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## **Abstract**

The purpose of this study was to examine pain perception during and following two separate 30-min bouts of exercise above and below the Lactate Threshold (LT). Pain Threshold (PT) and Pain Intensity (PI) were monitored during (15 and 30 min) and after exercise (15 and 30 min into recovery) using a Cold Pressor Test (CPT) and Visual Analog Scale (VAS) for pain of the non-dominant hand. Significant differences in PT scores were found both during and after exercise conditions. Post hoc analysis revealed significant differences in PT scores at 30 min of exercise ( $P=0.024$ ,  $P=0.02$ ) and 15 min of recovery ( $P=0.03$ ,  $P=0.01$ ) for exercise conditions above and below LT, respectively. No differences ( $P=0.05$ ) in PT scores were found at any time point between exercise conditions. No differences were found in PI scores at any time point within each trial ( $P=0.05$ ) as well as between exercise conditions ( $p=0.05$ ). Based upon these data, the effects of moderate exercise on PT appear to be similar at exercise intensities just above and below LT. This may indicate that the requisite intensity needed to elicit Exercise-Induced Hypoalgesia may be lower than previously reported. Because a hypoalgesic effect was not observed in either condition until 30 min of exercise had been completed, total exercise time may be an important factor in the augmentation of pain perception under these conditions.

## **Keywords**

Pain Perception, Lactate Threshold, Exercise Induced Hypoalgesia

## **Disciplines**

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## Exercise Intensity as a Determinant of Exercise Induced Hypoalgesia

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**ABSTRACT**

**Wonders KY, Drury DG.** Exercise Intensity as a Determinant of Exercise Induced Hypoalgesia. **JEPonline** 2011;14(4):134-144. The purpose of this study was to examine pain perception during and following two separate 30-min bouts of exercise above and below the Lactate Threshold (LT). Pain Threshold (PT) and Pain Intensity (PI) were monitored during (15 and 30 min) and after exercise (15 and 30 min into recovery) using a Cold Pressor Test (CPT) and Visual Analog Scale (VAS) for pain of the non-dominant hand. Significant differences in PT scores were found both during and after exercise conditions. Post hoc analysis revealed significant differences in PT scores at 30 min of exercise ( $P=0.024$ ,  $P=0.02$ ) and 15 min of recovery ( $P=0.03$ ,  $P=0.01$ ) for exercise conditions above and below LT, respectively. No differences ( $P=0.05$ ) in PT scores were found at any time point between exercise conditions. No differences were found in PI scores at any time point within each trial ( $P=0.05$ ) as well as between exercise conditions ( $p=0.05$ ). Based upon these data, the effects of moderate exercise on PT appear to be similar at exercise intensities just above and below LT. This may indicate that the requisite intensity needed to elicit Exercise-Induced Hypoalgesia may be lower than previously reported. Because a hypoalgesic effect was not observed in either condition until 30 min of exercise had been completed, total exercise time may be an important factor in the augmentation of pain perception under these conditions.

**Key Words:** Pain Perception, Lactate Threshold, Exercise Induced Hypoalgesia

## INTRODUCTION

A number of investigators have raised the idea that various forms of exercise can result in a reduced sensitivity to painful stimuli. This phenomenon is referred to as Exercise-Induced Hypoalgesia (EIH), and has been observed repeatedly using a variety of noxious stimuli, including electrical, thermal, pressure, and ischemic (1,24,23,20,29,33). This effect appears to be most reliable following relatively high intensity exercise (23,24,27).

