germany) at a pedal rate of 70–80 rev·min^{-1}. Ventilatory and gas exchange variables were monitored breath-by-breath via an open-circuit sampling system (CPX, Medical Graphics, St. Paul, MN) and averaged every 20 sec. The metabolic cart was calibrated with calibration gas mixtures of known O_2 and CO_2 concentrations (accuracy 0.01%) which were provided by the manufacturer (CPX, Medical Graphics). In our laboratory, $\dot{V}O_2$ and $\dot{V}CO_2$ during submaximal cycling has been assessed with a coefficient of variation lower than 5%. The highest 20-s $\dot{V}O_2$ value attained during either incremental exercise test was taken as the $\dot{V}O_2$max whereas the intensity attained just before exhaustion was referred to as Wmax. The Wmax was calculated from the last completed exercise intensity, plus the fraction of time spent exercising at the final noncompleted intensity multiplied by 20 (the intensity increment), as suggested by Kuipers et al. (1985).

Cycling Economy. A week later, cycling economy was determined by 6 to 7 submaximal workloads at intensities from 60 to 90% $\dot{V}O_2$max. Exercise intensities were administered in random order, separated by rest periods of 3–5 min. To reduce thermal stress and minimize water losses from sweating, subjects were fan-cooled and drank 100 ml of water during the resting periods. Tests were conducted at 20–24 °C, ~70% relative humidity and 740–750 mmHg atmospheric pressure. The duration of each submaximal bout was set at 6 min. The mean $\dot{V}O_2$ registered during the last 2 min was taken as representative of each submaximal load. To relate $\dot{V}O_2$ to power, we calculated individual linear or parabolic regression equations by least squares. The general model used in the parabolic regression equations was: $\dot{V}O_2 = a \cdot w^2 + b \cdot w + c$; where *a*, *b*, and *c* are constants and *w* represents exercise intensity. The equation with the lowest standard error of estimation (*SEE*) was chosen to predict supramaximal O_2 demand. The *SEE* was always lower than 50 $ml \cdot min^{-1}$ of O_2.

High-Intensity Intermittent Exercise. The cycling economy tests were followed by a resting period of approx. 30 min, then subjects warmed up and performed a supramaximal constant-intensity exercise bout until exhaustion at 110% Wmax (~120% $\dot{V}O_2$max, as estimated from the $\dot{V}O_2$/power relationship). This intensity was chosen because it produces exhaustion in about 2 min (Calbet et al., 1997), since it has been shown that a minimal duration of approx. 2 min is needed to allow for a whole utilization of anaerobic capacity (Medbo et al., 1988). The aim of this high intensity bout was to further familiarize the subjects with the supramaximal constant-intensity ride. After another week, three kinds of HITs were carried out. Each HIT consisted of four cycling bouts until exhaustion, at 110% Wmax, corresponding to a mean intensity (± *SEM*) of 370 ± 16 W. Exhaustion was defined as either the sudden interruption of exercise or the inability to maintain a pedaling rate of at least 70 $rev \cdot min^{-1}$ during a minimum of 10 consecutive seconds despite vigorous encouragement to regain the target pedaling rate (70–80 $rev \cdot min^{-1}$).

All exercise bouts were followed by 5-min recovery periods. The type of activity performed during the recovery periods was the independent variable. Consequently, the high-intensity intermittent sessions differed on type of activity performed during the recovery periods: pedaling at 20% $\dot{V}O_2$max (active recovery or HITA); stretching exercises of the lower limbs (stretching recovery or HITS); or lying supine without doing any exercise (passive recovery or HITP). Stretching exercises consisted of static stretches maintained during 20–30 sec of the hamstrings, hip extensors, sural triceps, quadriceps, and hip flexors. During the recovery periods of the HITA, subjects resumed cycling as soon as possible at an intensity eliciting a $\dot{V}O_2$ equivalent to 20% $\dot{V}O_2$max.

Before the HIT, the ear lobe was hyperemized with Finalgon® and a blood sample was taken to measure whole blood lactate concentration [La] (YSI 1500 Sport, Yellow Springs, OH). Once the blood sample was taken, subjects began warming up for 7 min as described above. The warm-up was followed by a 5-min resting period on the bike. Just before the beginning of the HIT, another blood sample was obtained for [La]. Capillary [La] was measured at the end of each bout and every minute during the recovery periods (Figure 1). The HITA, HITS, and HITP were performed in random order 10 days apart.

