



January 2017

The Effects Of Active Vs Passive Recovery On Subsequent Bouts Of High Intensity Performance In Recreational Adult Runners

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THE EFFECTS OF ACTIVE VS PASSIVE RECOVERY ON SUBSEQUENT BOUTS OF
HIGH INTENSITY PERFORMANCE IN RECREATIONAL ADULT RUNNERS

by

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Bachelor of Science, University of North Dakota, 2013

A Thesis

Submitted to the Graduate Faculty

of the

University of North Dakota

In partial fulfillment of the requirements

for the degree of

Master of Science in Kinesiology

Grand Forks, North Dakota

May

2017

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This thesis, submitted by Matthew Aldrin McCreary in partial fulfillment of the requirements for the Degree of Master of Science from the University of North Dakota, has been read by the Faculty Advisory Committee under whom the work has been done and is hereby approved.

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April 1, 2017

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ACKNOWLEDGMENTS

I would like to express my deep appreciation to the University of North Dakota's Kinesiology Department for the education and skills they provided me during my time as a student. Thank you to my committee advisor, Dr. John Fitzgerald, for your support and feedback during this whole endeavor. Thank you to my other committee members, Dr. James Whitehead and Dr. Jesse Rhoades, for your supporting efforts and wisdom. I would also like to thank my friends and participants that contributed to my completion of this thesis. I would not have been able to proceed with this project without you, and I'm truly grateful that you chose to give your time and your effort for me. Thank you to my family for your love and support. I'm forever thankful for the opportunities that you have provided so I can grow to be the best person I can.

ABSTRACT

Background: Runners can perform training runs designed to elicit desired adaptations for future competition. When performed at a high-intensity, these running bouts will lead to fatigue that needs to be diminished to sustain the desired workload for the training session. Performing an active recovery or remaining passive are two methods that runners could use.

Purpose: The purpose of this study was to investigate the effects of active vs passive recovery on a subsequent running bout of 400 meters in recreational adult runners. It was hypothesized that the active recovery condition would maintain performance better than passive recovery.

Methods: A crossover design experiment was used. 20 recreational adult runners (10 males, age: 22.50 ± 2.72 ; 10 women, age: 22.20 ± 1.75) participated in three sessions. The first session was familiarization and the next two sessions were experimental. The experimental sessions were separated by at least 72 hours. A recovery condition of active or passive was randomly assigned for the first session and the opposite would be done for the second. Participants performed two max-effort runs over a distance of 400m separated by 15 minutes of recovery. Blood-lactate levels were measured at 5 and 12 minutes of the recovery period. The absolute difference for performance time and blood-lactate was calculated for each participant in each condition. A change score was calculated as the percentage change between run 1 and run 2 and between blood-lactate in both recovery conditions for each participant. A dependent sample t-test was used to analyze the data to detect any statistically significant differences.

Results: There was a statistically significant difference between mean pre- and post- recovery times (in seconds) in the active (pre: $M = 76.31$, $SD = 13.42$; post: $M = 79.57$, $SD = 14.62$, $p = .01$) and passive conditions (pre: $M = 76.23$, $SD = 14.20$; post: $M = 78.74$, $SD = 13.23$, $p = .001$). There was no statistical difference in the absolute time difference between conditions ($M = -.75$, $SD = 6.61$, $p = .616$). There was also no statistical difference when the change scores between active and passive were compared ($M = .66$, $SD = 7.26$, $p = .688$). The active recovery condition produced a statistically significant difference between blood-lactate measurements taken at 5 minutes ($M = 12.65$, $SD = 2.72$) and 12 minutes ($M = 10.07$, $SD = 3.41$, $p = .012$) of the recovery time. Mean blood-lactate measurements for the passive recovery condition were not statistically different between 5 minutes ($M = 12.76$, $SD = 3.15$) and 12 minutes ($M = 12.04$, $SD = 4.00$, $p = .251$). Absolute blood-lactate difference between conditions didn't produce a statistically significant difference ($M = 2.00$, $SD = 5.18$, $p = .130$). Change score difference between the active and passive conditions approached but did not reach statistical significance ($M = -10.75$, $SD = 23.01$, $p = .081$).

Conclusion: Performing high-intensity 400m runs results in fatigue that could be alleviated with adequate recovery. Although active recovery trended towards lowering blood-lactate values at a faster rate, this did not lead to an improvement in the second 400m run. Passive recovery overall provided a smaller performance decrement than active although this was not statistically different. Runners and coaches should attempt to determine which recovery method may work better for themselves or their athletes by utilizing both in a training session.

CHAPTER I

INTRODUCTION

The use of an active recovery method after high-intensity bouts of exercise has been substantially researched and published within the literature (Devlin et al., 2014; Connolly, Brennan, & Lauzon, 2003; Menzies et al., 2010). Much of the research focuses on how performing an active recovery affects blood-lactate levels and how this could possibly be beneficial in racing sports such as track, swimming, and cycling since more than one event may be done in competition (Dodd et al., 1984). It should be noted however, that much of the findings on the effects of active recovery on subsequent performance remains equivocal. There are several studies that have shown that performing active recovery accelerated lactate clearance, which may have led to improvements in the remaining bouts of exercise within the training session (Greenwood et al., 2008; Spierer et al., 2004). Other studies however, concluded that active recovery did not lead to improved performance, and may not be a superior recovery choice within a training session (Abderrahman et al., 2012; Dupont et al., 2003). Therefore, it is important to understand the parameters that these studies used, such as exercise modality, intensity and duration of the exercise, and intensity and duration of the active recovery performed. These variations likely contribute to the lack of agreement among researchers whether or not active recovery is superior to passive recovery.

Muscular fatigue is the decreased ability to generate appropriate amounts of muscle force or power during on-going contractile activity (Finsterer, 2012). The sensations of fatigue and

exhaustion are natural after both prolonged, low-intensity and acute, high intensity exercise. These sensations are thought to be a safety mechanism essential to maintaining the physical integrity of the body (Finsterer, 2012; Ament & Verkerke, 2009). The accumulation of lactate in the blood after an exercise bout of high intensity and its' relationship to muscle fatigue remains controversial (Devlin et al., 2014). Current research suggests that a high level of blood-lactate is correlated with muscular fatigue, but may not share a cause-and-effect relationship (Ament & Verkerke, 2009). Rather, it is thought that the reliance on non-mitochondrial ATP turnover and the resulting accumulation of metabolites during high-intensity exercise is the primary cause of fatigue. High levels of metabolites such as inorganic phosphate (Pi), adenosine diphosphate (ADP), and hydrogen ions (H⁺) are thought to reduce the efficiency and activation of the cross-bridge cycles within muscle leading to reduced force generation (Fitts, 2008; Ament & Verkerke, 2009; Debold, 2012; Allen & Trajanovska, 2012). Active recovery is thought to help buffer the H⁺ ions and remove other metabolites faster by increasing blood flow throughout the periphery, increasing venous return to the heart, and promoting the uptake of lactate into the working muscle itself or muscles that did not contribute primarily to the activity (Yoshida, Watari, & Tagawa, 1996; Robergs, Ghiasvand, & Parker, 2004).