While the modulation of pain during and following exercise is well documented in the literature, its underlying physiological mechanisms have yet to be elucidated. Much attention has been focused on a possible physiological link between pain augmentation and exercise. More specifically, pH changes, hypoxia, and lactic acid accumulation have all been identified as important modulators of pain (19). During moderate to high intensity exercise, lactic acid is continually produced and eliminated in working cells. As the intensity of exercise increases, the production of lactic acid begins to exceed the rate of its elimination, thereby causing lactic acid to accumulate inside and around the active cells. Eventually, enough lactic acid accumulates inside a tissue so that it surpasses the cell's holding capacity, causing lactic acid to spill out of the cell into the bloodstream (termed "onset of blood lactic acid" or OBLA). Once high levels of lactic acid have accumulated in the tissue, lactic acid dissociates a hydrogen ion, which can stimulate pain receptors in the brain and produce an uncomfortable sensation in the working muscles. From a practical perspective, one's OBLA is an early indicator of a switch in metabolism from aerobic to anaerobic. From a pain perspective, this change in metabolism may be an important factor in understanding how and when EIH occurs.

Since OBLA and EIH are both typically observed during relatively high intensity of exercise, it seems plausible to suggest that the metabolic byproducts associated with anaerobic energy production (pH change and lactic acid accumulation) may somehow be related to the temporary change in pain perception observed during and after exercise. Therefore, the purpose of this investigation was to examine Pain Threshold (PT) and Pain Intensity (PI) during and following two metabolically different bouts of exercise. One exercise was performed at an intensity corresponding to a heart rate 10% above and the other 10% below the heart rate (HR) corresponding to the OBLA.

## METHODS

### Subjects

A total of 27 normotensive healthy males were included in this study. Exclusion criteria included acute or chronic pain of any kind and the use of psychoactive drugs, analgesics, or medications affecting the cardiovascular system. Subjects were asked to refrain from caffeine, nicotine, alcohol, and strenuous exercise for at least 4 hr before their arrival at the laboratory. All methods were approved by the Wright State University Institutional Review Board prior to data collection and compliance to the exclusion criteria was self reported with a health history questionnaire. The estimated sample size required to detect significant differences using PT and PI and their interaction was calculated based on an alpha level of  $P=0.05$ , a power level of 0.80, and a moderate effect size (4). Through a power analysis it was estimated that approximately 26 to 28 subjects would be needed to detect significant differences.

### Pain Threshold Assessment

Pain Threshold (PT) was measured using the Cold Pressor Test (CPT) (10,40). The apparatus for the cold pressor consisted of a container filled with ice and water that was maintained between 1°C and 3°C. The use of a water circulator (Micro-Mark 83345) prevented the water from warming near the subject's hand. In order to control for possible variations in skin temperature, subjects placed

their non-dominant hand and forearm in a water bath of 37°C for 3 min prior to testing. At the onset of the test, subjects were instructed to submerge their non-dominant hand to a marked line at the level of the styloid process of the ulna and to remain still. Subjects were asked to indicate when the sensations in their hand first became painful. The time (sec) it took each subject to first feel painful sensations in their hands was recorded and served as their PT score. A maximum time limit of 5 min was imposed, though subjects were not informed of this limit prior to testing.

### **Pain Intensity Assessment**

In an effort to assess the intensity of the perceived pain, a Visual Analog Scale (VAS) was used (3). A 10-cm line was presented to each subject at the conclusion of the CPT in order to quantify the Pain Intensity (PI). The subject was asked to draw a line somewhere on the line with the left hand side of the line corresponding with no pain and the right end of the line indicating the greatest pain ever felt by the subject. Scores were obtained by measuring the distance from the left side of the line and were recorded in millimeters (mm).

### **Data Collection Procedure**

Subjects reported to the laboratory on four separate occasions within a 10-day period with each session being completed at approximately the same time of day. On day one, basic anthropometric measurements (height, weight, and body composition) were collected and, then, the subject was then prepped for a 12-lead electrocardiogram using standard procedures.

### **Baseline Condition**

After being prepped, the subject sat quietly for 10 min to promote a resting physiological state. At the conclusion of the rest period, nociceptive variables (PT and PI) and HR were obtained as baseline measurements. The subject remained seated for a total of 60 min. The Dependent Variables (PT and PI) were measured at 15, 30, 45, and 60 min.