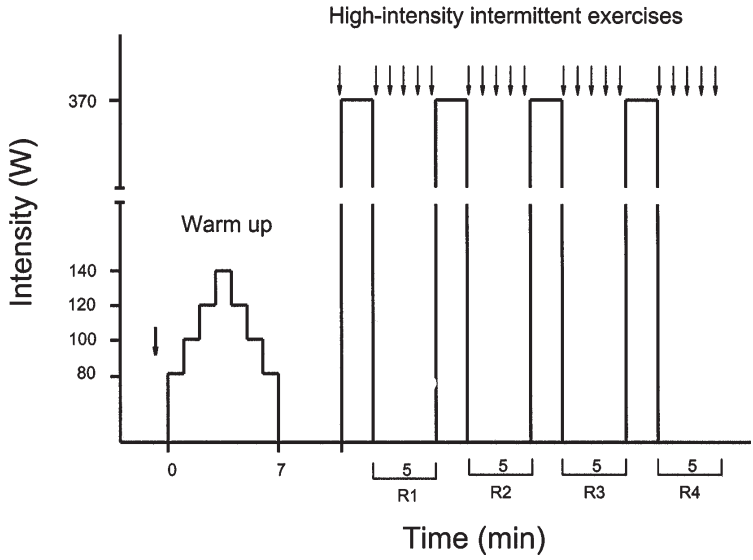

Figure 1. High-intensity intermittent exercise protocol. After a standardized warm-up subjects performed 4 bouts of supramaximal exercise at an intensity equivalent to 120% of their $\dot{V}O_2$max. Each bout was followed by a 5-min recovery period (R1 to R4) during which blood samples were obtained to measure capillary blood lactate concentration (vertical arrows).

$\dot{V}O_2$ Kinetics. To determine the kinetics of $\dot{V}O_2$ response to high-intensity exercise, we averaged breath-by-breath data every 10 sec and fit to a curve using a monoexponential model (Ozyener et al., 2001) via the least-squares error approach. To minimize any possible interference from the slow component of $\dot{V}O_2$, we included in the mathematical analysis only the data obtained during the first 120 sec of exercise. Furthermore, the first 10 sec of exercise were also excluded from the analysis, as they do not reflect actual O_2 exchange at the muscular level (Bangsbo et al., 2000). The curve-fitting procedure was iterated until any further changes in the parameters for the model did not result in a reduction in mean squared error between the curve obtained from the model and the original data set. The model used to fit the $\dot{V}O_2$ data had an amplitude term (A), a delay constant (TD), and a time constant (TC) as follows:

$$\dot{V}O_2(t) = A\,(1-e^{-(t-TD)\,/\,TC})$$

where *t* is the time in seconds and $\dot{V}O_2(t)$ is the time-dependent variation in $\dot{V}O_2$.

$\dot{V}O_2$peak. The $\dot{V}O_2$ data collected during high intensity exercise bouts was averaged every 10 sec. $\dot{V}O_2$peak was defined as the maximal 10-sec $\dot{V}O_2$ value attained during each high intensity exercise bout (Scheuermann and Barstow, 2003). Thus four $\dot{V}O_2$peaks were obtained per HIT protocol.

Average $\dot{V}O_2$. The average $\dot{V}O_2$ was calculated by dividing the oxygen con-

sumed during a given exercise bout by the corresponding endurance time in seconds. The total amount of O_2 consumed during each bout of exercise was taken as the aerobic energy yield.

Anaerobic Energy Yield. The anaerobic energy yielded during each supramaximal exercise bout was assumed to be equivalent to the accumulated oxygen deficit through the exercise bout (Medbo et al., 1988). The O_2 demand was estimated individually by extrapolating the linear or curvilinear relationship between $\dot{V}O_2$ and power output measured at submaximal loads. The accumulated O_2 demand was calculated by multiplying the O_2 demand by the supramaximal test duration. The AOD was computed as the difference between the accumulated O_2 demand and the O_2 consumed during the supramaximal bouts.

STATISTICAL ANALYSIS

Descriptive statistics were performed on each variable to confirm the assumptions of normality and homoscedasticity. The effect of type of activity performed during the recovery periods on the dependent variables was assessed using a two-way repeated-measures analysis of variance (ANOVA: 4 bouts $\times$ 3 types of recovery). Mauchly's test of sphericity was run before the ANOVA and, in cases of violation of the sphericity assumption, the degrees of freedom were adjusted according to the Huynh and Feldt test. In the case of a significant *F*-value, we carried out planned comparisons using a Student paired *t*-test with Bonferroni corrections to account for multiple comparisons. The sum of the work performed in the 2nd, 3rd, and 4th bouts of supramaximal exercise was compared between conditions using a Student paired *t*-test.

