

EFFECTS OF SHORT DURATION ACTIVE AND PASSIVE RECOVERY ON BLOOD LACTATE ACCUMULATION DURING HIGH INTENSITY WIND SPRINTS IN COLLEGE AGED STUDENTS

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Abstract

Aim: Sprint exercise training has become a popular training method for increasing aerobic and anaerobic performance in individuals of differing athletic abilities. 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 short rest recovery intervention on blood lactate accumulation between multiple set sprinting activities. **Methods:** Thirty-six healthy college student's age 18-23 yr. participated in the high intensity sprinting exercise sessions. Participants were randomized into an active recovery (AR) or passive recovery intervention (PR). Both groups performed three repeated sets of wind sprints (75 yards each set) separated by a 3-minute rest recovery. Blood lactate, HR, RPE, and sprint time were collected after each set of sprints to assess performance changes. Differences in dependent variables were assessed using two-way repeated measures ANOVA. **Results:** Blood lactate levels were significantly different between the three sets of sprints for both the active and passive recovery groups. Between group differences were observed with the active recovery group displaying statistically significant lower lactate concentrations following sprint set two and three compared to the passive recovery intervention group. **Conclusions:** These results suggest that a short active recovery intervention during high intensity sprinting activity could have positive effects of lowering the magnitude of blood lactate accumulation during the activity.

Keywords: Blood lactate, active recovery, sprinting

1. Introduction

Maximal oxygen consumption is considered one of the most important physiological determinants when investigating cardiorespiratory fitness in different populations. Effective training methods focusing on moderate to submaximal intensity for enhancing aerobic capacity have been definitively established, but research has recognized that training methods that focus

at near maximal oxygen consumption levels may be an effective addition to training paradigms in this regard (Wenger & Bell, 1986; Midgely et al., 2016). Sprint interval training alternating sessions of maximal exercise intensity paired with varying rest period interventions has been demonstrated in different populations to elicit improvements in aerobic and anaerobic power (Burgomaster et al., 2006; Burgomaster et al., 2008; Richards et al., 2010; Trilk et al., 2011; Freese et al., 2013). This type of high intensity exercise requires energy production from the phosphocreatine and glycolytic energy pathways, but negative effects on exercise performance could occur due to an excess accumulation of fatigue agents such as lactate acid and hydrogen ion concentrations. Lactate is mainly produced in skeletal muscle because the acceleration of glycolysis at the onset of muscle activity is faster than the acceleration of the oxidative pathway, and because the maximal glycolytic capacity exceeds the maximal oxidative capacity (Juel, 2001). Therefore, high intensity exercise can result in excess concentrations of lactate due to the primary energy systems being utilized to complete mechanical work.

Higher exercise intensity has been documented to increase both intramuscular and circulating levels of lactate (Rowell et al., 1986; McLoughlin et al., 1991), and these increases in lactate reflect an increased hydrogen ion concentration which is commonly associated with increased muscular fatigue (Karlsson et al., 1975; Stamford et al., 1981) and may disrupt muscle contractility (Sahlin, 1992). Modifiable factors such as training status, exercise intensity, type of exercise, recovery duration, and recovery type have been linked as mitigating factors that can affect the magnitude and/or rate of lactate production during an exercise session. Hence, it appears that the rate of lactate accumulation could be altered by manipulating these modifiable factors to assist in exercise performance training by lowering the degree of lactate accumulation.