### **Workload Determination Visit**

During the second visit, each subject was prepped for continuous electrocardiogram measurement using the methods described above. After being prepped, each subject sat quietly for approximately 10 min and then a resting HR was recorded. Next, each subject completed a peak treadmill exercise test using the Bruce protocol (2) to estimate maximal  $\text{VO}_2$ . Heart rate and Rate of Perceived Exertion (RPE) Scores were monitored at each stage of the protocol. In addition, blood lactate levels were obtained at the end of each stage and were used to determine the stage corresponding to the onset of blood lactic acid (OBLA). Lactate analysis was completed using a finger stick portable lactate analyzer (Accutrend). OBLA was defined when blood lactate concentrations reached 4 mmol/L (15,25) and the HR corresponding with this level was used to determine exercise workloads.

Once each subject's OBLA was identified, two workloads were estimated based upon the HR and stage where OBLA was observed. The two exercise conditions were determined by subtracting or adding beats onto the OBLA HR. Therefore, the Below (BLW)-OBLA was determined as follows:  $(\text{OBLA HR}) - (10\% \text{ OBLA HR}) = \text{BLW-OBLA HR}$ . The Above (ABV) OBLA HR was calculated in a similar manner by adding the same number of beats instead of subtracting. During the exercise trials the treadmill speed was adjusted so that the subject would maintain a steady state HR ( $\pm 3$  beats) at these pre-determined HRs.

### **Exercise Visits**

During the third and fourth visits, the subject exercised for 30 min at each of the workloads described above, with the order of these visits being randomized. During each visit, the cold pressor test was administered at baseline, during exercise (at the 15th and 30th min), and during recovery (at the 15th

and 30th min). Pain Threshold and PI were calculated based upon the duration of time (in sec) before the onset of pain and the quantity of the pain once it was experienced (VAS score).

### Data Analysis

Descriptive statistics have been computed as means and standard deviations. A two-factor repeated-measures ANOVA using within subjects main effect was used to determine if exercise intensity significantly altered nociceptive variables. A standard repeated measures ANOVA was used to compare pain scores to one another during the baseline trial. In the presence of significant differences, a Tukey *post hoc* was performed. A significance level of  $P=0.05$  was used for all statistical analyses.

## RESULTS

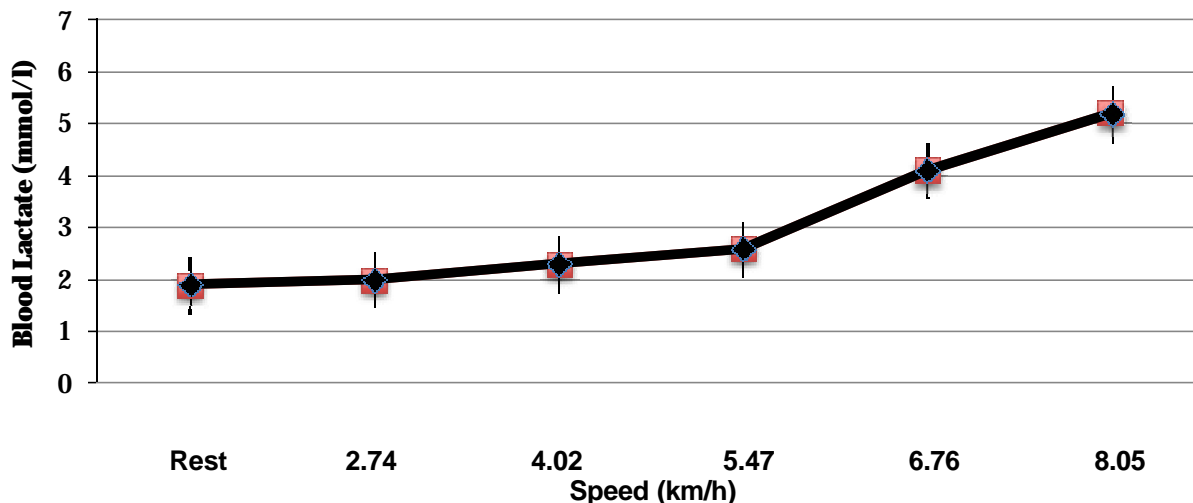
### Anthropometric and Exercise Pre-test Data