To assess the reliability of endurance time and $\dot{V}O_2$ during a constant-intensity test to exhaustion at an intensity equivalent to 110% Wmax, we determined the Cronbach alpha intraclass correlation coefficient (ICC) and the pooled coefficient of variation for the four measurements obtained on each subject (one corresponding to the last familiarization session and three others corresponding to the first bout of each HIT session) (Hopkins et al., 2001). Significance was set at $p < 0.05$. Data were expressed as means ± standard error of the mean (*SEM*) unless otherwise stated.

Results

Reliability of endurance time and $\dot{V}O_2$ during exercise to exhaustion at 110% of Wmax: Cronbach alpha ICC was $r = 0.86$ and $r = 0.90$, while the pooled coefficient of variation was 9.1 and 10.6% for endurance time and $\dot{V}O_2$, respectively.

PERFORMANCE

The work performed and endurance time in the first bout were similar in all HITs. Between the 1st and 4th bouts the work performed (and endurance time) was reduced by 50.7 ± 3.4, 54.1 ± 3.1, and 54.1 ± 2.8% in the HITA, HITS, and HITP ($p < 0.001$), respectively (Figure 2). With active recovery, the sum of the work performed in the 2nd, 3rd, and 4th bouts was 13% and 9% greater than with passive recovery and static stretching exercise recovery ($p < 0.05$, and $p = 0.08$, respectively).

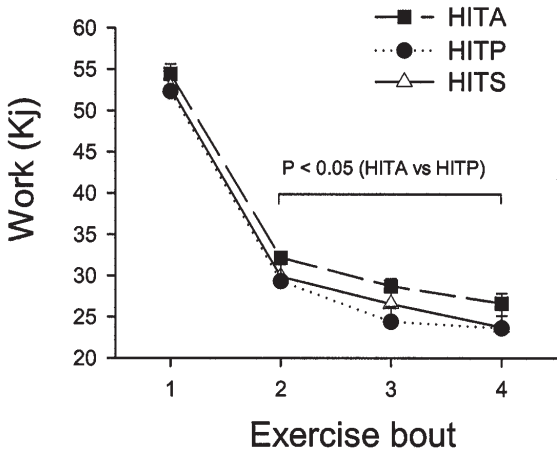

Figure 2. Work performed during high intensity exercise with active recovery (HITA), recovery with stretching exercises (HITS), and passive recovery (HITP). Values are mean ± *SEM*.

To reduce the risk of a type II error and also reduce the intertrial variability caused by small changes in the degree of exhaustion caused by the first bout of exercise, the work achieved in the 2nd, 3rd, and 4th bouts was normalized, i.e., expressed as a percentage of the performance achieved in the first bout. We conducted repeated-measures ANOVA using the normalized values (3 bouts × 3 types of recovery). The ANOVA test showed that the percentage of recovery was higher with active recovery than with the other two kinds of recovery ($p < 0.05$). A further comparison was made by calculating the mean percentage of recovery and comparing these means between conditions (paired *t*-test). This approach confirmed 3% and 4% superior work with active recovery compared with stretching and passive recovery, respectively ($p < 0.05$), while similar work was accomplished during the HITS and HITP.

AEROBIC ENERGY YIELD

The three models of HIT displayed a similar $\dot{V}O_2$peak and mean $\dot{V}O_2$ during the first exercise bout. As reflected in Figures 3 and 4, the kinetics of $\dot{V}O_2$ was also similar during the first exercise bout in each HIT, while the time constant (TC, in the model used to analyze $\dot{V}O_2$ kinetics) ranged between 26.5 ± 1.9 and 29.8 ± 1.9 sec. Between the 1st and 4th bouts the average $\dot{V}O_2$ was nearly maintained, decreasing from 46.9 ± 2.2 to 44.0 ± 2.2 $ml \cdot s^{-1}$ ($p < 0.05$). The decrease in endurance time from the 1st to the last bout, however, caused a reduction of 52, 56, and 58% in the HITA, HITS, and HITP, respectively, in total amount of oxygen consumed during the exercise bouts ($p < 0.001$).