When compared with passive recovery, active recovery has been shown to facilitate an acceleration of lactate clearance. The question lies, however, with its' effects on the repeated performances of the individual during their training session. Although lactate levels have been shown to decrease with active recovery, studies have shown that glycogen levels within muscle fibers tend to also be lower with the use of active recovery (Choi et al., 1994; Fairchild et al., 2003). This could potentially be counterproductive as muscle glycogen re-synthesis is necessary to fuel the subsequent exercises. Another problem that is seen within the literature is the different

methodologies implemented for the active recovery protocol (McAinch et al., 2004). The type of modality used for the recovery usually mimics the exercise modality for sport-specific purposes, and has been shown to lower blood lactate levels compared to passive rest (Tokmakidis, Toubekis, & Smilios, 2011). However, performing an active recovery that uses the same muscle-mass can still lower blood-lactate levels quicker than a passive recovery, even if the modality is different (Felix et al., 1997). Some studies use lower percentages of VO_{2max} (Spierer et al., 2004; Fairchild et al., 2003) while others use percentages of lactate threshold (Greenwood et al., 2008; Del Coso et al., 2010) while also using various modalities. Although these results are useful for data purposes, it may be of little use to recreational athletes or even coaches who don't have access to their own VO_2 or threshold data. The expression of active recovery as a percentage of speed attained in a racing distance may be more helpful (Tokmakidis, Toubekis, & Smilios, 2011).

Purpose of Study

In spite of an abundance of literature looking at active vs. passive recovery, we are unaware of any studies investigating the effect of active recovery on repeated middle distance running performance. Therefore, the purpose of this study is to determine if performing an active recovery between two bouts of high-intensity 400m runs will elicit a better maintenance of performance when compared to passive recovery in recreational runners.

Hypothesis

In this study, it is hypothesized that performing an active recovery will help maintain performance on a subsequent 400m bout when compared to passive recovery. It is also hypothesized that blood-lactate values will be lower following the active recovery.

Significance

Running distance-specific repetitions in training sessions can elicit near VO_2 max levels and surpass the lactate threshold. These high-intensity efforts can lead to fatigue that the runners need to recover from to continue their training session. The question that arises within the training session is how to best alleviate this fatigue to continue performance at a high-intensity. The better quality the training is, the better the potential stimulus to achieve the desired adaptations. This recovery within the training session is a critical component, not only so high effort workloads can be achieved, but also to prevent potential injuries so they are able to participate in competition.

CHAPTER II

REVIEW OF THE LITERATURE

There is much debate within the literature on the use of active recovery as a mechanism to reduce fatigue and improve or sustain performance. Although there is an abundance of research that looks at active recovery as a method to reduce lactate levels after high-intensity exercise, there is less research that focuses on this reduction of lactate in the blood and its' effect on high-intensity, repeated performance bouts. The findings on the efficacy of active recovery are equivocal at best. This review has been divided into three sections. The first section focuses on the physiology background concerning lactate, hydrogen ions (H^+), and their effects on the body during and after intense exercise. The second section discusses research studies that have demonstrated active recovery to have a positive influence on subsequent performance within an exercise session. The third section will focus on studies that show the opposite of the second section, where active recovery may not improve subsequent performance.

Physiology Background

The transition from rest to exercise causes many physiological effects within the body that can be seen as a deviation from homeostasis. These effects combine to prepare the body and result from the stress that exercise induces on the body. Some of these changes are increasing blood flow to active muscles, increasing ventilation, and secretion of specific hormones. Among the many phenomena that accompany physiological changes during exercise is the increased production of lactate. High levels of lactate is produced within skeletal muscle because of the

accelerated use of the glycolytic energy pathway compared to the oxidative energy pathway during high intense exercise and because the glycolytic capacity is higher than that of the oxidative capacity (Juel, 2001). ATP demand is met immediately at the onset of exercise via the phosphagen system, which breaks down creatine-phosphate to form ATP. As exercise progresses and/or intensity increases, other energy pathways must be utilized to sustain the formation of ATP and thus prolonging exercise. The glycolytic pathway uses glucose (glycolysis) or glycogen (glycogenolysis), to form this ATP. This energy system is the main focus surrounding lactate, metabolites, and metabolic acidosis that contributes to fatigue.

It was long believed that the production of lactate was the direct cause of the onset of metabolic acidosis and thus the cause of fatigue during intense exercise (Cairns, 2006). However, a review done by Robergs, Ghiasvand, and Parker (2004) disputes this claim. It is stated that there has been no evidence to support a cause-and-effect relationship pertaining to the production of lactate and the onset of acidosis, but instead only demonstrates a correlation between the two. The underlying mechanism for metabolic acidosis is not from the production of lactate, but from non-mitochondrial ATP turnover at a high rate (Robergs, Ghiasvand, & Parker, 2004; Siegler et al., 2006; Moxnes & Sandbakk, 2012). As exercise intensity increases, the glycolytic system takes on more of the load in generating ATP to sustain this intensity. As glycolysis progresses, NAD^+ is rapidly reduced to NADH. Consequently, the rate of glycolysis will slow if NAD^+ is not regenerated fast enough, indicating that the aerobic conversion of NAD^+ to NADH in the mitochondria is unable to keep up with the high demands of the exercise intensity. The conversion of pyruvate to lactate occurs to regenerate NAD^+ at a faster rate, therefore keeping glycolysis running faster and longer (Robergs, 2011). ATP turnover is still high at this time, which leads to metabolite production and accumulation such as H^+ , as previously described.

Although this is likely the true mechanism behind acidosis and the subsequent decline in muscle performance, lactate still plays a role in the recovery process from high intensity exercise.

Lactate accumulation only occurs insofar as production exceeds removal. It has been proposed by Brooks (2004) and his colleagues that an intracellular lactate shuttle helps to move lactate from the cytosol to areas such as the mitochondria for oxidation. When exercise intensity is high, glycolytic flux is also high and relies more on non-mitochondrial ATP. When this occurs, oxidative pathways are unable to keep up with the demand for ATP, and lactate begins to accumulate in the cytosol. Although lactate acts in assisting proton efflux from the muscle, this particular transport is rate-limiting. Eventually lactate production will decrease, slowing down its clearance rate. Lactate Dehydrogenase (LDH), which is the terminal enzyme of glycolysis, is also affected by the accumulation of lactate. The lactate shuttle moves the lactate to the mitochondria of skeletal muscles, liver, and other cells for it to be used as a substrate for oxidation, thus providing a link between glycolytic and aerobic metabolism (Brooks, Fahey, & Baldwin, 2004; De Pauw et al., 2011). The shuttled lactate is converted back to pyruvate in the mitochondria, and this pyruvate is then broken down into Acetyl CoA, which then enters the Krebs cycle and produces ATP via the oxidative pathway. This ability to clear high levels of lactate after intense exercise may help delay the onset of acidosis, therefore enabling the individual to delay fatigue and continue to perform at a high level.

Table 1 summarizes studies focusing on the effects of active recovery compared to passive recovery. Parameters of the studies are varied, but many of them prescribe active recovery as a percentage of the participant's VO_{2max} or their individual lactate threshold.

Table 1. Summary of studies comparing Active Recovery (AR) to Passive Recovery (PR)

Study	Participants	Exercise Protocol	Active Recovery	Results
Abderrahman et al., 2012	24 adult males	3 groups: Control, 30s run/30s passive, 30s run/30s active	50% of Maximal Aerobic Velocity	VO _{2max} increased with AR, Time to exhaustion was longer with PR.
Koizumi et al., 2011.	10 active males (9 baseball, 1 Track), Average age: 20.4 years	Two max cycles for 30s with 20 min rest of either PR or AR between cycles	30% of $\dot{V}O_2@VT$	Muscle O ₂ and blood lactate was lower in AR. Work and Peak Power were higher in the second bout after AR.
Menzies et al., 2010	10 moderately trained adult males	5 min. high intensity run at 90% VO ₂ max	100,80,60, or 40% of LT, and a self-selected intensity, until a return to baseline	AR at 80-100% of LT provided the fastest Lactate clearance.
Spierer et al., 2004	6 sedentary adults (3 M, 3 F), 9 Male, moderately trained ice hockey players	Repeated Wingates separated by 4 min. of AR or PR	Work rate corresponding to 28% of VO _{2max}	Total work was higher with AR, Lactate was lower with AR for Hockey players but not for Sedentary.
McAinch et al., 2004	7 adult males	Two 20 min. bouts of cycling with 15 min. recovery in between	40% of VO _{2max}	Work done in bout 2 was less than bout 1 for both AR and PR.
Dupont et al., 2003	12 active adult males	15s runs at 120% of Maximal Aerobic Speed (MAS) with 15s rest until exhaustion	50% of MAS	Time to exhaustion was longer using PR.
Dupont et al., 2004	12 males	15s cycling at 60 RPM with 15s rest until exhaustion.	40% of VO _{2max}	Time to exhaustion was longer using PR, decline of oxyhemoglobin was slower with PR.

