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Research Article

THE EFFECTS OF ACTIVE RECOVERY ON LACTATE CLEARANCE DURING HIGH INTENSITY RESISTANCE TRAINING IN COLLEGIATE ATHLETES

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Abstract

With the development of intense strength and conditioning programs, it is necessary to investigate the best means for recovery promotion during workout sessions for optimal performance. The objective of this study is to evaluate blood lactate concentrations and the effects of active and passive recovery during high intensity resistance sytrength training session. Fifteen healthy female collegiate athletes age 18-22 yr. participated in a strength and conditioning workout session. Participants were randomized into an active recovery (AR) or passive recovery intervention (PR). Both groups performed 2 sets of 8 repetitions at 70% 1RM in the following order: Power Clean, Barbell Squat, Barbell Bench Press, Barbell Bent-Over Row, and Kettlebell swings. Each set and exercise were separated by 3-min rest. The active recovery group cycled on an ergometer and the passive recovery group remained seated for the recovery. The blood lactate was collected at baseline, between each exercise and post testing. Blood lactate (BL) was quantified using finger pricks blood analysis with the handheld lactate analyzers. The changes in blood lactate were compared between active and passive groups using a one-way ANOVA statistical analysis. No significant effect was observed for blood lactate levels between the two interventions at rest, between each exercise or post value.

Keywords: Cluster set, Passive recovery, Fatigue

Introduction

Recovery interventions in sports play an essential role when subsequent athletic performance may be required during athletic competition. Many sports illustrate that performance is based on maintaining high-level physical outputs during repeated bouts (McAinch et al., 2004; Siegler et al., 2006) and declines in force production have been linked to numerous metabolic alterations during athletic performance. It has been relativity accepted (Astrand et al., 1996; Bogdanis et al., 1995; Karlsson et al., 1971) that intense physical activity manifests lactate production or H+ ions and this accumulation is commonly associated with increased muscular fatigue (Karlsson et al., 1975; Stamford et al., 1981) and may disrupt muscle contractility (Sahlin, 1992). A common definition of muscle fatigue is any decline in muscle performance that is associated with muscular activity. The magnitude of fatigue and/or lactate production can be related to several modifiable physical activity factors such as exercise intensity, type of exercise, recovery type, recovery duration or training status.

Altering recovery type and duration have shown promising results in the delay of developmental fatigue related to an increased exercise intensity and lactate production. Active and passive recovery types have gained the most attention as modulators in the investigation on athletic performance fatigue. Active recovery involves performing light aerobic activity in between sets of high-intensity exercise providing increased venous blood flow (Crisafulli et al., 2003) while passive recovery consists of a relaxed seated rest period. It has been demsotrated that lactate and H+ ion removal is more rapid during active recovery at 45% of maximaum oxygen uptake than during passive recovery (Belcastrao and Bonen, 1976; Bond et al., 1991; Belcastro & Bonen, 1975; Hermansen and Stensvold, 1972;

Koutedakis and Sharp., 1985). Gisolfi et al. (1966) suggests that the increased clearance rate of La2 during active recovery was probably a result of more rapid distribution of lactate to the liver for oxidation or reconversion to glycogen, increased utilization of lactate by the cardiac muscle, and increased utilization of lactate by active and inactive skeletal muscles. Futhermore, active recovery increases exercise performance during repeated bouts of moderate and high intensity aerobic exercise when compared with passive recovery or rest recovery (Belcastro and Borne, 1975; Hinzpeter et al., 2014; Menzies et al., 2010; Spencer et al., 2006).

Majority of research has focused on high intensity sprinting activity (running, cycling, swimming), which lends to the question of different intervention strategies for resistance training. Resistance training research has focused on the manipulation of sets, repetitions and workload on resistance strength training performance. Minimal research has invesigated different activity interventions during recovery periods of resistance training (Corder et al., 2000; Mohamad et al., 2012) and reviews suggest that light aerobic activity during interest rest periods of resistance training may increase strength and power, muscle hypertrpohy, and mechanical performance (Mohamad et al., 2012).