Active recovery resulted in greater contribution of aerobic metabolism to overall energy yield during the 2nd, 3rd, and 4th bouts than either passive or stretching recovery ($p < 0.05$; Figure 5a). This was due to an enhancement of peak $\dot{V}O_2$ (Figure 5b) and mean $\dot{V}O_2$ (both $p < 0.05$), as well as to faster $\dot{V}O_2$ kinetics when the high intensity bouts were preceded by active recovery ($p < 0.05$) (Figures 3 and 4). During the HITA in fact, the time constant for mean $\dot{V}O_2$ kinetics of the

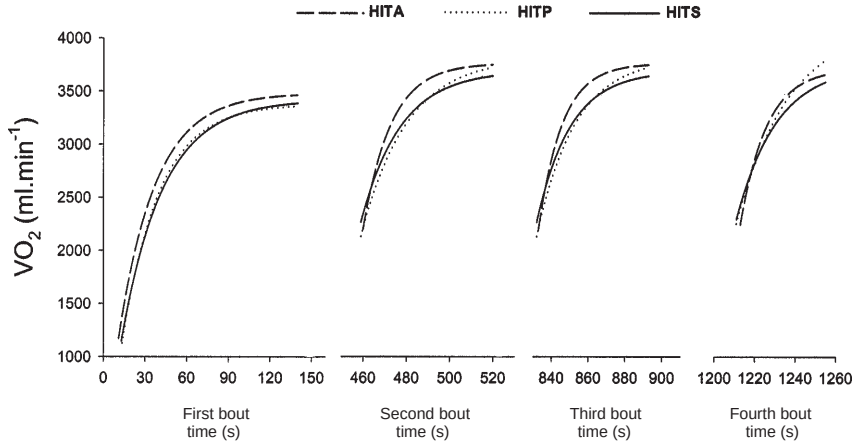

Figure 3. Mean $\dot{V}O_2$ during high-intensity intermittent exercise with active recovery (HITA), recovery with stretching exercises (HITS), and passive recovery (HITP). The data have been fitted to the model: $\dot{V}O_2(t) = A(1–e^{-(t–TD)/TC})$, where A is the amplitude term, TD the delay constant, TC the time constant, and *t* the time in seconds.

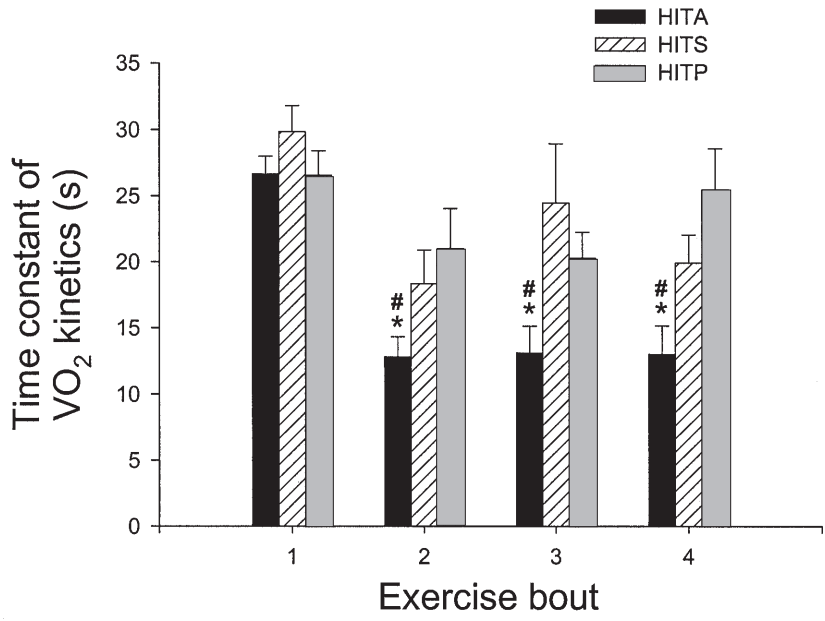

Figure 4. Time constants of $\dot{V}O_2$ kinetics during high intensity intermittent exercise with active recovery (HITA), recovery with stretching exercises (HITS), and passive recovery (HITP). Values are mean ± *SEM*. *$p < 0.05$ active recovery vs. HITP; #$p < 0.05$ active recovery vs. stretching exercise recovery.

