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Original scientific paper

EFFECTS OF DIFFERENT TIMED RECOVERY INTERVENTIONS ON BLOOD LACTATE LEVELS DURING HIGH INTENSITY INTERVAL EXERCISE IN COLLEGE AGED STUDENTS

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Abstract

Aim: HIIT (Hit Intensity Interval Training) consists of low volume, but increased intensity in a short period of time followed by a relative recovery period, which can be active or passive. In addition, the implementation of rest recovery intervals between high intensity short duration activities is optimal for maintaining exercise functionality and has been shown to improve overall exercise performance. The objective of this study was to evaluate the effects of a long and short rest recovery intervention on blood lactate accumulation between multiple sets of sprinting activities.
Methods: Forty students aged 21.9 ± 0.3 years participated in the high intensity sprinting sessions. Subjects were randomly assigned into recovery conditions (modality x duration) which was one of four groups: active recovery for 10 minutes (AR10), active recovery for 5 minutes (AR5), passive recovery for 10 minutes (PR10), or passive recovery for 5 minutes (PR5). Blood lactate, heart rate, RPE, and sprint time were collected after each sprint (3-repeats) to assess performance changes. Differences in dependent variables were assessed using two-way repeated measures ANOVA.
Results: A statistically significant difference was observed between the AR10 and PR5 intervention groups for blood lactate concentrations at 5-minute post exercise ($p = .034$) and for the mean lactate values across trails ($p = .031$).
Conclusions: Active recovery could be more beneficial for lactate clearance when compared to passive recovery during high-intensity interval training and longer recovery periods incorporating physical activity may have a greater benefit of optimizing exercise performance during high intensity power activities.

Keywords: Recovery, lactate clearance, high-intensity exercise, performance.

1. Introduction

High-intensity interval training (HIIT) is a type of exercise training that can be utilized to elicit improvements in cardiorespiratory fitness across different populations. Studies have shown that a structured HIIT program can increase VO2max and resting glycogen content, reduce the rate of glycogen diminishment, increase muscle lipid oxidation, improve vascular structure and function, reduce lactate accumulation, and improved time to exhaustion similar to adaptations attained during continuous, aerobic training (Gibala et al., 2012). And it has been shown that sprint interval training alternating sessions of maximal exercise intensity coupled with varying rest recovery interventions has been demonstrated in different populations to produce improvements in aerobic and anaerobic power (Richards et al., 2010; Trilk et al., 2011; Freese et al., 2013). Wenger and bell (1986) have suggested that the greatest challenge to increasing aerobic power occurs when exercise intensity is between 90 and 100% of maximal O2 consumption (VO2max). And this high intensity exercise performed at levels greater than an individual's lactate threshold, which subsequently correlates with excess accumulation of lactate in the working skeletal muscles, can lead to an induced muscle fatigue (Karlsson et al., 1975; Stamford et al., 1981) which can disrupt muscle contractility. Thus, the purpose of recovery interventions needs to be focused on modalities that encourage optimal performance while simultaneously reducing excess accumulated lactate production during high intensity exercise.
At low exercise intensity working skeletal muscles will oxidize the accumulating lactate, and since the lactate

redistribution occurs via the blood flow (Gladden, 2004), active rather than passive recovery after lactate accumulation exercise has been shown to be more effective clearing excess lactate accumulated (Belcastro & Bonen, 1975; Boileau, Misner, Dykstra, & Spitzer, 1983). While no clear strategy has been defined it has been shown that active recovery interventions (i.e., 30-40% of VO2max) have been documented to promote an increased blood lactate clearance when performed between bouts of high-intensity exercise (Monedero & Donne, 2000; Billat et al., 2001; McAinch et ala. 2004). Similar research has demonstrated that active recovery intervals from 30 seconds to 40 minutes (Bangsbo et al. 1994) can have positive effects of improving power output and exercise performance with most of the research being conducted in recovery periods lasting greater than 5 minutes. Hence, the removal excess lactate may be beneficial in terms of maintain exercise performance. These findings lead to interesting questions relating to active and passive recovery (modality x duration) on the rate metabolic re-conversion and which has the greater training effect on assisting with maintaining exercise performance. With no concessive definition having been created regarding the optimal time for active recovery intervention the purpose of this study was to investigate different timed active recovery interventions on blood lacate accumulation when performing repeated sprinting activities in healthy young adults.