The purpose of this research project was to examine two different recovery interventions on blood lacate concentrations during a high intensity resistance training cluster circuit. We hypothesized that active recovery work rest during high intensity resistance training will elicit lower blood lactate levels compared to the passive recovery rest intervention in female athletes.

Methods

Participants

Subjects were enrolled from university's varsity athletic program with permission from the strength and conditioning coach. For intial inclusion into the study subjects had to be a current university student, a collegiate athlete for the university and be out of season for their sport. Subjects were recruited during the summer so athletes varied during this training time. Sports that were recruited were football, volleyball and softball. Institutional Review Board approval was abotained prior to any data collection.

Protocol

Preliminary screening

All subjects completed an informed consent, PARQ, and blood health questionnaire upon arrival at the exercise physiology lab. The BHQ form assesed the subjects 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 during the weight training portion of data collection. Anthropometric data including height, weight, fat mass and fat free mass was collected in addition to resting blood pressure values utilizing a standard blood pressure cuff. Body composition was quantified using the BodPod system (COSMED, Concord, CA). The participants were instructed to not consume a meal two hours prior to arrival, void their bowels, to remain hydrated and to have refrained from an moderate to intense exercise for 12-hours. Subjects were then randomized in the active recovery (AR) or passive recovery (PR) intervention groups following completing the informed consent.

Strength training session

Prior to the training session the subjects completed a brief 5-10 minute dynamic warm-up
conducted by the strength coaches who also oversaw the resistance training sessions for athlete safety. The resistance training protocol consisted of 5 exercises using power cluster sets (Roll and Omer, 1987). The power clusters consist of performing 70% of an athletes 1RM on each specified exercise. The 70% of 1RM on exercises was determined by the records of the strength and conditioning coaches of the university. The specific exercises specified by the coaches were: Power Cleans, Barbell Squats, Barbell Bench Press, Barbell Bent-Over Row, and Kettle Bell Swings. The power cluster set consisted of the subjects performed 2 repetitions followed by a 15 second rest and repeating this four times for a total of 8 repetitions for each set. Subjects were not allowed to rest when performing the repetitions and had to complete 2 successful repeptiotions as quickly as possible. Subjects completed two power cluster sets for each of the five compound exercises (Figure 1). A total of 10 sets were completed during the five exercises (two cluster sets for each exercise). There was a active or passive recovery between each power cluster and between each exercise. During the first recovery period subjects completed the intervention and then performed the second power cluster set. During the second recovery period before switching exercises data was collected by the primary investigator. The completion of the ten sets lasted about sixty minutes with the rest periods.

Figure 1: Resistance training prescription

Power	Reps	Rest	Reps	Rest	Reps	Rest	Reps	Rest
Cluster 1	2	15 sec	2	15 sec	2	15 sec	2	15 sec

3 minute active or passive rest

Power	Reps	Rest	Reps	Rest	Reps	Rest	Reps	Rest
Cluster 2	2	15 sec	2	15 sec	2	15 sec	2	15 sec

3 minute active or passive rest – data variables were collected

Recovery protocol

The AR group completed a 3-minute aerobic cycle protocol on a Monark-Model 864 Cycle Ergometer at 55-65% of their maximal age predicted heart rate. Revolutions per minute was set at 60. Heart was collected using a standard polar Ft1 heart rate monitor (Polar USA, Bethpage, NY). The PR group's passive intervention was comprised of seated on a bench or a cycle ergometer for 3-minutes remaining still. Data was recorded during the second 3-minute rest phase before switching to the next exercise.

Data collection

Blood lactate was collected at baseline, between each exercise, and post exercise intervention. Lactate values were quantified using Nova plus blood lactate analyzer (Nova Biomedical, Waltham, MA). Subjects performing the active recovery had their blood lactate measured while pedaling on the cycle ergometer (60 RPM) and the passive group while they were seated. Blood lactate was measured twice in succession at the same site using the NOVA lactate analyzer. Subjects' fingers were cleaned with an alcohol wipe and then pricked using a Unistik 3 lancet (Owen Mumford, Oxfordshire, UK) and the initial drop of blood was wiped away with a gauge pad. A second drop of blood was then sampled and wiped away for a third sample. Heart rate and RPE were collected directly after completion of the second power cluster set.