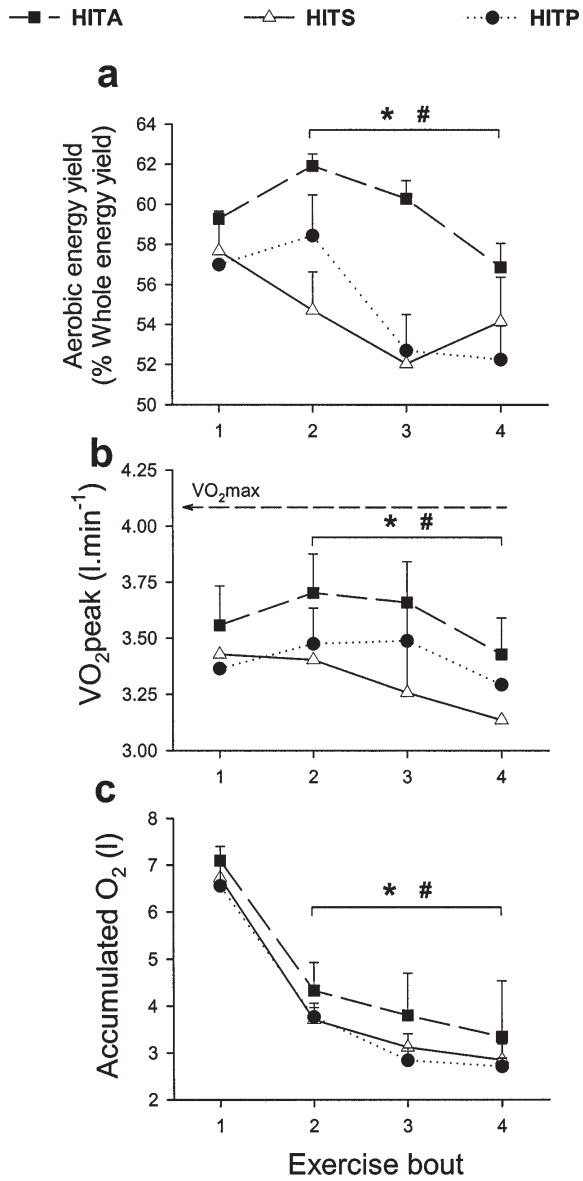

Figure 5. Effect of recovery mode on aerobic metabolism during high-intensity intermittent exercise with active recovery (HITA), recovery with stretching exercises (HITS), and passive recovery (HITP). (a) Contribution of aerobic metabolism to the energy yield during high intensity exercise; (b) Maximal O_2 consumption in a 10-sec period during high intensity exercise ($\dot{V}O_2$peak); (c) Accumulated O_2 consumed during high intensity exercise. Values are mean ± *SEM*. *$p < 0.05$ active recovery vs. HITP; #$p < 0.05$ active recovery vs. stretching exercise recovery.

2nd, 3rd, and 4th bouts was 38 and 42% lower than in the HITS and HITP ($p < 0.01$), respectively (Figure 4). Consequently, the total amount of oxygen consumed during the last three exercise bouts (11.5 ± 0.82 L) was 23 and 19% greater in the HITA than with either passive (9.32 ± 0.67) or stretching recovery (9.67 ± 1.31 L, both $p < 0.05$) (Figure 5c).

The contribution of aerobic metabolism to overall energy yield was similar for the four bouts with active recovery (57–62%). In contrast, with stretching and passive recovery, the percentage of energy derived from aerobic metabolism decreased by 6.1 ± 3.4 and 8.3 ± 1.5 % (both $p < 0.01$), respectively.

ANAEROBIC ENERGY YIELD

Between the 1st and 4th bouts, AOD was reduced in all conditions by 48–49% ($p < 0.001$). In contrast, maximal blood [La] after the 4th bout was 19–22% higher that after the 1st bout ($p < 0.001$, all conditions pooled). Neither accumulated O_2 deficit nor peak blood lactate concentration ([La]peak) were affected by the recovery mode (Figure 6).

Between the 2nd bout and the last one, blood lactate concentration just prior to each bout increased between 16 and 19% ($p < 0.001$), with no significant difference between recovery modes (Figure 6c).

Discussion

The principal finding of this study is that active recovery enhances work capacity during high-intensity intermittent exercise by increasing the aerobic energy yield without significant changes in the estimated anaerobic contribution to total energy turnover. The higher aerobic energy production is due to a faster $\dot{V}O_2$ kinetics combined with a longer working time. Nevertheless, the magnitude of improvement gained by active recovery is rather small, between 3 and 4%.