Recovery type is an easily modifiable intervention and active recovery interventions (i.e., 30-40% of $\text{VO}_{2\text{max}}$) have been documented to promote an increased blood lactate clearance when performed between bouts of high-intensity exercise (Thiriet et al., 1993; McLoughlin et al., 1991; Monedero & Donne, 2000; Billat, 2001; Billat et al., 2001; McAinch et al., 2004). Furthermore, active recovery has been demonstrated to improve power output (Bogdanis et al., 1996; Connolly et al., 2003; Thiriet et al., 1993) and increase exercise performance during repeated bouts of moderate and high intensity aerobic exercise (Belcastro & Bonen, 1975; Menzies et al., 2010; Hinzpeter et al., 2014). Previous research studies have incorporated recovery periods ranging from 30 seconds to 40 minutes (Bangsbo et al., 1994) and the greatest duration implemented for recovery periods has been in excess of 5 minutes. Bogdanis et al., (1996) showed that a short 4-min period of active recovery, as opposed to passive recovery, enabled an increase in power output when a 30-s sprint was repeated, yet they didn't find a significant difference in blood lactate concentrations between recovery interventions. And, Connolly et al., 2003, found similar results with regards to peak power and lactate values between intervention groups. Based on these results, short duration active recovery bouts between high intensity activity seem to not have significant effect on reducing the magnitude of lactate production during intense metabolic work. Therefore, the purpose of this study was to investigate the effects of short 3-minute recovery durations on blood lactate accumulation when performing repeated bipedal sprinting activities in healthy young adults.

2. Methods

Participants

Thirty six non-athlete subjects (Male: 22, Female: 14) ages 19-28 years old were recruited from the student population for participation in this study (Table 1). No significant differences were observed for baseline measures for the subjects in the intervention groups. For initial inclusion into the study subjects had to be a current university student, classified as a non-university athlete, and free of cardiovascular disease as assessed by the health history questionnaire (HHQ) and PARQ. Institutional Review Board approval was obtained prior to any data collection and informed consent was collected from all participants after a detailed explanation of the aims, benefits, and risks involved with this investigation.

Preliminary screening

All subjects cleared for participation completed an informed consent, PARQ, HHQ, and a blood history questionnaire. The blood history questionnaire assessed the subject's risk for coming in contact with any blood borne pathogens prior to the study and to be cleared for the finger prick blood analysis procedure. Anthropometric data including weight, height, fat mass, and fat free mass was collected along with resting blood pressure and heart rate. Body composition was quantified by bioimpedance analysis using the Inbody 520 (Version 520DM-1520; Biospace, Inc., Los Angeles, CA, USA). Participants remained standing for 15 minutes prior to testing to allow for normal circulation of blood and fluid movement according to the manufacturer's guidelines. The participants were instructed to refrain from consuming a meal two hours prior to arrival, void their bowels, to remain hydrated, and to have abstained from moderate to intense exercise for 12-hours before the screening and testing sessions. Subjects were then randomized in the active recovery (AR) or passive recovery (PR) intervention groups following completing the informed consent.

Testing Protocol

Upon arriving at the laboratory testing facility all subjects were asked if they adhered 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 separated by the recovery intervention of 3-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 decelerating 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 the 3-minute 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 lactate levels.

Blood Sample

Samples were drawn with a standard hygienic finger puncture method and samples were analyzed as whole blood. Blood lactate was measured twice in succession at the same site 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 60 seconds of completing the exercise work bout before starting the recovery intervention.

Recovery Intervention protocol

The AR group completed a 3-minute aerobic walking on a flat surface at 55-65% of their maximal age predicted heart rate which corresponded to a walk pace of 2.5-2.8 mph. Heart rate was monitored using a standard polar Ft1 heart rate monitor. The PR group's passive intervention was comprised of remaining in a seated position for the intervention duration with no bipedal movement.

Statistical Analysis

Group differences with respect to demographic, anthropometric, and baseline variables were assessed via one-way ANOVA. A mixed two-way repeated measures ANOVA was performed to assess the effects of repeated sprint trials and recovery modality with top-down application of univariate ANOVA's and pairwise comparisons as needed. All statistical analyses were performed using SPSS v.24 (IBM Corp., Armonk, New York). Alpha level was set to 0.05 for all tests.

4. Results:

Table 1. Participant baseline characteristics.