Table 1 presents the subject anthropometric characteristics, maximal HR scores as well as the respective  $VO_2$  predictions scores. On average, subjects reached OBLA during stage 4 of the graded exercise test and at a corresponding  $VO_2$  of approximately 73% of their estimated  $VO_2$  peak (Figure 1). However, the workloads for ABV-OBLA and BLW-OBLA were individualized according to the specific stage and corresponding HR each subject reached at the OBLA. Therefore, the two different exercise bouts corresponded to HRs maintained at approximately 63% (BLW-OBLA) and 83% (ABV-OBLA) of each subject's OBLA HR. Table 2 presents the HR response to each workload as well as the responses that corresponded to OBLA.

**Table 1. Subject Characteristics. Values are  $M \pm SE$ .**

Age (yrs)	$21.8 \pm 0.2$
Percent body fat (%)	$14.04 \pm 5.24$
Height (in)	$71.8 \pm 2.57$
Weight (lbs)	$171 \pm 13.72$
BMI ( $kg/m^2$ )	$23.06 \pm 1.41$
Max HR ( $beats \cdot min^{-1}$ )	$186 \pm 8.2$
Estimated $VO_{2max}$ ( $mL \cdot kg^{-1} \cdot min^{-1}$ )	$47.5 \pm 4.62$