Table 1. cont.

Study	Participants	Exercise Protocol	Active Recovery	Results
Greenwood et al., 2008	14 male collegiate swimmers	Two 200 yd. swims with 10 min. rest in between	Speed corresponding to their LT, 50% of LT, or 150% of LT	AR at LT improved the subsequent swim bout, AR at 150% of LT maintained the time achieved in bout 1.
Fairchild et al., 2003	8 endurance-trained male college students	2.5 min. cycling at 130% VO_{2max} followed by 30s sprint, then rest for 45 min.	40% of VO_{2max}	AR lowered lactate levels and raised pH faster than PR, glycogen resynthesis was reduced with AR.
Del Coso et al., 2010	11 moderately trained college-aged males	Four cycling bouts for 1.5 min. at 163% of their RCT (Respiratory Compensation Threshold)	4.5 min. at 24% RCT, 6 min. at 18% RCT, and 9 min. at 12% RCT on 3 separate days	AR at 12% RCT facilitated the best lactate removal and return to homeostasis.
Felix et al., 1997	10 Female collegiate swimmers	Two 200 yd. swims with 14 min recovery in between	65% of their best 200 yd. freestyle time	AR maintained performance in the second swim better than PR.
Toubekis et al., 2005	8 males and 8 females	8x25m swim sprints with either 45 or 120s rest, followed by a 50m sprint 6 min. later	60% of their individual best 100m velocity	Performance decreased after the 2 nd sprint with AR compared to PR, 50m sprint was better with the 120s rest for both AR and PR.
Siegler et al., 2006	10 males	Two trials with three intense cycling bouts to exhaustion, each bout separated by 12 min.	60 RPM at 20% of their MWO (Max Work Output)	Times to exhaustion did not differ between recovery conditions.
Spencer et al., 2006	9 males	Four cycle-sprint tests consisting of 6x4s sprints every 25s	32% of VO_{2max}	Lower peak power for the last sprint and a greater power decrement in AR compared to PR.
Brown & Glaister, 2014	10 males	4 trials using a 30s cycle sprint with rest of 45 or 180s then 7x5s sprints	70% of power output at LT	Mean peak power output was higher in PR ₄₅ than AR ₄₅ and in AR ₁₈₀ than PR ₁₈₀ .

Active recovery as a beneficial method

There is a general consensus within the literature that active recovery accelerates clearance of blood-lactate when compared to passive recovery (Koizumi et al., 2011; Del Coso et al., 2009; Spencer et al., 2006). As described previously, the production and clearance of lactate helps in delaying the onset of acidosis, which is detrimental to exercise performance. Although active recovery facilitates the clearance of lactate, it is important to understand how this recovery method could improve or maintain performance in repeated bouts of exercise. Several studies have shown that an improvement in subsequent performance was achieved by performing a type of active recovery protocol.

Greenwood and colleagues (2008) looked at active recovery intensity, blood lactate disappearance, and subsequent performance within male collegiate swimmers. The initial lactate profiling session used seven graded incremental 200 meter freestyle swims, where the first swim was targeted as 30 seconds slower than the individual swimmer's best 200 meter time. Each additional swim had a target time of 5 seconds faster than the previous. The lactate threshold (V_{LT}) was found to be the highest speed attained before the curvilinear increase in blood lactate. Two other speeds were used as a means for an active recovery. $V_{LT.5}$ represents speed at 50% of the lactate threshold and was determined as 50% of the difference between the baseline speed and V_{LT} ; $V_{LT1.5}$ represents 150% of the lactate threshold and was determined to be 50% of the difference between their maximum speed reached and V_{LT} . The experiment consisted of four conditions that were separated by approximately one week. Within each condition the subject would complete a 200 yard maximal swim in their primary stroke, then complete 10 minutes of recovery consisting of swimming at V_{LT} , $V_{LT.5}$, $V_{LT1.5}$, or a passive recovery where they sat on the pool deck. All of the recovery swims were done using the freestyle stroke. The results from

the study showed that the recovery swim at V_{LT} had the greatest lactate clearing effect and improved the subsequent swimming performance in all 14 swimmers. Before the subsequent swim after the recovery, mean lactate levels were 7.1 mmol for passive, 4.0 mmol for $V_{LT,5}$, 3.1 mmol for V_{LT} , and 3.8 mmol for $V_{LT,1.5}$. In addition to lactate levels, performance times in the subsequent swim had a mean decrease of 1.67 seconds when recovery at V_{LT} was performed, compared with a decrease of only .07 seconds with $V_{LT,1.5}$ and increases in time of 1.32 seconds and 1.01 seconds for passive and $V_{LT,5}$, respectively. This study provided some important insights as to what intensity the active recovery protocol should be performed at and perhaps the time frame where active recovery could be beneficial in improving subsequent performance. Another swimming study conducted by Felix (1997) also resulted in active recovery helping subsequent performance compared to passive recovery.

Menzies and colleagues (2010) reiterates what was found by the previously described study; that the clearance of blood lactate is perhaps intensity dependent and that performing active recovery near the lactate threshold had the greatest effect on clearance rate. The lactate threshold was determined by incremental ramp test protocol, where the speed was increased by 0.5 km per hour with a 0% grade every 4 minutes. Maximal oxygen consumption (VO_{2max}) was also assessed using progressive treadmill protocol. The experimental trials consisted of a 10 minute warm-up with a 5 minute run at 90% of VO_{2max} . After the 5 minute run the participants completed a recovery protocol at 100%, 80%, 60%, or 40% of LT. In addition to LT percentages, there was also a passive recovery and an active recovery protocol where the participants could self-select the intensity. The results indicated that the fastest clearance rates were seen when active recovery was performed at 80-100% of LT. There was also no difference in clearance between passive recovery and recovery performed at 40% of LT. The self-regulated intensity that

offered the best clearance rate was also in the 80-100% of LT range. Although the effects of performance after using these protocols were not done in this study, it does provide more insight into what intensities should active recovery be performed at to elicit the greatest removal of accumulated lactate.

The use of an active recovery method to accelerate the removal of lactate compared to passive rest appears concrete. As mentioned earlier, this is of primary importance because lactate is transported with metabolites such as H^+ out of the cells, possibly reducing the effects of fatigue. Active recovery maintains a higher rate of blood flow to and from the exercising muscles, aiding in transporting the lactate and H^+ to the mitochondria, which can then lead to more aerobic metabolism. The efflux of lactate and H^+ out of the cytosol and into the blood has been shown to be connected by Monocarboxylate Transporters (MCT) within muscle fibers, where oxidative fibers are shown to possess more MCT1 (Thomas et al., 2005; Hashimoto & Brooks, 2006). The MCT1 isoform has a high affinity for lactate, allowing for the rapid exchange of lactate between tissue compartments and its subsequent utilization in metabolic processes (Thomas et al., 2012). The two studies that were just discussed perhaps provide some parameters that could better understand the best way to utilize active recovery. Determining lactate thresholds (LT) of athletes and programming active recovery protocols could have a better effect than programming using % of VO_{2max} (Menzies et al., 2010). Although, a study done by Spierer and colleagues (2003) showed improvement in total work performed in subsequent Wingate tests using active recovery corresponding to 28% of VO_{2max} in both sedentary participants and in moderately trained hockey players.