Regardless of the kind of recovery utilized, anaerobic energy release, as estimated by the AOD, was markedly reduced (40–45%) 5 min after the first bout. A similar reduction of anaerobic energy release has been reported for exhausting exercise at 120–130% of $\dot{V}O_2$max (Bangsbo et al., 1992; Saltin et al., 1992). This reduction in the anaerobic energy yield elicited by high intensity exercise has been proven to be caused by a partial inhibition of glycogenolysis and glycolysis, as reflected by reduced lactate production and not by lack of muscle glycogen (Bangsbo et al., 1992; Gaitanos et al., 1993; Saltin et al., 1992). This reduction in the glycolytic flux has been also observed during repeated all-out cycling bouts of a fixed duration of 30 sec (Bogdanis et al, 1996a; Spriet et al., 1989).

In the present study the first bout of exercise increased blood lactate concentration by 13 mmol·L^{-1} (difference between blood lactate concentration before exercise and peak [La] achieved during the 5-min recovery), while the subsequent bouts elicited only an additional increase of 1–2 mmol·L^{-1}. This finding is compatible with some degree of glycogenolytic and/or glycolytic inhibition in our experimental conditions unless a great amount of lactate has accumulated inside muscle after the 2nd and consecutive bouts of exercise. But the latter is rather unlikely since muscle lactate accumulation inhibits glycogenolysis and glycolysis (Bangsbo et al., 1992; Bogdanis et al., 1996a; Gaitanos et al., 1993; Saltin et al.,

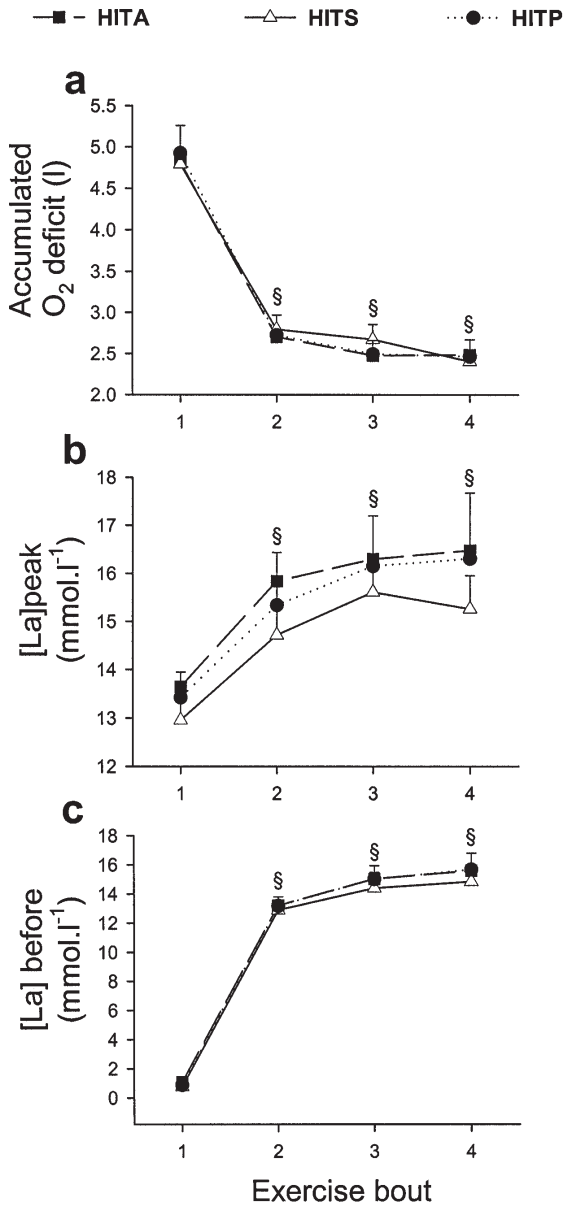

Figure 6. Effect of recovery mode on anaerobic metabolism during high-intensity intermittent exercise with active recovery (HITA), recovery during stretching exercises (HITS), and passive recovery (HITP). (a) Accumulated O_2 deficit during high intensity exercise; (b) Peak blood lactate concentration after each bout of high intensity exercise ([La]Peak); (c) Blood lactate concentration just prior to each bout of high intensity exercise. Values are mean ± *SEM*. § $p < 0.05$ compared with first bout of exercise in all conditions.

1992; Spriet et al., 1989). Moreover, it has been shown that a lack of increase in blood lactate during repeated all-out sprints is associated with a dramatic reduction of muscle lactate production (Gaitanos et al., 1993).