2. Methods

2.1. Participants: Forty non-athlete subjects (Male: 30, Female: 10) ages 19-28 years old were recrutied from the student population for particiaption in this study (Table 1). No significant differences were observed for baselime measures for the subjects in the intervention groups. For intital inclusion into the study subjects were current university students, classified as a non-university athlete or trained recreational athlete (< than 3 training sessions per week for a sporting activity), and free of cardiovascualr disease as assessed by the health history questionnaire (HHQ) and PARQ. Institutional Review Board approaval was obtained prior to any data collection and informed consent was collected from all participants after a detialed explanation of the aims, benefits, and risks involved with this investigation.

2.2. Preliminary screening: All subjects completed an informed conmsent, PARQ, HHQ, and a blood history questionnaire before being granted clearance to participate in this research project. A blood history questionnaire was used to assess a subject's risk for coming in contact with any blood borne pathogens prior to the study, to be cleared for the finger prick blood analysis procedure, and to provide safety for the laboratory technicians. Demographic data which indclude weight, height, fat mass, and fat free mass was collected along with resting blood pressure and heart rate. Bioimpedance analysis was used to quantify individual body composition and was condcuted using a the Inbody 520 (Version 520DM-1520; Biospace, Inc., Los Angeles, CA, USA). Participants were asked to stand for 15 minutes prior to testing to allow for compartmentalization of blood and fluid according to the manufacturer's guidelines. Subjects were instructed to not consume a meal two hours prior to arrival, void their bowels within 30 minutes, to remain hydrated, and to have abstained from moderate to intense exercise for 24-hours before the screening and testing sessions. Upon arrival at the Human Performance lab subjects were randomized into one of four groups: active recovery 5, active recovery 10, passive recovery 5, or passive recovery 10.

2.3. Testing Protocol: Upon arriving at the laboratory testing facility all subjects were asked if they adheared to the pre-exercise instructions. Resting heart rate (Polar Heart Rate monitor, model FT7), RPE and blood lactate (BL) concentration were collected after a 5-minute seated resting period. Following data collection, the subjects completed a brief 5-10 minute dynamic warm-up (50% Max Heart Rate) instructed by the exercise physiology technicians who were conducting the testing sessions. The subjects completed three sets of wind sprints seperated by the recovery interventon of 5-minutes or 10-minutes of active recovery (AR) or passive recovery (PR). The sprint sets consisted of the subjects accelerating from point A in a straight line covering a distance of 12.5 yards (37.5 feet) and then deaccelerating to the point B marker. They then turned and

accelerated in the same manner from point B to the point A marker completed a distance of 25 yards. This was repeated two additional times for a total distance of 75 yards for one set of wind sprints which was then followed by the recovery intervention. Following recovery, the subjects then completed set 2 and set 3 with the same recovery intervention used between sets. Between each sprint set blood lactate, heart rate, RPE and sprint duration were recorded before the subjects performed their rest relief intervention. Following the third set of variable measurements the subjects then performed a 15-20 minute light/moderate aerobic cool down that consisted of walking coupled with dynamic lower body movements to facilitate the oxygen transport of the tissues and assist with clearing excess blood lacate levels.

2.4. Blood Sample: Samples were drawn with a standard hygenic finger puncture method and samples were analyzed as whole blood following each sprint test. All samples were measured twice in succession at the same sight using two different Nova plus blood lactate analyzers (Nova Biomedical, Waltham, MA). Portable blood lactate meters were used to quantify lactate accumulation have been shown to be an accurate and reliable method in field testing (Pyne et al., 2000). Samples were drawn within 30 seconds of completeing the exercise work bout before starting the recovery intervention.

2.5. Recovery Intervention protocol: The AR group completed a 5-minute or 10-minute aerobic walking on a flat surface at 55-65% of their maximal age predicted heart rate which corresponded to a walk pace of 2.2-2.5 mph. Heart rate was monitored using a standard polar Ft1 heart rate monitor (Polar Electro USA, Bethpage, NY). The PR group's passive intervention was comprised of remaining in a seated position for the intervention duration with no bipedal movement for either 5 or 10 minutes. All subjects were monitored closely by certified exercise physiologists for recovery completion and any abnormal changes in health or wellness.

2.6. Statistical Analysis: Group differences with respect to demographic, anthropometric, and baseline variables were assessed via one-way ANOVA. A general linear model repeated measures ANOVA (2 x 2 x 4 design, modality x duration x time) was performed to assess the effects of repeated sprint trials and recovery modality with top-down application of univariate ANOVA's and Tukey pairwise comparisons as needed. All statistical analyses were performed using SPSS v.26 (IBM Corp., Armonk, New York). Alpha level was set to 0.05 for all tests.

3. Results

Table 1. Participant baseline characteristics.