Active recovery as a non-factor

Although active recovery has been shown to facilitate better lactate removal than passive recovery, it still remains equivocal in the literature whether or not this is beneficial at improving performance within a training session. This has importance in repeated, higher intensity tasks that are usually done for sports where racing is the primary objective (swimming, cycling, running).

An experimental study done by McAinch and colleagues (2004) demonstrated that subsequent cycling performance was not enhanced with active recovery, despite a reduction in lactate levels. VO_{2peak} was determined prior to the experimental trials by using an incremental exercise test on a cycle ergometer. This was done by beginning at a work rate of 50 watts and increasing by 50 W every 3 minutes until 12 minutes, where the work was increased by 25 W every minute until volitional fatigue. A work rate required to elicit 40% of VO_{2peak} was determined from these tests. The seven male subjects performed two experimental trials. Each trial had the participant perform as much work as possible in a 20 minute cycling bout followed by a 15 minute rest consisting of either passive recovery or an active recovery performed at 40% VO_{2peak} . Results from this study indicate that total work done in the second bout of cycling was lower regardless of recovery protocol. Active recovery did result in lower lactate concentrations before the second bout, however it was also shown that glycogen levels were also lower in the active recovery trial when compared to passive recovery. These lower glycogen levels could help in explaining the decrease in work output, although work output in the second bout was also lower with passive recovery. This study makes the case for active recovery to be a non-factor in improving subsequent performance and thus an unnecessary aspect within a training session.

Dupont, Blondel, & Berthoin (2003) looked at the effects of active vs. passive recovery in shorter, intermittent runs separated with short rest periods of 30 seconds and total time to exhaustion (TTE). VO_{2max} and maximal aerobic speed (MAS) were determined via a graded test that was done on a 200 meter indoor track. The first two intermittent tests were done by repeating runs at 120% MAS for 15 seconds separated by either passive recovery (IR-PR1) or active recovery (IR-AR). The active recovery was set at 50% of MAS and both recovery periods also lasted for 15 seconds. A third intermittent test was done where the exercise time was equal to the TTE for the active recovery protocol (IR-PR2). Their hypothesis was that TTE would be longer with active recovery compared to passive recovery. Their hypothesis was rejected, as TTE was significantly longer for IR-PR1 when compared to IR-AR. The mean TTE for IR-PR1 was 745 seconds compared to only 445 seconds with IR-AR. Mean blood lactate levels were lower with IR-AR (10.7 mmol) compared to IR-PR1 (11.7 mmol). However, this did not aid in prolonging TTE as previously described. Subsequently, total distance covered at 120% of MAS for IR-PR1 was 2,077 meters, compared to only 1,219 meters with IR-AR. IR-PR2 was performed to match the duration of the IR-AR to compare metabolic values. Blood lactate levels were lower (9.2 mmol) compared to IR-AR and IR-PR1. It could be determined from this study that the use of runs at 120% of MAS interspersed with slower runs that act as a form of active recovery could mimic workouts similar to a “fartlek”. Nevertheless, the results from this study indicate that performing shorter runs at supramaximal speeds with a short rest period favors passive recovery rather than active recovery.

Other research has shown similar findings, stating that active recovery does not enhance performance in subsequent bouts of performance (Abderrahman et al., 2012; Barnett, 2006). Another possibility for active recovery not being beneficial could be because of genetics, where the

ability to produce and remove lactate could be independent of recovery modality and more dependent on training status or the body's natural control of energy systems and mechanisms (Siegler et al., 2006; Denadai and Higino, 2004; Bret et al., 2012; Thomas et al., 2005).

Summary

Lactate has long been thought of as the culprit to decreased exercise performance. While there is more literature today that disputes this, there is still much published literature that refers to “lactic acidosis” as the cause of fatigue. It is important to recognize that the production and clearance of lactate now appears to be beneficial in delaying the onset of acidosis. A high reliance on non-mitochondrial ATP turnover during high-intensity exercise and the subsequent accumulation of metabolites is likely the real cause of acidosis and fatigue.

Although the clearance of lactate is important to continue exercise, it remains equivocal in the literature whether or not using active recovery protocols are able to improve subsequent performance within a training session. Several studies demonstrate that the improvement of lactate clearance is enhanced when active recovery is performed at or near the lactate threshold more so than at other intensities. This can be seen as a positive tool for training performance. In contrast, several studies acknowledge that active recovery promotes greater lactate clearance than passive recovery, but fails to improve performance in subsequent bouts of exercise. Much of these conflicting results could be due to the fact that the mode of exercise, the duration and intensity of the exercise bout, the duration and intensity of the active recovery protocol, and the training status of the participants have a wide range of variability. Studies with short, intermittent exercises separated by shorter periods of rest seem to benefit more from passive recovery to maintain performance (Dupont et al., 2003; Spencer et al., 2006; Toubekis et al., 2005; Brown & Glaister, 2014). As noted by Tokmakidis, Toubekis, and Smilios (2011), there were no studies

as of 2011 that looked at running and the effects of active and passive recovery on repeated performance where the running duration is long (40 to 120 seconds). Review of the current literature still failed to find a study that used repeated, high-intensity running as the modality when looking at active and passive recovery when the duration lasted between 40 and 120s. This gap in the literature signifies a missing piece of the recovery spectrum as it pertains to running. More research also needs to be done using more elite athletes such as collegiate or professionals and incorporating recovery protocols into their training sessions. This could potentially have more of a practical application for both athletes and coaches by programming recovery based off the intensity of the training for that day and the duration of the training session. Since the breadth of literature contains experiments that take place within a lab setting and not a training session, the usefulness of active recovery as a beneficial modality for athletes or recreational individuals can easily come into question. There could be a time window where the duration and intensity of an activity that elicits a high lactate accumulation and metabolic acidosis can be countered with an active recovery protocol that has a specific duration and is performed within a range of intensities. This time window for possible positive effects may only be perpetuated when the individual is in the actual training environment. However, this remains to be in question.

CHAPTER III

METHODS

Participants

Twenty adults, consisting of ten males and ten females, volunteered to participate in this study. Their demographic data is presented in Table 2. The participants were all physically active on a recreational basis. The study was approved by the University of North Dakota's Institutional Review Board and each participant signed an informed consent prior to the familiarization session. A Physical Activity Readiness Questionnaire (PAR-Q) was also completed by each participant to ensure no pre-existing or current conditions would be negatively affected through participation in the study.

Table 2. Participant demographics presented as Mean \pm Standard Deviation (Range)

Demographics	Males (n=10)	Females (n=10)
Age (years)	22.50 \pm 2.72 (19-28)	22.20 \pm 1.75 (19-28)
Height (in.)	69.90 \pm 2.92 (63-72)	64.60 \pm 1.90 (62-68)
Weight (lbs.)	166.56 \pm 19.47 (132-198.7)	138.11 \pm 17.45 (120.34-170.0)

Experimental Design

A crossover design was used to evaluate how the second 400m run was affected by the recovery condition when compared to the first 400m run. Before the experimental sessions took

place, a familiarization session was completed to introduce the components of the experimental session. The participants were taken through a standardized warm-up that consisted of a 5-minute aerobic run followed by various dynamic movements (high knees, lunges). After the warm-up, a 400 meter run was done to help participants be more comfortable with the track's length, turns, and the exhaustion that results from the run. After completing the run, the participants were instructed to perform an active recovery by either walking or jogging. The active recovery intensity was determined by the participant using Borg's Rating of Perceived Exertion (RPE) Scale (see Appendix C). Borg's RPE scale uses a 15-point grading system ranging in values from 6-20. The odd-numbered values on the scale correspond to a term that is used to describe the intensity associated with that particular value. For example, an RPE of 9 would be *very light* in intensity and an RPE of 17 would be *very hard* (Borg, 1982). Participants were instructed to perform an active recovery at an RPE of 11, which is *fairly light* intensity. The RPE scale and its' relationship to exercise intensity, which can be assessed by blood lactate or heart rate, has been shown to have a strong correlation and is independent of age, gender, level of physical activity, and exercise modality (Scherr et al., 2013).

