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Research article

EFFECTS OF ACTIVE VERSUS PASSIVE RECOVERY ON POWER OUTPUT DURING REPEATED BOUTS OF SHORT TERM, HIGH INTENSITY EXERCISE

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ABSTRACT

ATP repletion following exhaustive exercise is approximated to be 90-95% complete in 3 minutes, and is crucial in the performance of short duration, high intensity work. Few studies appear to have used this 3-minute interval in the investigation of recovery modes, blood lactate accumulation and power output. Thus, our aim was to investigate changes in peak power (PP), average power (AP) and blood lactate during repeated bouts of high intensity, short duration cycling, comprising active and passive recovery modes lasting 3 minutes. Seven male cyclists (age 21.8±3.3 yrs, mass 73.0±3.8kgs, height 177.3±3.4cm) performed both an active (3 min at 80rpm & 1kg resistance) and a passive recovery (no work between bouts) protocol. Following a warm-up, subjects performed six 15-second maximal sprints against a fixed workload of 5.5kg. Mean PP across the six trials was 775±11.2Watts (W) and 772±33.4W for active and passive protocols respectively; whereas mean AP was 671±26.4W and 664±10.0W, respectively. Neither was significantly different. There was a significant difference within trials for both peak power and average power ($p<0.05$), with both values decreasing over time. However, the decrease was significantly smaller for both PP and AP values during the active recovery protocol ($p<0.05$). In the current study, variation in power output cannot be explained by lactate values, as values did not differ between the active and passive protocol ($p=0.37$). Lactate values did differ significantly between trials within protocols ($p<0.05$). The results of this study suggest that an active recovery of 3 minutes between high intensity, short duration exercise bouts significantly increases PP and AP compared to a passive recovery, irrespective of changes in blood lactate levels.

KEY WORDS: Anaerobic power, light exercise, lactate, power output.

INTRODUCTION

The advantage of active versus passive recovery on subsequent performances in short duration, high intensity exercise has been well documented (Ainsworth et al., 1993; Stanley et al., 1988). Furthermore, it has been suggested that low intensity work lasting 20-40 minutes is appropriate to prevent decreased power output on repeated bouts of short duration, high intensity exercise (Bangsbo et al., 1994). High intensity exercise results in increased levels of both intramuscular and circulating levels of lactate (McLoughlin et al., 1991; Rowell et al., 1986). Furthermore, these increases in lactate, reflecting hydrogen ion concentration, have been shown to inhibit contractile performance and cause premature fatigue. Lactate removal may occur via several organs even though tracer studies using labelled lactate have shown that a significant proportion of lactate is taken up by skeletal muscle and subsequently metabolized via re-conversion of pyruvate and entry into the Krebs' cycle (Brookes, 1986). Hence, it appears that lactate removal is beneficial in terms of maintaining performance levels, and the literature reports numerous and varied protocols that are effective in reducing lactate.

Studies have used recovery periods ranging in length from 30 seconds to 40 minutes (Bangsbo et al., 1994) and the majority of investigations appear to have used recovery periods in excess of 5 minutes. However, the literature is equivocal with

regard to whether lactate reduction under certain circumstances results in improved performance (Ainsworth et al., 1993; Bangsbo et al., 1994; Rowell et al., 1986; Stanley et al., 1988a).

Athletes often train, particularly for power and speed, using recovery intervals that are much shorter than 5 minutes. Furthermore, athletes often train anaerobically to near exhaustion over short durations of 15-30 seconds. Theoretically, this is related to adenosine tri-phosphate (ATP) release and repletion. Interestingly, few studies appear to have considered the effects of recovery mode on power output in relation to ATP use and its time course to repletion. Following exhaustive exercise, adenosine tri-phosphate stores are approximated to be 90-95% repleted in three minutes. This repletion is crucial to the reproduction of short duration, high intensity work (Signorile et al., 1993). To our knowledge, few studies appear to have specifically used this three-minute interval in the investigation of recovery modes, lactate removal and more importantly power output. It would be of interest to investigate this 3-minute recovery period particularly to determine if active recovery can remove lactate in such a short time. Ratel et al. (2002) examined the effects of recovery duration on peak power output after repeated 10s sprints on a cycle ergometer, and found that in men, lactate concentration increases were reduced with a 5 min passive recovery period compared to a 30 sec or 1 min recovery. Furthermore, Hebestreit et al., (1993) in their investigation of muscle power after 1, 2 and 10 minute recovery sessions from subsequent Wingate Anaerobic Power tests, found that even after a 10 min passive recovery subjects had not returned to baseline power values. Signorile and colleagues (1993) reported significant increases in peak power and total work done over 30 seconds for active versus passive recoveries. Unfortunately, lactate values are not reported so it is not known whether the short recovery period facilitated increased lactate clearance.