During the 3rd and 4th exercise bouts, anaerobic energy production tended to stabilize at around 50% of pre-exercise values. Interestingly, with the anaerobic energy release reduced by 50%, endurance time was also reduced by 50%. During the 3rd and 4th exercise bouts, therefore, all of the remaining anaerobic energy potential (only 50% of initial value) was utilized, which in turn was nearly completely reestablished to the 50% level during the 5-min recovery periods. Although there were only minor differences in $\dot{V}O_2$peak during the four exercise bouts, the amount of O_2 consumed was dramatically reduced during the last exercise bout (52–58% less than the first exercise bout).

The reduction in aerobic energy production in our experimental conditions seems at odds with the observation of increased aerobic energy production during repeated all-out sprints of a fixed duration of 30 sec (Bogdanis et al., 1996a; Spriet et al., 1989). These two studies reported increased aerobic metabolism during the 30-sec all-out exercise after the first sprint, even though mean power output was lower during the second and third sprints than during the first. To compare our findings with those of Spriet et al. (1989) and Bogdanis et al. (1996a), we calculated the O_2 consumed during the first 30 sec of exercise in the first and subsequent exercise bouts. During the first bout the mean $\dot{V}O_2$ was 2.2 liters and, in agreement with previous work (Bogdanis et al., 1996a; Spriet et al., 1989), it was significantly increased (18 to 22%) during the subsequent exercise bouts.

This study does not support an effect of the recovery strategy on anaerobic energy yield, inasmuch as the magnitude of AOD and peak blood [La] were similar in the three types of HIT. In addition, blood [La] just prior to each bout was nearly identical regardless of mode of recovery; that is, active recovery failed to significantly reduce blood lactate concentration during the recovery periods, probably due to its low intensity (Thiriet et al., 1993). Despite the latter, the best performance was achieved with active recovery.

Nevertheless, the present study clearly shows that active recovery resulted in a superior contribution of aerobic metabolism to the total energy turnover. The greater O_2 utilization was due to longer duration of the exercise bouts preceded by active recovery and a larger mean rate of O_2 utilization. In addition, the fractional contribution of aerobic metabolism to total energy turnover was almost maintained with active recovery during the consecutive exercise bouts, while it diminished by 6 to 8% when stretching exercises or passive recovery were used. Some of the improvement observed in the average $\dot{V}O_2$ can be explained by the faster $\dot{V}O_2$ kinetics during the high intensity bout preceded by active recovery. This finding is in agreement with other studies reporting faster $\dot{V}O_2$ kinetics to constant-intensity exercise when preceded by a warm-up (Bangsbo et al., 1994; Geor et al., 2000).

It should be noted that when the $\dot{V}O_2$ kinetics is speeded up, the fractional contribution of anaerobic metabolism to total energy turnover is diminished (Karlsson and Saltin, 1970). Another factor that could have contributed to the faster $\dot{V}O_2$ kinetics is the fact that baseline $\dot{V}O_2$ was already elevated at the start of each exercise bout preceded by active recovery. However, $\dot{V}O_2$ kinetics during sub-maximal constant-intensity exercise was slower when baseline $\dot{V}O_2$ was higher (di

Prampero et al., 1989; Hughson and Morrissey, 1983). An important difference between the di Prampero et al. (1989) and Hughson and Morrissey (1983) studies and the present one is that in our experimental conditions the exercise bouts were performed at supramaximal exercise intensities.

Consequently, not only was baseline increased prior to the exercise bouts but the level of blood lactate was also enhanced. In fact, a high intensity warm-up speeds $\dot{V}O_2$ kinetics more than a low intensity warm-up (Billat et al., 2000), likely due to the induction of lactic acidosis (Bangsbo et al., 1992; Gerbino et al., 1996). It is worth mentioning that a faster $\dot{V}O_2$ kinetics was associated with lower performance in Billat et al.'s study. A similar finding was obtained in the present study: high intensity exercise speeds $\dot{V}O_2$ kinetics during subsequent bouts of exercise, but performance is impaired. Interestingly, our results show that $\dot{V}O_2$ kinetics is even faster when subjects remain active between the high intensity exercise bouts by performing exercise at low intensity. This attenuates the reduction in performance caused by high intensity exercise.

Active recovery could have improved $\dot{V}O_2$ kinetics by two mechanisms: first, by increasing leg flow and consequently O_2 delivery prior to the high intensity bout (Balsom et al., 1994; Gerbino et al., 1996; Hughson and Imman, 1986). For example, $\dot{V}O_2$ kinetics is more rapid when the O_2 content of arterial blood is elevated (Balsom et al., 1994). Second, active recovery might have contributed to maintain the regulatory enzymes of aerobic metabolism at a higher level of activation, reducing the time required by these enzymes to reach full activation at the beginning of a high intensity exercise bout (Bangsbo et al., 1994; Saltin et al., 1992).