The two experimental sessions were completed by each participant and were separated by at least 72 hours. A recovery condition (active or passive) was randomly assigned for the first session and the opposite condition was performed during the second session. Each session consisted of two running bouts of 400 meters separated by a 15 minute recovery period. Blood-lactate levels were taken 5 minutes and 12 minutes after the completion of the running bout during the 15 minute recovery period. The first blood-lactate measurement was done at 5 minutes because blood-lactate levels are estimated to peak between 3-8 minutes after maximal exercise (Goodwin et al., 2007). The second blood-lactate measurement was done at 12 minutes

to allow at least 3 minutes of passive recovery before the second run for PC resynthesis to occur. A schematic illustration of the experimental session procedure is presented in Figure 1.

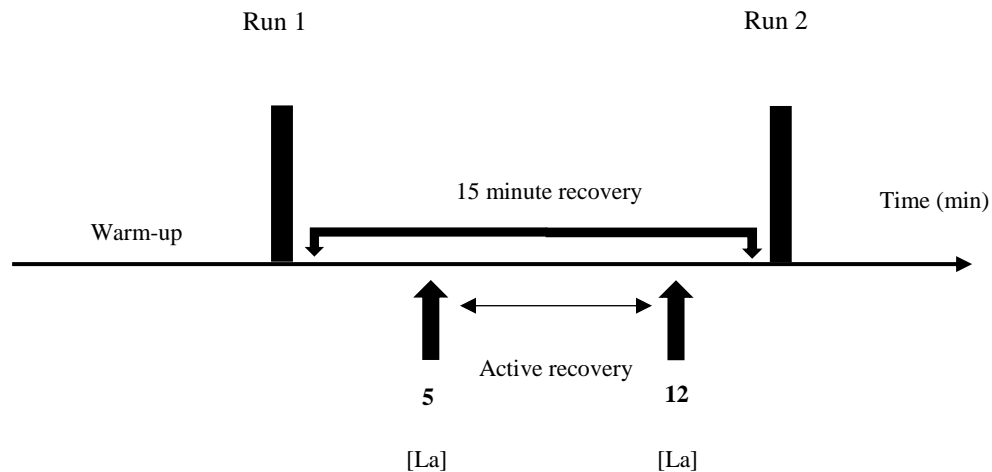


Figure 1. Experimental session protocol. Two 400m runs separated by 15 minutes. [La]: blood-lactate measurements taken. Active recovery condition performed between blood-lactate measurements. Passive recovery condition lasted entire 15 minutes.

Instrumentation

Height and weight were taken using a portable stadiometer (Seca Corp, Model 213, Hamburg, Germany) and an electronic scale (Seca Corp, Model 876, Hamburg, Germany). The familiarization and experimental sessions were conducted on an indoor running track that measures 146 meters in the lane the participants were designated to run in. Therefore, participants had to complete $2 \frac{3}{4}$ laps to reach the distance of 400 meters. A cone was placed at the starting line and two cones were placed at the finish line. Time was kept both for recovery times and performance time using a digital wristwatch (TIMEX Ironman 10 Lap memory, TIMEX, Middlebury, CT). Lactate levels were measured using a lactate meter analyzer (Nova Biomedical, Waltham, MA).

Procedure

Participants first attended a familiarization session where they were briefed on the study and provided consent as well as having their height and weight measured. Participants were then taken through the standardized warm-up and completed one run of 400 meters at max-effort. After the run, participants performed an active recovery at an intensity equivalent to a value of 11 (*fairly light*) according to Borg's RPE scale. After at least 48 hours, the participants would meet for session 1. A recovery condition of either active or passive was randomly assigned for this first session and the opposite condition would be done for the second session. After performing the standardized warm-up, participants completed the first 400 meter run at max effort. Verbal commands were given (On your marks, Get set, Go) at the beginning of the run. Verbal encouragement and the notification of one lap remaining was also given. After the first run was completed, participants were given 15 minutes of recovery before performing the second 400 meter run. Similar verbal instructions and encouragement for the first run were given on the second run.

Active Recovery Condition

When participants had the active recovery condition, the first 5 minutes of the recovery time would be passive. This was done to minimize bodily discomfort that could result after a high-intensity activity and also allowed blood-lactate levels to peak. After a lactate reading was gathered around the 5 minute mark, the participants began their active recovery equivalent to a value of 11 on the RPE scale. Participants performed this recovery for about 6 minutes until they were prepped for the second lactate measurement. After the second lactate measurement was gathered, participants remained passive for the remaining 3 minutes to provide restoration of PCr until it was time to perform the second 400 meter run.

Lactate Measurements

Blood-lactate values were measured at the 5 and 12 minute marks of the recovery period. To prepare for the measurement of blood- lactate, participants would have the distal end of their index finger of their non-dominant hand sterilized with an alcoholic pad. A lancet was used to prick this finger to draw a small amount of blood. The first drop of blood was wiped away and the second drop was used for the lactate strip inserted into the lactate analyzer. Participants were offered a bandage for the prick site if it didn't clot and stop on its' own. For the second blood-lactate measurement, the middle finger was used with a similar protocol to the first measurement (Maud & Foster, 2006).

Analysis

Data collected was analyzed using IBM SPSS software (SPSS v. 23, Chicago, IL). The difference in performance times between the two running bouts for each participant in each condition was calculated. This is the absolute time difference (measured in seconds) for the active condition and the passive condition. The difference in blood-lactate values between the first and second measurements for each participant in each condition was calculated. This is the absolute blood-lactate difference (measured in mmol) for the active and passive condition. The change scores are presented as a percentage. Change scores for performance times and blood-lactate were determined by taking the second value minus the first value, dividing by the initial value, and then multiplying by 100. A dependent-sample t-test was used to detect statistically significant differences between the data of the two recovery conditions. The significance level was set at $p < .05$.

CHAPTER IV

RESULTS

The main objective of this study was to investigate how performing an active vs. passive recovery would affect subsequent performance in a 400m run. The mean and standard deviation values of the 400 meter run times (in seconds) and change score percentages are presented in Table 3. Performance times were measured in seconds. Mean blood-lactate measurements and change scores are presented in Table 4. There was a statistically significant difference in performance times between pre- (M = 76.31, SD = 13.42) and post- (M = 79.57, SD = 14.62) recovery for the active condition; $t(19) = -2.88, p = .01$. There was also a statistically significant difference between the pre- (M = 76.23, SD = 14.20) and post- (M = 78.74, SD = 13.23) recovery times for the passive condition; $t(19) = -3.73, p = .001$. Performance times for the second 400 meter run were slower by an average of 3.26 (4.3%) seconds in the active condition and 2.51 (3.7%) seconds in the passive condition, respectively. When the absolute time differences were compared, there was no statistical difference between the conditions (M = -.75, SD = 6.61); $t(19) = -.510, p = .616$. There was also no statistical difference when the change scores between active and passive were compared (M = .66, SD = 7.26); $t(19) = .407, p = .688$.