Bond et al. (1991) found that after 20 minutes of either active or passive recovery from a 60sec bout of supra maximal work that peak isokinetic torque values were not significantly different between active and passive recovery modes, but that lactate values were significantly lower after 20 minutes for active as compared to passive recovery. These findings lead to interesting questions concerning the rate of metabolic re-conversion as is relates to recovery mode over the short term. Thus, the purpose of this study was to investigate changes in peak power, average power, and lactate accumulation during repeated bouts of high intensity, short duration cycling comprising active and passive recovery modes lasting three minutes.

METHODS

Subjects

Seven healthy, male cyclists (age 21.8±3.3years; mass 73.0±3.8kgs; height 177.3±3.4cm) volunteered to participate in this study. All subjects completed a health history questionnaire and signed an informed consent prior to participation. All subjects were recreationally active cyclists but were not highly trained. All procedures were approved prior to testing by The University Review Committee for the Use of Human Subjects at The University of Vermont.

Experimental Protocol

All subjects underwent both an active and a passive recovery protocol, therefore serving as their own control. A period of at least seven days separated each trial. The order of tests was randomized and both protocols were identical except for the recovery procedure. During passive recovery subjects remained stationary on the cycle ergometer for two minutes fifty seconds. Ten seconds prior to the work phase subjects pedalled lightly. During active recovery, subjects pedalled at 80rpm with 1kg resistance for three minutes (Ainsworth et al., 1993; Stanley et al., 1988). The complete anaerobic test protocol was as follows. Subjects performed a 3-minute warm up against 1kg resistance at 80rpm. Subjects then performed six, fifteen-second, all out sprints on the cycle ergometer. There were 3 minutes of recovery between each work bout. The work bout required all subjects to work against a standard workload of 5.5kg in accordance with previous relevant work (Ainsworth et al., 1993). Workloads were also standardized due to the large variation in previously reported workloads for optimal lactate clearance. We felt the use of this protocol would facilitate some comparison with previous literature.

Wingate procedures were administered in line with those described by Lakomy (1986). Subjects were given a command of 3, 2, 1 Go! All trials began from a stationary start to aid in consistency between bouts. Subjects were verbally encouraged to pedal as fast and as hard as they could until they were told to stop. Subjects were given no feedback on performance differences between protocols or trials. Upon cessation, the workload was adjusted to accommodate the recovery mode. This procedure was repeated for six trials. All anaerobic data was collected using a basket loaded Monark cycle ergometer (model 864) and power data was recorded using Sports Medicine Industries Power software V3.02 (SMI, St. Cloud, MN, USA). This allowed determination of peak power (PP), average power, time to peak (TPP), and fatigue index expressed as a

percent from PP and lowest PP, typically observed at the last data point.

Blood Sampling

All blood samples were drawn via a standard, hygienic finger puncture method. Thirty micro-litres were drawn for each sample. Samples were analyzed immediately as whole blood in duplicate using an Accusport portable lactate analyzer (Boehringer Mannheim, Indianapolis, IN, USA). Seven blood samples were drawn for each test. A sample was drawn 2 minutes following each 15-second work bout and an additional sample was drawn 5 minutes following the last work bout. Diet was not controlled in any subject prior to testing; however subjects were requested to eat the same diet on the day of testing.