	AR	PR	p-value
N	18	18	
Age (yrs.)	22.3 ± 2.4	22.2 ± 2.7	.833
Weight (lbs.)	148.4 ± 37.7	152.1 ± 57.7	.826
Height (inches)	68.1 ± 4.1	70.1 ± 4.1	.164
FFM (lbs.)	124.7 ± 32.3	123.7 ± 45.3	.945
FM (lbs.)	23.4 ± 17.1	28.3 ± 24.4	.491
Resting BL (mmol/L)	2.1 ± 1.6	2.3 ± 1.2	.561

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

Blood lactate concentrations

Normality was met as assessed by the Shapiro-Wilk test and no outliers were observed greater than ± 3 standard deviations. There was sphericity for the two-way interaction effect, ($p > 0.5$). A significant interaction was observed between intervention and time on BL concentrations, $F(2,68) = 4.857$, $p = .011$, partial $\eta^2 = .125$. Pairwise comparisons revealed that after set one mean BL concentration was lower in in the AR group compared to the PR group, -14.61 (95% CI, -2.935 to 0.013) mmol/L, a difference that was not significant, $F(1,34) = 2.206$, $p = .147$, partial $\eta^2 = .061$, (Table 2). After set two, BL concentration were lower in the AR group, -27.39 (95% CI, -4.476 to 1.002) mmol/L, and this was a statistically significant difference, $F(1,34) = 10.267$, $p = .003$, partial $\eta^2 = .232$. Furthermore, BL concentration after set three was lower in the AR group, -27.61 (95% CI, -4.233 to 1.289) mmol/L, compared to the PR group, and this was a statistically significant difference, $F(1,34) = 14.530$, $p = .001$, partial $\eta^2 = .299$, (Figure 1).

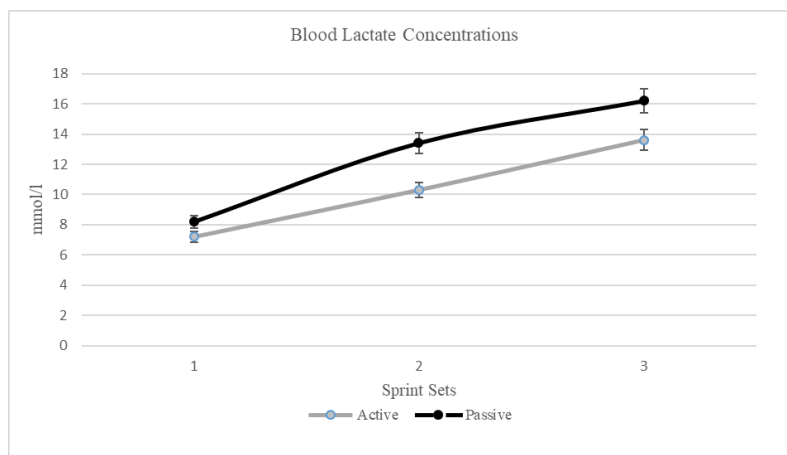
Table 2. Intervention variables after the completion of each exercise trail set

BL (mmol)	Set 1	Set 2	Set 3
AR	7.2 ± 1.9	10.3 ± 5.3	13.6 ± 6.3
PR	8.7 ± 2.4	13.4 ± 2.9	16.3 ± 2.5
Sprint Time (sec)			

AR	29.1 ± 3.8	30.1 ± 3.1	30.6 ± 3.4
PR	28.4 ± 2.8	30.2 ± 2.8	31.4 ± 2.9
Heart Rate (bpm)			
AR	172 ± 19.1	182 ± 11.2	183 ± 9.3
PR	174 ± 19.2	181 ± 11.6	180 ± 9.5
RPE			
AR	12.6 ± 1.7	14.7 ± 1.6	16.1 ± 2.0
PR	13.6 ± 2.4	15.4 ± 2.4	16.8 ± 2.0

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

Figure 1. Blood lactate concentrations following sprinting sets.