Statistical Analysis

Demographic data, independent variable, and dependent variables were recorded in Excel (Microsoft Corporation, Redmond, WA) and then analyzed using SPSS Statistics 21 (SPSS, Inc., Chicago, IL). One-way ANOVA was utilized to examine baseline demographic differences between intervention groups. An alpha of $p<.05$ was considered statistically significant for all comparisons. Differences in the dependent variables (blood lactate clearance, rate of perceived exertion, and heart rate) that were elicited by the intervention were analyzed using mixed factorial ANOVA. This analysis included three separate 5x2 mixed factorial ANOVAs to analyze differences from each variable over time between the two groups (active recovery & passive recovery).

Results

Twenty-three total participants were screened during the preliminary research of the study. Only twenty of the participants were eligible to participate within the experimental procedure (15 females and 5 males). Two subjects dropped out for personal reason and one was dropped due to noncompliance. For this publication only the female athletes were included in the data analysis due to the low recruitment rate for the male athletes. Significant differences were observed for age and years of athletic experience between the AR and PR groups for the females. (Table 1).

Table 1. Participant baseline characteristics.

	Active - AR	Passive - PR	p-value
N	8	7	
Age	19.6 ± .74	20.7 ± .95	.027*
Experience (yrs.)	1.86 ± .64	2.57 ± .53	.041*
Weight	166.1 ± 20.9	150.9 ± 20.1	.176
Height	66.8 ± 3.1	66.7 ± 2.2	.926
FFM	121.3 ± 6.2	115.1 ± 15.2	.300
FM	44.8 ± 19.2	35.9 ± 8.1	.276
Resting BL	1.81 ± .72	1.71 ± .44	.096
Post BL	1.86 ± .67	2.11 ± .74	.478

Values are mean±SD. n, number of subjects. FFM, fat free mass. FM, fat mass, BL, blood lactate.
* Sig. difference p <.005.

Dependent variables

Blood lactate

Baseline and post-test blood lactate levels were not different between the active and passive recovery groups (Table 2). Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $X^2(9) = 26.034$, p =.002. There was no significant interaction between intervention and time on blood lactate concentrations, $F(4,52) = .515$, p =.622, partial $\eta^2 = .038$. The main effect of time showed a significant difference in mean blood lactate concentration levels at the different time points, $F(4,52) = 4.802$, p =.001, partial η^2 =.270. Main effect for group showed that there was no significant difference in mean blood lactate concentrations between intervention groups, $F(1,13) = 1.612$, p =.226, partial η^2 =.110. (Table 2)

Heart rate

No significant interaction between intervention and time for heart rate was observed, $F(4,52) = .472$, p =.633, partial $\eta^2 = .035$. The main effect of time showed a significant difference in mean heart rate values at the different time points, $F(4,52) = 6.695$, p <.001, partial $\eta^2 = .340$ and no significant difference was observed for the main effect of group, $F(1,13) = 1.488$, p =.224, partial $\eta^2 = .103$.

Rating of perceived exertion

There was no significant interaction between intervention and time on rate of perceived exertion, $F(4,52) = .223$, p =.830, partial $\eta^2 = .017$. No main effect for time was observed at the different time points, $F(4,52) = 3.503$, p =.162, partial $\eta^2 = .116$ and no main effect for group was observed between the invention groups, $F(1,13) = 19.362$, p =.216, partial $\eta^2 = .115$.

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

	AR	**PR**	**AR**	**PR**	**AR**	**PR**
	Blood lactate		Heart rate		RPE	
Cleans	3.3 ± 1.3	4.6 ± 3.1	124.1 ± 19.4	120.3 ± 25.6	9.4 ± 2.6	8.9 ± 2.3
Squats	3.4 ± 1.0	4.7 ± 3.9	135.4 ± 17.1	125.2 ± 21.1	10.4 ± 1.9	9.5 ± 2.8
Bench press	3.6 ± 1.1	3.9 ± .71	119.6 ± 17.4	105.7 ± 19.1	9.9 ± 1.3	8.7 ± 1.3
Barbell row	2.5 ± 1.3	3.0 ± 2.1	122.5 ± 12.2	110.7 ± 17.9	9.3 ± 1.6	7.9 ± 1.3
Kettlebell	1.9 ± .49	1.9 ± .28	131.6 ± 12.1	121.4 ± 17.1	9.9 ± 2.2	8.7 ± 1.7