Statistics

Data were analyzed using a 2 x 6 repeated measures analysis of variance (SPSS, V11.0). Alpha was set at $p<0.05$ for all analyses.

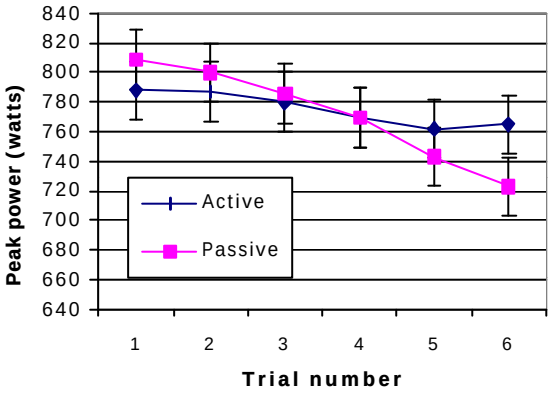

Figure 1. Peak power for both active and passive trials.

RESULTS

Lactate data are reported in mmol· l^{-1}. Data show no significant differences in mean peak power output across six trials between recovery modes, 775±11.2 watts versus 772±33.4 watts, for active and passive protocols, respectively ($p=0.785$, F 0.08). No differences were observed for average power, 671±26.4watts versus 664±10.0 watts, for active and passive, respectively. Figure 1 shows power values for peak and average power within and between trials. As one would expect there were significant differences observed within trials for both peak power and average power ($p<0.004$, F 4.47) with both values decreasing with time. A significantly greater decrease was observed in the passive protocol ($p<0.002$, F 4.78). Average power was significantly different within trials, and decreased over time ($P<0.008$) but not between protocols ($p=0.57$). These changes in power output do not appear to be explained by changes in lactate values as lactate concentrations did not differ significantly between protocols ($p=0.37$), 9.09±2.37 mmol· l^{-1} versus 10.05±2.84 mmol· l^{-1}, for active and passive, respectively. However, values did differ within protocols between trials as lactate values increased over time ($p<0.001$, F 6.4). Lactate data is presented in Figure 2.

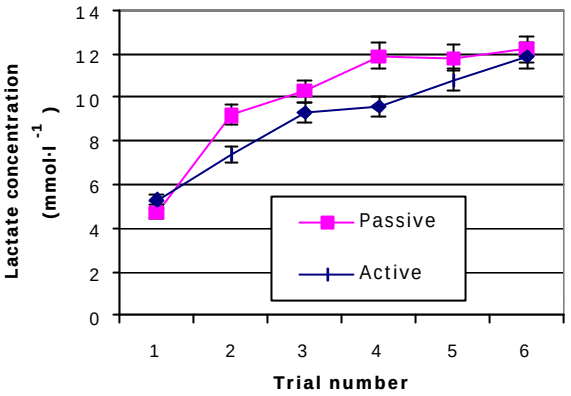

Figure 2. Lactate data for both passive and active trials.

DISCUSSION

The present study focused on a short (3 minutes) recovery period, a protocol not widely tested to our knowledge, though widely practiced by athletes. In agreement with the findings of Bangsbo and colleagues (1994), we found no statistically significant difference in blood lactate concentration between the active and passive protocols for periods up to 3 minutes. However, the same authors found that when the recovery period extended beyond three-minutes there was a higher net metabolism of lactate within active muscle during recovery.

One suggested mechanism for this reduction in lactate concentration is the distribution of circulating lactate to sites of metabolism such as the liver, heart and previously inactive muscle (Belcastro and Bonen, 1975). However, others have suggested that lactate is taken up and oxidized by mild to moderately active skeletal muscle during recovery (Brookes, 1986; Thiriet et al., 1993).

Lactate has long been recognized as a metabolite that accumulates during exercise and contributes to muscle fatigue (Gregg et al., 1984; Hetenyi et al., 1983; Jorfeldt, 1970), and that during exercise a rise in epinephrine causes an increase in lactate accumulation (Roth and Brooks, 1990; Stanley and Lehman, 1988). It is also well

established that following intense exercise, low intensity recovery exercise results in a reduction of circulating lactate (Dodd et al., 1984; Hetenyi et al., 1983).