Sprint duration time

Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $X^2(2) = 20.259$, $p = .002$. There was no statistically significant two-way interaction between recovery mode and time $F(2,68) = 2.523$, $p = 0.88$, partial $\eta^2 = .069$. The main effect for time was statistically significant across sprint sets $F(2,68) = 26.558$, $P < .000$, partial $\eta^2 = .439$, and there was no significant group effect $F(2,68) = .008$, $p = .931$, partial $\eta^2 = .001$, (Table 1).

Heart rate

The assumption of sphericity was violated for the two-way interaction, $X^2(2) = 36.705, p = .001$. There was no statistically significant two-way interaction between recovery mode and time $F(2,68) = .526, p = .593, \text{partial } \eta^2 = .015$. The main effect for time was statistically significant across sprint sets $F(2,68) = 9.408, P < .001, \text{partial } \eta^2 = .217$, and there was no significant group effect $F(2,68) = .037, p = .848, \text{partial } \eta^2 = .001$.

Ratings of perceived exertion

Sphericity was violated for the two-way interaction effect, $X^2(2) = 11.140, p = .004$. There was no statistically significant two-way interaction between recovery mode and time $F(2,68) = .110, p = .896, \text{partial } \eta^2 = .003$. The main effect for time was statistically significant across sprint sets $F(2,68) = 74.471, P < .001, \text{partial } \eta^2 = .687$, and there was no significant group effect $F(2,68) = 1.860, p = .182, \text{partial } \eta^2 = .052$.

6. Discussion and conclusions

It has been 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 (Thiriet et al., 1993; McLoughlin et al., 1991; Monedero & Donne, 2000; Billat, 2001; Billat et al., 2001; McAinch et al. 2004). This present study indicated that short duration active recovery exercise (3-minutes) produced lower lactate concentrations compared to the passive recovery group between wind sprint sets. The major finding was that blood lactate concentrations following the second and third set of wind sprints was significantly lower in the AR group compared to the PR group for the same metabolic work completed.

This study was aimed at testing short (3 minutes) recovery periods in healthy college students and we found our results to differ from Connolly et al., (2003) and Bangsbo et al., (1994) who observed no statistically significant difference in blood lactate concentration between 3-minute active and passive recovery interventions. This current study differed in the exercise modality and we believe that the wind sprints consisting of multiple acceleration and deceleration stages may have resulted in a larger accumulation of blood lactate compared to the prementioned research projects. We conclude that the larger significant differences between the two intervention groups was observed due to greater nature of the metabolic work needed to complete these quick and power acceleration phases during the wind sprints. Both groups displayed a significant increase in blood lactate concentrations across all three-sprint trails and the present data demonstrated a difference between the active and passive recovery modalities, which could be explained by a difference in skeletal muscle metabolism.

It has been recognized that elevated levels of skeletal muscle and blood lactate are associated with impaired skeletal muscle function and exercise performance (Andrews et al., 1996; Minshull et al., 2007; Hogan et al., 1995). Potential mechanisms for the reduction in accumulated

blood lactate include the redistribution of lactate to the metabolic tissues such as the liver, heart and inactive muscle (Belcastro & Bonen, 1975) and being oxidized by skeletal muscle during recovery (Brooks, 1986, Thiriet et al., 1993). In addition, the clearing of accumulated lactate can vary depending on several factors including training status, exercise intensity, type of exercise, recovery duration, and recovery type. Thus, delivering strategies to assist in clearing accumulated skeletal muscle and blood lactate is very relevant to supporting training adaptations that occur from subsequent overloading of high intensity exercise. Our results suggest that 3-minute active recovery periods may assist with producing 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.

While a significant difference was observed between the AR and PR groups in lactate concentrations, the modality of testing did still result in large accumulations of blood lactate, which still could alter exercise performance and increase skeletal muscle fatigue. Different recovery modalities of longer durations could have a greater effect on the magnitude of lactate appearance, the rate of blood lactate clearance during active recovery periods, and decreased onset of muscular fatigue leading optimized exercise performance. Nevertheless, the implementation of 3-minute short duration recovery periods could be a useful tool when completing high intensity sprinting activities in training populations.

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