Table 3. Mean averages and change scores for 400m finish times

Run time #1 - Active	76.31 ± 13.42 s
Run time #2 - Active	79.57 ± 14.62 s*
Run time #1 - Passive	76.23 ± 14.20 s
Run time # 2 - Passive	78.74 ± 13.23 s*
Absolute time difference between conditions	-.75 ± 6.61 s
% change - Active	4.34 ± 6.26 %
% change - Passive	3.67 ± 4.43 %
Change score difference between conditions	.66 ± 7.26 %

*Statistically significant difference from run time #1 ($p < .05$)

The active recovery condition produced a statistically significant difference between blood-lactate measurements taken at 5 minutes ($M = 12.65$, $SD = 2.72$) and 12 minutes ($M = 10.07$, $SD = 3.41$) of the recovery time; $t(16) = 2.82$, $p = .012$. Mean blood-lactate measurements for the passive recovery condition were not statistically different between the 5 ($M = 12.76$, $SD = 3.15$) and 12 ($M = 12.04$, $SD = 4.00$) minute marks; $t(17) = 1.19$, $p = .251$. Blood-lactate decreased by an average of 2.58 mmol in the active recovery condition and .722 mmol in the passive recovery condition, respectively. When the absolute blood-lactate difference was compared between conditions, there was no statistically significant difference ($M = 2.00$, $SD = 5.18$); $t(16) = 1.60$, $p = .130$. The calculated change scores indicate blood-lactate levels decreased by an average 16.3% in the active condition compared to a 5.5% reduction in the passive condition. However, this difference failed to reach statistical significance as well ($M = 10.75$, $SD = 23.01$); $t(15) = -1.87$, $p = .081$.

Table 4. Mean blood-lactate at 5 minutes and 12 minutes of recovery and change scores

Lactate @ 5 – Active	12.65 ± 2.72 mmol
Lactate @ 12 - Active	10.07 ± 3.41 mmol*
Lactate @ 5 - Passive	12.76 ± 3.15 mmol
Lactate @ 12 - Passive	12.04 ± 4.00 mmol
Absolute blood-lactate difference between conditions	2.01 ± 5.18 mmol
% change - Active	-16.29 ± 31.46 %
% change - Passive	-5.54 ± 22.73 %
Change score difference between conditions	10.75 ± 23.01 %

*Statistically significant difference from blood-lactate value at 5 minutes (p < .05)

CHAPTER V

DISCUSSION

The purpose of this study was to examine the effects of an active vs. passive recovery condition on subsequent, high-intensity running bout performance in recreational adult runners. The results of this study indicate that there may not be any advantage of using an active recovery when performing a repeated, max-effort 400m run compared to a passive recovery in a recreationally active population. The active recovery condition appeared to trend towards lower blood-lactate levels, but this did not lead to an improvement in subsequent performance times. On average, time to completion after performing an active recovery increased by 3.26 seconds (4.34%) and by 2.51 seconds (3.67%) using a passive recovery. However, these differences are not statistically significant. Blood-lactate levels were similar at 5 minutes for both recovery conditions (12.65 mmol for active, 12.76 mmol for passive), indicating similar workloads.

It has been well-established that performing an active recovery leads to a faster reduction in blood-lactate concentrations after a max-effort exercise trial (Weltman, Stamford, & Fulco, 1979; Menzies et al., 2010; Devlin et al., 2014). The blood-lactate values obtained in this study during the active recovery condition somewhat reiterated this concept, as there was a lower value of blood-lactate taken at 12 minutes compared to the passive condition. However, this difference was not statistically significant and the completion times for the second 400m run were slower in both conditions. This contradicts previous research that has found that performing an active

recovery improves or sustains subsequent performance (Felix et al., 1997; Greenwood et al., 2008).

The recovery duration could have contributed to the absence of an improved performance in the second 400m run in this study. The recovery duration of 15 minutes is similar to previous studies using 10 minutes (Greenwood et al., 2008) and 14 minutes (Felix et al., 1997). This recovery duration is also a realistic component in a training session that uses longer sprint repetitions (Tokmakidis, Toubekis, and Smilios, 2011). Although 15 minutes of total recovery was given, the participants only performed an active recovery for approximately 6 minutes. This may not have been enough time to facilitate substantial lactate removal and provide a more favorable condition for subsequent performance.

The intensity of the active recovery was self-selected by the participants using Borg's 6-20 RPE scale. The participants were instructed to perform their active recovery at a value of 11 on the scale, which subjectively equates to a fairly-light intensity. This resulted in the participants performing a light jog or walking, while some performed a combination of both. It is possible that the participants in the current study did not perform the active recovery at a high enough intensity to maximize blood-lactate reduction and improve subsequent performance when compared to passive recovery. There has been an increased understanding that there may be an intensity-dependent relationship regarding the clearance time of blood-lactate. Menzies and colleagues (2010) reported that performing an active recovery at 80-100% of lactate threshold was the most effective intensities at reducing blood-lactate following a 5 minute run at 90% VO_{2max} . Devlin and colleagues (2014) were able to demonstrate the same effectiveness of using 80% of one's lactate threshold as the ideal intensity to maximize lactate clearance after maximal

running. Utilizing lactate threshold is thought to be beneficial because of the increase in blood-lactate clearance without the production of more lactate.

The results of this study do not support the use of active recovery based off of a RPE of 11 and can't be recommended as general practice. Individuals performing high-intensity workloads could choose to attempt an active recovery using RPE and compare their performances to when they remain passive. As the exercise duration increases, there may be more of a benefit to incorporate an active recovery, particularly if the allotted recovery duration is 10-20 minutes in training sessions (Tokmakidis, Toubekis, & Smilios, 2011). In this study, neither recovery condition was favorable over the other when the participants were asked if they had a preference, however this data was not collected.

Future research should attempt to understand the implications of different recovery methods on repeated, middle-distance running performance. Although RPE is a good indicator of intensity during exercise, active recovery intensities based off individual lactate threshold may be superior, and can be used if the values are known. It is unknown how recovery intensities based off of performance speeds could affect subsequent bouts, providing an opportunity for future research as well. The participants in the current study were instructed to complete the 400m run as fast as possible with no specific pacing strategy required. It would be interesting to examine the use of a specific 400m pacing strategy, such as that described by Saraslanidis et al., (2011), and how it affects completion times using a similar experimental set-up in the current study. Although research exists for short-duration sprints, such as 30 seconds and under, there appears to be a limited body of research that examines middle-distance running performance with respect to active and passive recoveries. These future researchers should also attempt to mimic training conditions within the experimental set-up, such as using a running track instead

of a treadmill; and prescribing intensity based of off previously completed performance times. This may have a better application for coaches and individuals runners.

Limitations

The design of this study had several limitations that may have influenced the results. Although the participants self-reported as recreationally active runners, some may have been in a less advanced training state, leading to an increased variability in their performance times regardless of recovery condition. The sample size is relatively small, affecting the statistical power and the ability to detect small effects. The completion times for the running bouts were gathered manually rather than using an automated-timing system, possibly leading to some error in completion time.

Conclusion

This study looked into the effects of performing an active vs. passive recovery when attempting to repeat high-intensity 400m runs in recreational adult runners. Our results do not support the use of active recovery based of off RPE to improve subsequent running performance. If individual runners prefer active recovery, it does not appear to be detrimental. It may be more beneficial for individual runners or coaches to prescribe active recovery at a percentage of lactate threshold to maximize lactate clearance and improve performance. Future research should examine the use of percentages of performance speed or higher RPE values as the intensity for active recovery.

APPENDICES

Appendix A

Consent Form

Effects of Active vs. Passive Recovery on Subsequent Bouts of High-Intensity Performance in Recreational Runners

University of North Dakota

You are invited to participate in a research study assessing the use of two recovery protocols on subsequent bouts of high-intensity performance. Please read this form and ask any questions that you may have.