Studies examining recovery periods and reduction in circulating lactate are equivocal (Bangsbo et al., 1994). Bangsbo and colleagues (1994) examined lactate concentration in muscle biopsy samples taken from active and inactive muscle during recovery and found that lactate concentration was similar up to 10 minutes into recovery and that arterial and venous lactate concentration showed a similar trend over the same period. Other studies have demonstrated that low intensity exercise has minimal effects on blood lactate until 15-20 minutes into recovery (Dodd et al., 1984; Hermansen and Stensvold, 1972).

Active recovery has been advocated because it is thought to aid in lactate removal following intense exercise, hence reducing performance decrements in subsequent bouts. This study demonstrates differences in power output as a function of recovery mode, but those differences are not readily explained by lactate values. Our data demonstrate a consistent and expected pattern of diminishing power as subjects advanced from the first to sixth trial. Our within trial lactate data pattern was also as expected with lactate concentrations increasing with advancing trials. However, we considered that we might see a difference in the lactate pattern between our protocols. No such difference was observed.

The concentration of circulating lactate is also a function of the intensity of exercise. It is well established that the potential for lactate production is highly dependent upon the rate of glycogenolytic / glycolytic flux, and it is exercise intensity that determines the flux rate in these pathways. In the current study a fixed resistance of 5.5kg was used for each subject independent of body mass. The main reason for this was that in the majority of previous relevant and cited work, this workload was used, therefore allowing us to make comparisons. Additionally, our body mass values have a small range with relatively small standard deviations, which serve to reduce the concern over this limitation.

During short-term high intensity bouts of exercise, skeletal muscles become rapid producers of lactate and consequently lactate clearance is slowed. Later, during recovery there is a transition to a net lactate uptake from the blood by previously active skeletal muscle. The mechanism responsible for lactate flux during the first few minutes of exercise is unclear. However, it appears that the muscles responsible for initial increases in lactate concentration during intense exercise require a recovery period in excess of three minutes to modify intracellular lactate metabolism resulting in lactate gradient in favour of uptake by the same muscle. The explanation of a lactate gradient is incorporated in the lactate shuttle hypothesis (Brooks, 1986). So, can lactate levels explain any variation in power output? We found no statistical difference in lactate accumulation between trials. Although there was statistical difference in both average and peak power output within and between trials, we are unable to explain it using lactate. This is interesting, especially in relation to peak power, as one would expect high levels of lactate to inhibit high power production. It is apparent that with active recovery blood flow is maintained or increased to the muscle. However, it should be noted that the intracellular lactate concentration was not measured in the present study. Thus, although the plasma lactate concentrations were similar for both recovery protocols, the enhanced blood flow during the active recovery may have allowed for a decreased intracellular lactate concentration without a concomitant decrease in plasma lactate. Our methods did not address this issue. It is quite likely that blood flow plays a key role in the repletion of ATP, perhaps via creatine phosphate resynthesis, leading to a lesser decrement in power output. This was not one of our objectives and again our methods did not address this issue. An increased facilitation of aerobic metabolism to the energy supply may also have contributed to the maintenance of power output.

CONCLUSIONS

The results of this study suggest that there do not appear to be any significant effects in terms of lactate clearance using active versus passive recovery between bouts of high intensity, short duration exercise. While our findings appear to add to the equivocality of existing findings the nature of our protocol should be considered. We are unaware of any work that has utilized this three-minute recovery phase and this may explain any variation in findings.

One should note that a comparison of power output from first to last bout of exercise showed a 2.5% higher power output for the passive protocol after trial one versus a 5.5% higher output for the active protocol after trial six. Additionally, one should consider that the decrease in power output from bout one to bout six during the passive protocol was 10.6% but only 2.9% for the active protocol across the same time frame. These numbers are substantial findings in terms of both power output and their implications for performance. Thus, while active recovery provides some benefit in terms of increased peak power, we cannot explain this using a reduction in plasma lactate. The mechanism for

improved power production with active recovery warrants further investigation.

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