**Figure 1. Lactate Curve. Values are  $M \pm SE$**



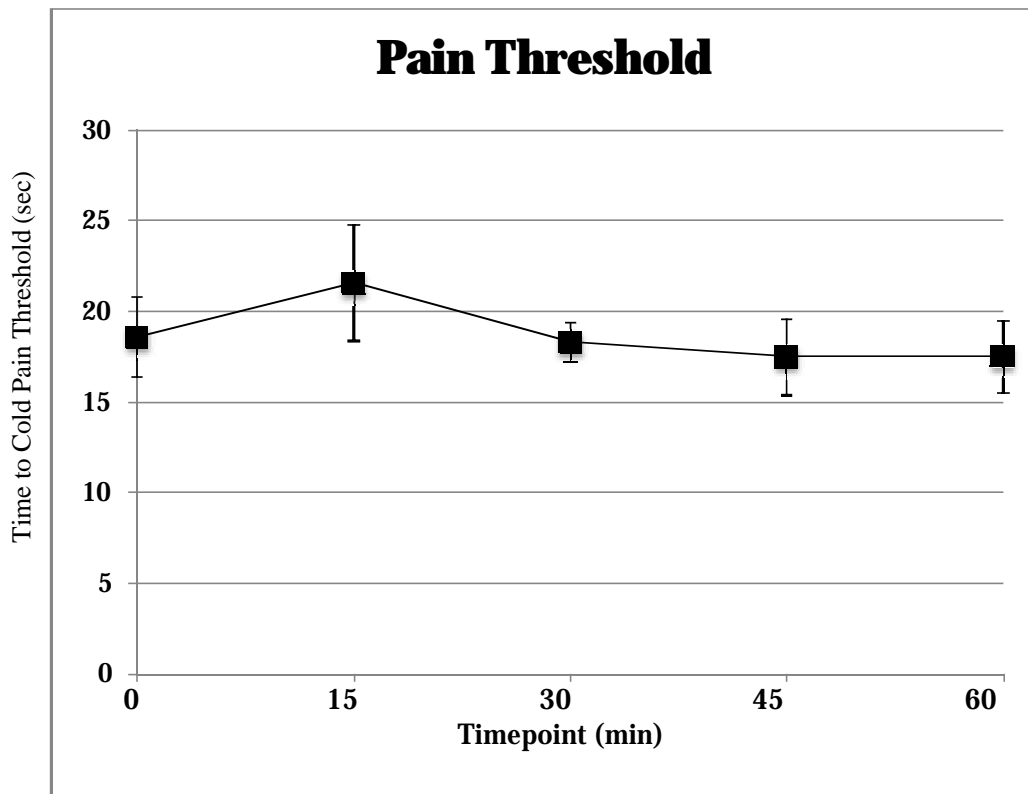
**Table 2. Mean Heart Rate Scores.**

		BLW-OBLA	ABV-OBLA
Baseline	HR	82 ± 5.1	83 ± 6.8
15 min Exercise	HR	144 ± 6.8	157 ± 7.9
30 min Exercise	HR	152 ± 5.9	164 ± 7.4
15 min Recovery	HR	76 ± 7.4	83 ± 7.1
30 min Recovery	HR	69 ± 6.6	82 ± 5.5

### Baseline Trial Pain Assessment

In an effort to establish that multiple CPT assessments separated by 15 min did not influence the temporal summation of subsequent trials, a non-exercising trial was incorporated into the design of the study. A repeated measures ANOVA revealed no differences among the time points for either PT or PI. More specifically, the PT values were  $F=1.195$ ,  $P=0.25$  and the PI values were  $F=1.030$ ,  $P=0.34$ . These values can be found in Figure 2.

**Figure 2. Pain Threshold during resting Cold Pressor Tests.**  
Values are  $M \pm SE$ .



### Exercise Induced Hypoalgesia

Significant differences ( $P=0.001$ ) were found among the ABV-OBLA scores as compared to baseline values. Significant differences ( $P=0.001$ ) were also found among BLW-OBLA scores in comparison to baseline values. Post Hoc analysis revealed that mean PT scores were significantly higher at 30 min into exercise as well as 15 min into recovery for the ABV-OBLA ( $P=0.024$  and  $P=0.03$ , respectively) and BLW-OBLA ( $P=0.02$  and  $P=0.01$ , respectively) trials. Data specific to each time point can be found in Table 3.

**Table 3. Time to Cold Pain Threshold during exercise. P-values are vs. baseline values for Above and Below OBLA. Values are M  $\pm$  SE.**

	Baseline				
	0 min	During Exercise		Post Exercise	
		15 min	30 min	15 min	30 min
ABV-OBLA	13.6 $\pm$ 2.2	21.68 $\pm$ 2.5	34.91 $\pm$ 3.4	31.33 $\pm$ 3.3	19.4 $\pm$ 3.1
p-value		P = 0.41	P = 0.24	P = 0.03	P = 0.22
BLW-OBLA	12.8 $\pm$ 1.8	19.34 $\pm$ 2.2	29.91 $\pm$ 3.2	30.65 $\pm$ 2.7	19.21 $\pm$ 3.4
p-value		P = 0.30	P = 0.02	P = 0.01	P = 0.44

### Exercise Pain Tolerance Comparisons

No significant differences were found when comparing mean PT scores between exercise trials at each time point. Mean and Standard Error PT scores used for comparison at each time point can be found in Table 3.

### Exercise Pain Intensity Comparisons

No significant differences were found when comparing mean PI scores between exercise trials at each time point. Mean and Standard Deviation PI scores used for comparison at each time point can be found in Table 4.

**Table 4. Visual Pain Ratings. Values are M  $\pm$  SE.**

	Time point	Visual Pain Scale
Resting		2.3 $\pm$ 0.2
A-OBLA	0	2 $\pm$ 0.1
	15	1.9 $\pm$ 0.3
	30	2.3 $\pm$ 0.1
	15	2.1 $\pm$ 0.1
	30	2 $\pm$ 0.4
B-OBLA	0	2 $\pm$ 0.2
	15	2.1 $\pm$ 0.3
	30	2.6 $\pm$ 0.1
	15	2.2 $\pm$ 0.3
	30	2 $\pm$ 0.2

## DISCUSSION

The purpose of this investigation was to examine variations in pain perception during and following two bouts of exercise that were 10% above and 10% below the HR corresponding to the HR of OBLA. The primary finding was that both bouts of exercise produced a similar hypoalgesic effect following 30 min of exercise. This hypoalgesic effect persisted for approximately 15 min into recovery. Pain perception was not significantly different between the two exercise trials at any time point. Thus, based upon these data, we can conclude that the threshold for the augmentation of pain perception appears to be below the theoretical anaerobic threshold as indicated by OBLA. Furthermore, total accumulated exercise time appears to be an important factor in the modulation of pain.