The principal investigator (person conducting the research) is Matthew McCreary, B.S. He is a graduate student in the Kinesiology Department at the University of North Dakota. This research study is being done as a Thesis project.

Study Purpose

The purpose of this study is to assess how the performances in bouts of running performed at a high-intensity are affected by the use of active recovery (AR) and passive recovery (PR). Active recovery is done during the designated recovery period and is typically the same modality (e.g. running, cycling) as the exercise previously completed. Active recovery is also performed at an intensity that is lighter than that of the exercise bout (similar to a cool-down). Passive recovery usually consists of no movement at all; however slow walking will be permitted in this study.

Study Procedures

If you choose to participate in this study, you will be asked to partake in three sessions: a familiarization session and two experimental sessions. All sessions will take place approximately one week apart from each other at the Hyslop Sports Arena.

Session 1: Familiarization: This session is essentially a practice for the experimental sessions. First, demographic data such as age, height, and weight will be recorded. Next, you will complete two runs at a distance of 500 meters with 15 minutes of AR in between. Intensity of the AR will be determined using the principle of RPE (Ratings of Perceived Exertion). RPE is a tool used to assess self-perception of effort during exercise. RPE will be assessed by using Borg's RPE Scale, where values range from 6-20.

During the recovery period, two small capillary blood samples will be taken on your index finger of your non-dominant hand, one at approximately 5 minutes of recovery and again at approximately 12 minutes. A dynamic-warm-up will be performed before beginning the first running bout.

Session 2:

- This session will have you perform either an active or passive recovery. You will be randomized to perform one of them during the 15 minute period between the 500 meter runs.
- AR will be performed at what you feel to be an RPE of 11.
- The same blood collection procedure done in session 1 will be done during this session.

Session 3: This session will be similar to session 2. The only difference is that you will perform the opposite recovery protocol from session 1 for this session.

Possible Risks

This study does not create any other possible risk than that already associated with a high-intensity training session. A warm-up will be performed prior to the sessions to prepare muscle contraction, blood flow, heart rate, and ventilation for the stress associated with exercise. Feelings of discomfort may follow the initial completion of your running bout and may last even during your recovery period. This test involves collecting two (2) small capillary blood samples by a finger prick during your recovery period. There may be some slight discomfort and tenderness at the finger prick site. Proper steps will be taken to ensure the finger prick site is appropriately selected, sterilized, cleaned, and bandaged if necessary.

Benefits of Study Participation

You may benefit from this study by understanding how recovery affects subsequent performances. You may choose to use what you learned in this study for future training sessions.

Compensation

You will not be compensated for participation in this study.

Confidentiality

The personal information gathered from this study will be kept private. Any publication or presentation will not include information that will be able to identify you as a participant. Only the research personnel will have access to your information.

Is this study voluntary?

Your participation is voluntary. You may choose not to participate or you may discontinue your participation at any time without penalty. Your decision whether or not to participate will not affect your current or future relations with the University of North Dakota.

Questions or Comments

If you have any questions, concerns, or comments regarding the research please do not hesitate to contact the primary investigator, Matthew McCreary.

- Phone: (218) 779-9481
- E-mail: matthew.mccreary@my.und.edu

If you have questions regarding your rights as a research subject, you may contact The University of North Dakota Institutional Review Board at (701) 777-4279.

Appendix B

Physical Activity Readiness Questionnaire

Physical Activity Readiness
Questionnaire - PAR-Q
(revised 2002)

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO	
<input type="checkbox"/>	<input type="checkbox"/>	1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?
<input type="checkbox"/>	<input type="checkbox"/>	2. Do you feel pain in your chest when you do physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	2. In the past month, have you had chest pain when you were not doing physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	4. Do you lose your balance because of dizziness or do you ever lose consciousness?
<input type="checkbox"/>	<input type="checkbox"/>	5. Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
<input type="checkbox"/>	<input type="checkbox"/>	7. Do you know of any other reason why you should not do physical activity?

If
you
answered

YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

If you answered NO honestly to **all** PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active — begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal — this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively. It is also highly recommended that you have your blood pressure evaluated. If your reading is over 144/94, talk with your doctor before you start becoming much more physically active.

DELAY BECOMING MUCH MORE ACTIVE:

- if you are not feeling well because of a temporary illness such as a cold or a fever — wait until you feel better; or
- if you are or may be pregnant — talk to your doctor before you start becoming more active.

PLEASE NOTE: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

No changes permitted. You are encouraged to photocopy the PAR-Q but only if you use the entire form.

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

NAME _____

SIGNATURE _____

DATE _____

SIGNATURE OF PARENT
or GUARDIAN (for participants under the age of majority) _____

WITNESS _____

Note: This physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if your condition changes so that you would answer YES to any of the seven questions.



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Appendix C

Borg's 6-20 Ratings of Perceived Exertion (RPE) Scale

6

7 very, very light

8

9 very light

10

11 fairly light

12

13 somewhat hard

14

15 hard

16

17 very hard

18

19 very, very hard

20

REFERENCES

1. Abderrahman, A., Zouhal, H., Chamari, K., Thevenet, D., de Mullenheim, P., Gastinger, S., Tabka, Z., & Prioux, J. (2012). Effects of recovery mode (active vs. passive) on performance during a short high-intensity interval training program: a longitudinal study. *European Journal of Applied Physiology*, *113*, 1373-1383.
2. Allen, D., & Trajanovska, S. (2012). The multiple roles of phosphate in muscle fatigue. *Frontiers in Physiology*, *3*, 1-9.
3. Ament, W., & Verkerke, G. (2009). Exercise and Fatigue. *Sports Medicine*, *39*(5), 389-422.
4. Barnett, A. (2006). Using Recovery Modalities between Training sessions in Elite Athletes: Does it help? *Sports Medicine*, *36* (9), 781-796.
5. Borg, G. (1982). Psychophysical bases of perceived exertion. *Medicine and Science in Sports and Exercise*, *14*(5), 377-381.
6. Bret, C., Lacour, J., Bourdin, M., Locatelli, E., Angelis, M., Faina, M., Rahmani, A., & Messonnier, L. (2012). Differences in lactate exchange and removal abilities between high-level African and Caucasian 400-m track runners. *European Journal of Applied Physiology*, *113*, 1489-1498.
7. Brooks, G., Fahey, T., & Baldwin, K. (2004). *Exercise Physiology: Human Bioenergetics and its Applications* (4th Ed.). New York, NY: McGraw-Hill
8. Brown, J., & Glaister, M. (2014). The Interactive Effects of Recovery Mode and Duration on Subsequent Repeated Sprint Performance. *Journal of Strength and Conditioning Research*, *28*(3), 651-660.
9. Cairns, S. (2006). Lactic Acid and Exercise Performance: Culprit or Friend? *Sports Medicine*, *36*(4), 279-291.