	AR 5	AR 10	PR 5	PR 10
N	10	10	10	10
Age (yrs.)	21.7 ± 2.2	22.3 ± 1.3	20.9 ± .74	22.8 ± 1.3
Weight (kg)	74.2 ± 12.6	80.3 ± 17.2	76.6± 15.9	83.1 ± 15.6
Height (inches)	77.4 ± 5.0	78.1 ± 4.4	77.6 ± 4.3	80.7 ± 5.5
LBM (kg)	62.1 ± 14.6	67.3 ± 14.1	59.9 ± 12.3	68.8 ± 12.7
FM (kg)	11.9 ± 4.2	13.0 ± 5.3	16.6 ± 10.7	14.4 ± 10.1
BF%	16.9 ± 7.1	16.0 ± 5.5	21.1 ± 11.0	16.8 ± 9.9
Resting BL (mmol/L)	1.2 ± .52	1.2 ± .18	1.2 ± .56	1.1 ± .52

Values are mean±SD. n, number of subjects. FFM, fat free mass. FM, fat mass, BL, blood lactate,

3.1. Blood lactate concentrations: Blood lactate concentration was normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). There was homogeneity of variances, as assessed by Levene's test of homogeneity of variance ($p > .05$). Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi 2(2) = 36.45$, $p = .001$. There was no statistically significant interaction

between the intervention and time on blood lactate concentration, F(9, 108), p = .075, partial η2 = .147. The main effect of time showed a statistically significant difference in mean blood lactate concentration at the different time points, F(3, 108) = 76.980, p = .001, partial η2 = .681. The main effect of group showed that there was not statistically significant difference in mean blood lactate concentration between intervention groups F(3, 36) = 1.821, p = .161, partial η2 = .132. One-way ANOVA demonstrated a statistically significant difference in post 5-minute blood lactate concentrations and mean lactate values between the AR10 and the PR5 groups (table 2).

Table 2. Blood lactate values taken after each timed sprint.

BL (mmol)	AR 5	AR 10	PR 5	PR 10
Sprint test 1	9.3 ± 2.3	9.7 ± 21	10.4 ± 3.0	10.7 ± 1.5
Sprint test 2	13.7 ± 2.2	12.3 ± 3.1	14.3 ± 2.1	13.9 ± 1.7
Sprint test 3	15.1 ± 2.3	13.6 ± 3.4	15.7 ± 1.4	15.8 ± 1.7
Post 5-min	14.5 ± 2.2	11.4 ± 3.9*	14.9 ± 1.7	13.1 ± 2.6
Mean Values	12.7 ± 1.7	11.9 ± 2.6**	13.5 ± 2.0	13.2 ± 1.4

*Values are mean±SD, AR, active recovery. PR, passive recovery. * = significant difference between AR10 and PR5, p = .034, One-way ANOVA*

Table 3. Intervention variables after the completion of each exercise trail set.

Time (sec)	AR 5	AR 10	PR 5	PR 10
Sprint 1	28.7 ± 2.9	28.5 ± 1.9	29.8 ± 2.3	28.6 ± 2.6
Sprint 2	29.2 ± 2.9	28.6 ± 2.2	29.7 ± 3.2	28.8 ± 2.8
Sprint 3	29.5 ± 2.9	28.4 ± 2.1	31.1 ± 2.7	29.3 ± 3.2
Mean Time	29.1 ± 2.8	28.5 ± 2.1	30.2 ± 2.6	28.9 ± 2.8
Heart rate	AR 5	AR 10	PR 5	PR 10
Sprint 1	175.5 ± 10.9	172.4 ± 19.2	181.8 ± 9.2	181.1 ± 6.2
Sprint 2	177.1 ± 7.5	183.3 ± 8.6	183.2 ± 7.6	182.9 ± 7.2
Sprint 3	179.7 ± 5.2	182.7 ± 7.4	182.6 ± 6.9	182.9 ± 7.8
Mean HR	177.4 ± 6.6	179.4 ± 9.8	181.6 ± 7.3	182.2 ± 6.5
RPE				
Sprint 1	13.5 ± 12.1	14.6 ± 1.8	13.8 ± 1.1	15.3 ± 1.9
Sprint 2	15.0 ± 1.0	15.0 ± 1.6	14.7 ± 1.1	15.9 ± 1.9
Sprint 3	16.8 ± 1.6	15.9 ± 1.9	16.2 ± 1.0	16.9 ± 2.0
Mean RPE	15.1 ± 1.0	15.2 ± 1.6	14.9 ± 1.6	16.0 ± 1.8

Values are mean±SD, AR, active recovery. PR, passive recovery.