The interaction between exercise duration and intensity are believed to both influence the onset of hypoalgesia during and following exercise (26). However, the minimal requisite intensity and duration of exercise needed to produce EIH is still unclear. A number of investigators have studied a variety of exercise protocols in this area. The protocols range from incremental increases in workloads (7, 20,21) to static prescribed workloads (27,13,14), to self-selected exercise intensities (18,38).



Although results from these studies are variable, they indicate that EIH occurs most consistently following relatively high-intensity exercise. Specifically, exercise intensities between 60% and 85% of  $\text{VO}_2$  max appear to produce a hypoalgesic response (14,27,38). In the present investigation, the average workloads employed corresponded to approximately 63% and 83% of the HR associated with OBLA. Thus, both the ABV-OBLA and BLW-OBLA workloads appear to have been sufficient to induce EIH. These findings are in conflict with the work by Drury and associates (8). In their investigation, significant differences were found when comparing the degree of EIH experienced at different workloads within the same submaximal exercise bout. However, the present investigation differs in that we incorporated two separate bouts of exercise at different intensities allowing the subjects to reach a cardiovascular steady state before assessing pain. Furthermore, pain was assessed using a consistent cold stimulus while Drury et al. (8) used a ramping electrodiagnostic pain stimulus.

In addition to the variety of exercise intensities used in the literature, there have also been a number of studies that have examined the effect of exercise duration on EIH. One investigation by Hoffman and associates (16) suggested that the minimal duration of exercise necessary to invoke hypoalgesia was 10 min. However, it appears as though longer durations of exercise are associated with further increases in pain thresholds (26). This is evidenced by several other investigations, including the present study, which have reported reductions in pain threshold following 30 min of exercise (24,34,27). Therefore, it is reasonable to think that total exercise time is likely an important factor in the augmentation of pain perception.

The mechanisms responsible for EIH are poorly understood. Exposure to a painful stimulus has been shown to result in a reduced sensitivity to a subsequent presentation of noxious stimuli; a phenomenon known as stress-induced analgesia (39). Naturally-occurring muscle pain has been studied extensively in numerous exercise-related topics, including the influence of pain on exercise performance (6); the relationship between pain and effort during exercise (17); the influence of demographic, social, and genetic factors on pain perceptions during exercise (5); and the effect of pharmacological manipulations on pain and exertion during exercise (32). While the findings from these and other studies are highly variable, the pain experienced during exercise appears to be a function of the force and frequency of muscle contraction. Specifically, muscle pain thresholds during exercise appear to occur around 50% of maximal exercise intensity (5,6).

Another physiological factor that has received some attention in the literature is the connection between blood pressure and pain perception. An interaction between pain modulation and the cardiovascular system has been previously reported (36). The baroreflex system is an important regulatory mechanism in the short-term control of blood pressure, and it is known to alter central nervous activity by exerting an inhibitory influence on parts of the brain (11,30). This inhibitory effect has been attributed to a decrease in pain sensitivity in humans, making the baroreflex system an important modulator of nociception (9,12,31,36,37). In a previous investigation, we reported a negative correlation between baroreceptor stimulation and the intensity of experienced pain following an orthostatic-induced challenge (41). Exercise has also been shown to elicit systemic pressure changes in the study of pain perception (28,35).

## CONCLUSIONS

Based upon our interpretation of these data, it appears that the OBLA and the corresponding changes in metabolism associated with this phenomenon do not directly impact EIH. Since we observed EIH both above and below the OBLA after 30 min of exercise, it appears that the

accumulation of exercise induced circulating opioids may be responsible for these changes in pain perception. Although beyond the scope of the current investigation, future investigations should consider monitoring opioids in an effort to determine the intensity and duration of exercise needed for pain augmentation.

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