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

Disscussion/Conclusion

The aim of the present study was to compare the effectiveness active and passive recovery methods during resistance training on blood lactate accumulation, heart rate response, and rating of perceived exertion. Corder et al. (2000), demonstrated lactate accumulation was decreased following resistance training workouts when low-intensity exercise was employed, most notable when subjects performed active recovery exercise. One of the mechanisms believed to enhance lactate clearance is the oxidation of lactate by the skeletal muscles (Gisolfi et al., 1966;

Weltman et al., 1977). The current study didn't find a significant difference between the active recovery and passive groups for blood lactate, heart rate or rating of perceived exertion. Blood lactate concentrations trended to be lower in the active recovery group compared to the passive recovery group, however, none of the exercises displayed a significant differences for lactate levels between groups. Conversely, the active recovery group trended to have higher heart rate values for all exercises, nevertheless, all the completed exercises didn't yield significant differences between intervention groups. Several mechanistic theories have been established and will be discussed.

Blood lactate concentration during exercise is often used as a marker of exercise intensity and training status (Schwaberger et al., 1985). It has been demonstrated that previous training reduces blood lactate concentrations during submaximal exercise by improving lactate clearance (MacRae et al., 1992), and can result in an increased mitochondria volume (Howald, 1982), and muscle capillarization (Ingjer, 1979). These adaptations are associated with increased resistance to fatigue and improved exercise capacity performance. Limited research has focused on athletes and intervention recoveries (Jemni et al., 2003; Lau et al., 2001; Suzuki et al., 2004; Watts et al., 2000) and a possibility is due to the trained status of the athletes and the ability to invoke greater chances in an untrained population. The average training years' experience for the athletes was 2.22 years at the collegiate level and past experience was not taken into consideration. One mechanism to explain the non-significant findings could be enhanced lactate clearance capacity of the athletes due to enhanced cellular mechanisms from previous training.

The resistance training intensity selected was based on the subjects 1 repetition maximal (1-RM) from previous strength workouts and their working load was set as 70% of their recorded 1-RM. Cluster sets characteristically use a minimum load of an individual's 5RM, performing 4-6 repetitions, and it's estimated that an individual's 5RM is about ~86% of their 1RM for most large mass exercises (Verkhoshansky et al., 2009). The workload of 70% was established by the coaches assisting with the project we believe that due to the enhanced training status of the athletes the percentage chosen should have been set higher around 80-85% of their one repetition maximal. We believe this factor had the greatest effect on not eliciting a higher lactate response and having the athletes produce a blood lactate level above the normal onset of blood lactate of 4 mmol/L (Sjödin and Jacob, 1981).

The ability to manipulate recovery duration variables during training has been shown to have proficient effects on reducing lactate accumulation and increasing power development. These can include recovery modality, duration, and intensity (Bielik, 2010; Lomax, 2012; Menzies et al., 2010; Miladi et al., 2001). This project examined a three minute rest recovery intervention on blood lactate concentrations and it has been suggested that recover sessions of longer durations (>5min) could have a greater effect on lactate clearance following high intensity exercise.

Conclusion/Future implications

No significant differences were observed for blood lactate concentration levels and active intervention modes. While no differences were observed after training we believe factors such as training experience, recovery duration, and exercise prescription may have played a mechanistic role in the results of this study. A first implication for future research would be to increase the resistance training intensity to 80-85% of the athlete's 1-RM for each selected exercise. With the athletes having an increased lactate clearance capacity, the increased training intensity could push the subject beyond their lactate buffering capacity. Another implication would be to recruit untrained subjects who could have a lower lactate threshold capacity compared to trained athletes. This may allow for the lower exercise intensity to elicit an increased blood lactate concentration between the intervention groups.

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