Active recovery has been reported to allow better performance than passive recovery during repeated all-out efforts (Bogdanis et al., 1996b; Signorile et al., 1993; Weltman et al., 1977), as well as during high-intensity intermittent exercise performed, like in this study, at a constant supramaximal intensity (Thiriet et al., 1993). It seems that active recovery has no effect on subsequent exercise performance when subjects are exhausted in approximately 5 min (Weltman and Regan, 1983; Weltman et al., 1979). In contrast, active recovery improves power output when performed between sprints lasting 6 to 60 sec (Bogdanis et al., 1996b; Signorile et al., 1993; Weltman et al., 1977).

Active recovery fails to show beneficial effects on performance when the intensity of recovery exercise is relative high (Dupont et al., 2003) and when recovery periods are 10 min or longer (Weltman and Regan, 1983; Weltman et al., 1979). However, Thiriet et al. (1983) reported increased performance after 20 min of active recovery vs. 20 min of passive recovery. In contrast, other studies found no effect of active recovery on performance when using exercise intensities of 35% $\dot{V}O_2$max and 50% maximal aerobic speed during the recovery period (Dupont et al., 2003; Saltin et al., 1992). For example, Saltin et al. (1992) reported a 7–8% greater performance with active recovery during HIT performed in the leg-extension ergometer. Even though the absolute magnitude of the difference was higher than that in the present study (3–4%), differences in Saltin's et al. work did not reach statistical significance.

Apart from the use of higher exercise intensities during recovery, the number of exercise bouts and level of blood lactate accumulation were lower in the Bangsbo et al. (1994) and Saltin et al. (1992) studies. Dupont et al. (2003) reported

lower performance with active recovery than with passive recovery during repeated 15-sec runs at 120% of maximal aerobic speed, but the exercise intensity during recovery (50% of maximal aerobic speed) was markedly higher than the 20% used by our subjects. Also, the recovery time of 15 sec was much shorter in Dupont et al.'s study than in present one.

The mechanism by which active recovery enhances work capacity during HIT remains unknown. However, it has been hypothesized that it can accelerate lactate and H^+ elimination (Signorile et al., 1993) and potassium clearance (Bangsbo et al., 1994), and promote the resynthesis of PCr (Yoshida et al., 1996). Despite the fact that active recovery markedly enhances leg blood flow, neither lactate (Bangsbo et al., 1994; Saltin et al., 1992) nor H^+ (Bangsbo et al., 1994) transport from the muscle to the extracellular space appear to be significantly increased. Another possibility is that active recovery could have promoted more lactate elimination within the muscle fiber itself. In fact, elegant experiments using the leg-extension ergometer, in which femoral blood flow measurements and muscle biopsies were combined, have shown that active recovery facilitates the removal of lactate (Bangsbo et al., 1994). Moreover, using nuclear magnetic resonance spectroscopy, it has been shown that active recovery accelerates the restoration of normal resting muscle pH after exercise-induced acidosis (Sairyo et al., 2003).

Active recovery might also improve performance by accelerating the restoration of ionic balance across the sarcolema of fatigued fibers. During repeated muscle contractions at high intensity, K^+ and Na^+ accumulate progressively in the external and internal side of the sarcolema, respectively, reducing muscle excitability and altering the propagation of the action potentials (Bangsbo et al., 1992; Saltin et al., 1992). The mild contractions elicited during active recovery could have potentiated the activity of the Na^+-K^+ pump, leading to a faster recovery of hydroelectrolytic balance across the sarcolema (Sjogaard, 1990). The effect of active recovery on PCr resynthesis remains unclear. It seems that active recovery could accelerate PCr resynthesis in type II fibers while slowing down PCr recovery in type I fibers (Yoshida et al., 1996), but the evidence is indirect and more studies are needed.

In summary, this study has shown that greater performance can be achieved during high-intensity intermittent exercise with active recovery at 20% $\dot{V}O_2$max than with resting recovery or just doing stretching exercises. The improvement in performance with active recovery was brought about by an increase in aerobic energy yield combined with a higher fractional contribution of aerobic metabolism to total energy turnover. In contrast, the estimated anaerobic energy production was similar in all three kinds of high-intensity intermittent exercise studied. However, it should be noted that the gain in performance observed with active recovery, while rather small, may be critical for the outcome of competitions requiring high-intensity intermittent exercises.

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