10. Choi, D., Cole, K., Goodpaster, B., Fink, W., & Costill, D. (1994). Effect of passive and active recovery on the resynthesis of muscle glycogen. *Medicine and Science in Sports and Exercise*, 26(8), 992-996.
11. Debold, E. (2012). Recent insights into muscle fatigue at the cross-bridge level. *Frontiers in Physiology*, 3(151), 1-14.
12. De Pauw, K., De Geus, B., Roelands, B., Lauwens, F., Verschueren, J., Heyman, E., & Meeusen, R. (2011). Effect of Five Different Recovery Methods on Repeated Cycle Performance. *Medicine and Science in Sports and Exercise*, 43(5), 890-897.
13. Del Coso, J., Hamouti, N., Aguado-Jimenez, R., & Mora-Rodriguez, R. (2010). Restoration of blood pH between repeated bouts of high-intensity exercise: effects of various active recovery protocols. *European Journal of Applied Physiology*, 108, 523-532.
14. Denadai, B. & Higino, W. (2004). Effect of the passive recovery period on the lactate minimum speed in sprinters and endurance runners. *Journal of Science and Medicine in Sport*, 7(4), 488-496.
15. Devlin, J., Paton, B., Poole, L., Sun, W., Ferguson, C., Wilson, J., & Kemi, OJ. (2014). Blood lactate clearance after maximal exercise depends on active recovery intensity. *The Journal of Sports Medicine and Physical Fitness*, 54(3), 271-278.
16. Dodd, S., Powers, S., Callender, T., & Brooks, E. (1984). Blood Lactate disappearance at various intensities of recovery exercise. *Journal of Applied Physiology*, 57(5), 1462-1465.
17. Dupont, G., Blondel, N., & Berthoin, S. (2003). Performance for short intermittent runs: active recovery vs. passive recovery. *European Journal of Applied Physiology*, 89, 548-554.
18. Dupont, G., Moalla, W., Guinhouya, C., Ahmaidi, S., & Berthoin, S. (2004). Passive versus Active Recovery during High-Intensity Intermittent Exercises. *Medicine and Science in Sports and Exercise*, 36(2), 302-308.
19. Fairchild, T., Armstrong, A., Rao, A., Liu, H., Lawrence, S., & Fournier, P. (2003). Glycogen synthesis in Muscle Fibers during Active Recovery from Intense Exercise. *Medicine and Science and Sports and Exercise*, 35(4), 595-602.
20. Felix, S., Manos, T., Jarvis, A., Jensen, B., & Headley, S. (1997). Swimming performance following different recovery protocols in female collegiate swimmers. *The Journal of Swimming Research*, 12, 1-6.

21. Finsterer, J. (2012). Biomarkers of peripheral muscle fatigue during exercise. *BMC Musculoskeletal Disorders*, 13(218), 1-13.
22. Fitts, R. (2008). The cross-bridge cycle and skeletal muscle fatigue. *Journal of Applied Physiology*, 104, 551-558.
23. Froyd, C., Millet, G., & Noakes, T. (2013). The development of peripheral fatigue and short-term recovery during self-paced high-intensity exercise. *The Journal of Physiology*, 591(5), 1339-1346.
24. Goodwin, M., Harris, J., Hernandez, A., & Gladden, L. (2007). Blood Lactate Measurements and Analysis during Exercise: A Guide for Clinicians. *Journal of Diabetes Science and Technology*, 1(4), 558-569.
25. Greenwood, J., Moses, G., Bernardino, F., Gaesser, G., & Weltman, A. (2008). Intensity of exercise recovery, blood lactate disappearance, and subsequent swimming performance. *Journal of Sports Sciences*, 26(1), 29-34.
26. Hashimoto, T., & Brooks, G. (2008). Mitochondrial Lactate Oxidation Complex and an Adaptive Role for Lactate Production. *Medicine and Science in Sports and Exercise*, 40(3), 486-494
27. Juel, C. (2001). Current aspects of lactate exchange: lactate/H⁺ transport in human skeletal muscle. *European Journal of Applied Physiology*, 86, 12-16.
28. Koizumi, K., Fujita, Y., Muramatsu, S., Manabe, M., Ito, M., & Nomura, J. (2011). Active recovery effects on local oxygenation level during intensive cycling bouts. *Journal of Sports Sciences*, 29(9), 919-926.
29. Maud, P. & Foster, C. (2006). *Physiological Assessment of Human Fitness*. (2nd ed.), Champaign, IL: Human Kinetics.
30. McAinch, A., Febbraio, M., Parkin, J., Zhao, S., Tangalakis, K., Stojanovska, L., & Carey, M. (2004). Effect of Active versus Passive Recovery on Metabolism and Performance during Subsequent Exercise. *International Journal of Sport Nutrition and Exercise Metabolism*, 14, 185-196.
31. Menzies, P., Menzies, C., McIntyre, L., Paterson, P., Wilson, J., & Kemi, O. (2010). Blood Lactate clearance during active recovery after an intense running bout depends on the intensity of the active recovery. *Journal of Sports Sciences*, 28(9), 975-982.
32. Moxnes, J. & Sandbakk, O. (2012). The kinetics of lactate production and removal during whole-body exercise. *Theoretical Biology and Medical Modeling*, 9(7), 1-14.

33. Robergs, R., Ghiasvand, F., & Parker, D. (2004). Biochemistry of exercise-induced metabolic acidosis. *American Journal of Physiology - Regulatory, Integrative and Comparative Physiology*, 287, 502-516.
34. Robergs, R. (2011). Letter: Nothing 'evil' and no 'conundrum' about muscle lactate production. *Experimental Physiology*, 96(10), 1097-1098.
35. Saraslanidis, P., Panoutsakopoulos, V., Tsalis, G., & Kyprianou, E. (2011). The effect of different first 200-m pacing strategies on blood lactate and biomechanical parameters of the 400-m sprint. *European Journal of Applied Physiology*, 111, 1579-1590.
36. Scherr, J., Wolfarth, B., Christle, J., Pressler, A., Wagenpfeil, S., & Halle, M. (2013). Associations between Borg's rating of perceived exertion and physiological measures of exercise intensity. *European Journal of Applied Physiology*, 113, 147-155.
37. Siegler, J., Bell-Wilson, J., Mermier, C., Faria, E., & Robergs, R. (2006). Active and Passive Recovery and Acid-Base Kinetics Following Multiple Bouts of Intense Exercise to Exhaustion. *International Journal of Sport Nutrition and Exercise Metabolism*, 16, 92-107.
38. Spencer, M., Bishop, D., Dawson, B., Goodman, C., & Duffield, R. (2006). Metabolism and Performance in Repeated Cycle Sprints: Active versus Passive Recovery. *Medicine and Science in Sports and Exercise*. 38(8), 1492-1499.
39. Spierer, D., Goldsmith, R., Baran, D., Hryniewicz, K., & Katz, S. (2004). Effects of Active vs. passive Recovery on Work Performed during Supramaximal Exercise Tests. *International Journal of Sports Medicine*, 25, 109-114.
40. Thomas, C., Bishop, D., Lambert, K., Mercier, J., & Brooks, G. (2012). Effects of acute and chronic exercise on sarcolemmal MCT1 and MCT4 contents in human skeletal muscles: current status. *The American Journal of Physiology – Regulatory, Integrative, and Comparative Physiology*, 302, R1-R14.
41. Thomas, C., Perrey, S., Lambert, K., Hugon, G., Mornet, D., & Mercier, J. (2005). Monocarboxylate transporters, blood lactate removal after supramaximal exercise, and fatigue indexes in humans. *Journal of Applied Physiology*, 98, 804-809.
42. Tokmakidis, S., Toubekis, A., & Smilios, I. (2011). Active Versus Passive Recovery: Metabolic Limitations and Performance Outcome. In M. Powell (Ed.), *Physical Fitness: Training, Effects, and Maintaining* (pp. 1-43). New York: Nova Science Publishers.
43. Toubekis, A., Douda, H., & Tokmakidis, A. (2005). Influence of different rest intervals during active or passive recovery on repeated sprint swimming performance. *European Journal of Applied Physiology*, 93, 694-700.

44. Weltman, A., Stamford, B., & Fulco, C. (1979). Recovery from maximal effort exercise: lactate disappearance and subsequent performance. *Journal of Applied Physiology: Respiratory, Environmental, and Exercise Physiology*, 47(4), 677-682.
45. Yoshida, T., Watari, H., & Tagawa, K. (1996). Effects of Active and Passive Recoveries on Splitting of the Inorganic Phosphate Peak Determined by ³¹P-Nuclear Magnetic Resonance Spectroscopy. *NMR In Biomedicine*, 9, 13-19.