3.2. Sprint duration time: Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction, $\chi 2(2) = 1.423$, p = .491. There was a statistically significant interaction between the intervention and time on sprint intervention duration, F(2, 72) = 2.389, p = 0.31, partial η2 = .166 (table 3). Conversely there was a statistically significant effect of time on sprint duration for the 5-minute passive recovery group, F(2, 72) = 4.899, p = .002, partial η2 = .352. The main effect of group was not statistically significant different for mean sprint duration at the different testing points, F(2, 36) = .800 p = .502, partial η2 = .06.

3.3. Heart rate: Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction, $\chi 2(2) = 39.554$, p = .001. There was a not a statistically significant interaction between the

intervention and time on heart rate values, $F(2, 72) = 118.671$, $p = .187$, partial $\eta 2 = .120$. The main effect of time showed a statistically significant difference in mean heart rate values at the different testing points, $F(2, 72) = 224.175$, $p = .008$, partial $\eta 2 = .126$. The main effect of group showed that there was a not statistically significant difference in heart rate values between intervention groups $F(3, 36) = 180.297$, $p = .392$, partial $\eta 2 = .079$.

3.4. Ratings of perceived exertion: Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction, $\chi 2(2) = 4.707$, $p = .095$. There was a not a statistically significant interaction between the intervention and time on RPE values, $F(2, 72) = 1.849$, $p = .102$, partial $\eta 2 = .134$. The main effect of time showed a statistically significant difference in mean RPE values at the different testing points, $F(2, 72) = 42.889$, $p = .001$, partial $\eta 2 = .544$. The main effect of group showed that there was a not statistically significant difference in RPE between intervention groups $F(3, 36) = 1.347$, $p = .274$, partial $\eta 2 = .101$.

4. Discussion and conclusions

Published research has demonstrated that active recovery modalities between bouts of high intensity exercise (supramaximal effort) aid in reducing the quantity of blood lactate accumulation during the exercise (Billat, 2001; Billat et al., 2001; McAinch et al. 2004). The current study investigated the use of different timed active recovery intervention modalities on blood lactate accumulation during high intensity interval exercise. It was observed that longer active recovery modalities (10 minutes) produced lower blood lactate values 5-minutes post training and lower mean values. This was demonstrated to be significant between the 10-minute active recovery and the 5-minute passive recovery intervention groups (table 2). This could imply that longer interventions of light aerobic activity could have an attenuation effect on accumulating blood lactate compared to an intervention of a short duration consisting of seated rest recovery.

It has been recognized that high intensity exercise performed above an individual's lactate threshold can lead to an excess accumulation lactate in the working skeletal muscles during repeated bouts interspersed with recovery periods. The reason for lactate accumulation is that more of the pyruvate is converted to lactate-by-lactate dehydrogenase, primarily as a result of changes in the intramuscular redox state, and because oxidation of the excess lactate relies on redistribution by the blood flow to other muscles and the heart and liver (Gladden, 2004; Wasserman, Beaver, & Whipp, 1986). This leads to muscle lactate mirroring blood lactate, and it's been demonstrated that elevated levels of skeletal muscle and blood lactate are associated with impaired skeletal muscle function and exercise performance (Minshull et al., 2007). Exercise performance for this current study was quantified as time to complete one specific sprint with optimal performance be measured as the lowest time to completion. Both the 10-minute recovery interventions demonstrated lower mean sprint times for the testing with the active recovery yielding the fasted time to completion. This could suggest that longer active recovery periods could assist more in maintaining exercise performance.

It has been established that the duration of the exercise as well as the rest interval separating successive sprints are crucial factors for performance maintenance (Holmyard et al. 1987; Balsom et al. 1992a, b).

Active recovery interventions at low intensities have been demonstrated for reducing accumulated blood lactate including the redistribution of lactate to the metabolic tissues such as the liver, heart, and inactive muscle (Belcastro & Bonen, 1975) and by being oxidized by skeletal muscle during recovery (Brooks, 1986, Thiriet et al., 1993). And the redistribution oxidation strategies can assist in clearing excess accumulated skeletal muscle and blood lactate which is important for supporting training adaptations that occur from subsequent overloading through high intensity training paradigms.

In summary, our results suggest that 10-minute active recovery periods may have a greater preservation effect on exercise performance by lower blood lactate concentrations compared with a passive recovery intervention and therefore could have implications in training or sporting events that incorporate repeated sprinting activities. Shorter rest periods comprised of stationary rest subsequently produced greater quantities of blood

lactate during and post 5-minutes following exercise compared to active recovery of a longer nature. This leads us to conclude that longer rest recovery periods comprised of lower intensity aerobic exercise can have a positive effect of reducing blood lactate levels following lactate accumulating exercise.

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