



Effects of Acute Eccentric Exercise Stimulus on Muscle Injury and Adaptation

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ABSTRACT

Frimpong E, Antwi DA, Asare G, Antwi-Boasiako C, Dzudzor B. Effects of Acute Eccentric Exercise Stimulus on Muscle Injury and Adaptation. **JEPonline** 2013;16:(6)18-30. The purpose of this study was to investigate the stimulus of acute eccentric aerobic exercise that would elicit minimal muscle injury but adequate to induce muscle tissue adaptation. Twenty healthy subjects were randomized into two groups: (a) the low stimulus eccentric exercise group (LSEEG); and (b) the high stimulus eccentric exercise group (HSEEG). Both groups performed acute exercise (bout 1) and a repeated exercise (bout 2). In the acute bout, the LSEEG exercised at 50% of heart rate reserve (HRR) for 30 min while the HSEEG exercised at 70% of HRR for 40 min on a treadmill declined at a gradient of 15°. Two weeks after the acute exercise for both groups, the subjects performed a repeated exercise bout at 80% of HRR for 40 min. Creatine kinase (CK) and lactate dehydrogenase (LDH), total white blood cells (TWBC), and perceived muscle soreness (SOR) before and 1, 24, and 48 hrs post-exercise were assessed as markers of muscle injury and adaptation. The results showed that muscle injury was significantly higher in the HSEEG than in the LSEEG in the acute exercises. However, both exercise groups developed similar muscle adaptations with no significant differences in attenuations in CK, LDH, and SOR in the repeated bout. Exercise at 50% of HRR for 30 min may be a threshold stimulus for acute eccentric aerobic exercise.

Key Words: Threshold Stimulus, Creatine Kinase, Perceived Muscle Soreness, Lactate Dehydrogenase

INTRODUCTION

Improvement in exercise capacity of human skeletal muscle to adapt to repeated bouts of physical activity over time results from exercise training (8,11). The major objective in muscle training is to cause physiologic adaptations to improve performance of a given task (1). Acute exercise stimulus (in terms of intensity and duration) and the type of exercise (eccentric exercise vs. concentric exercise) are important in inducing adaptation in skeletal muscles. For example, it has been found that, acute exercise usually results in evidence of skeletal muscle injury that involves eccentric (lengthening) muscle contractions when beginning an exercise program (5,15).

The microtrauma injury to muscle fibers and extracellular matrix is followed by an inflammatory response (6) that appears to be a consequence of both metabolic and mechanical factors (10). Thus, the performance of unaccustomed exercise results in mechanical disruption to the cellular structure of muscle (43) that changes the excitation-contraction coupling system (33). The markers of muscle injury include leakage of myofibrillar proteins such as creatine kinase (CK) and lactate dehydrogenase (LDH) into the blood (13,25), delayed onset of muscle soreness (DOMS) (31,37), muscle swelling and increased circumference of injured muscle, decrease in range of motion (ROM), and decreased muscle strength (26,42).

The performance of only one bout of eccentric exercise that produces muscle injury results in an adaptation such that there is less evidence of injury when the exercise bout is repeated after a week and even up to 6 months with no intervening exercise between the bouts (19). This muscle adaptation to injury (also known as repeated bout effect) is consistently characterized by lower perceptions of soreness, lower strength and performance decrements, and reduced creatine kinase activity relative to the acute or first exercise bout (23,29,32,35,36,42).

Exercise-induced muscle injury (EIMI) is inevitable in sedentary individuals who start an exercise program. Also, individuals in excellent athletic condition may experience muscle injury and soreness when performing exercises that are new to them (24). At the moment little is known about the relationship between muscle injury induced by acute eccentric aerobic exercise and the adaptation or repeated bout effect that occurs afterwards. That is, it is not known whether the more the muscle injury induced from high stimulus exercise, the better the muscle adaptation or that adaptation occurs regardless of the injury induced in the acute exercise.

Moreover, the particular threshold stimulus (in terms of intensity and duration) for which a novel aerobic exercise that is eccentrically biased should be performed to reduce the extent of muscle injury (while inducing adequate adaptation) has not been determined. This is important in exercise prescription and supervision in that an optimal exercise prescription either for competitive training or for fitness improvement is a balance between the intensity, duration, and the type of exercise (44). Furthermore, few previous studies in this area of EIMI have focused on anaerobic exercises on the upper and lower limbs (12,17,39).

Brown et al. (12) observed that similar amounts of protection were afforded to individuals completing 10 maximal voluntary contractions of the knee extensors as was afforded to those completing 50 contractions. Similar results were reported from studies of similar protocols (12,17,39). Hence, the implication is that if individuals perform exercises at different stimuli of same exercise type and have similar adaptations and protection against injury from subsequent exercises, then one would not have to overexert in the acute bout of the exercise.

The purpose of the study was to investigate the stimulus of acute eccentric aerobic exercise that elicited minimal muscle injury and adequately induced muscle tissue adaptation. Thus, the specific objectives of the study were: (1) to assess the effects of low and high stimuli of acute eccentric aerobic exercises on muscle injury; (2) to evaluate muscle adaptation developed from the acute exercises; and (3) to determine whether muscle adaptation depends on serious muscle injury.

METHODS

Subjects

This study consisted of 20 apparently healthy University of Ghana students (14 males and 6 females) of the Korle Bu Campus. The mean age, weight, and height of the subjects were 22.0 ± 1.5 yrs, 62.5 ± 5.9 kg, and 1.7 ± 0.1 m, respectively. None of the subjects had participated in a structured exercise program at least 6 months prior to the study, especially regarding eccentric exercises of the lower extremities such as down-hill running. They had no medical history for which the study's exercises were contraindicated. The research procedures and research design were approved by the Ethical and Protocol Review Committee of University of Ghana Medical School. All subjects gave written informed consent after having understood explanations of the experimental protocol and any potential risks that could be encountered.

Procedures

Exercise Protocol

After completing the Physical Activity Questionnaire (PAQ), the subjects were randomized into two groups. Subjects in each group performed two exercise bouts. Bout 1 consisted of the acute exercise group (Group 1) performing low stimulus eccentric exercises (LSEEG) at 50% of heart rate reserve (HRR) for 30 min. Group 2, the high stimulus eccentric exercise group (HSEEG), performed high stimulus eccentric exercise at 70% of HRR for 40 min. Two weeks later, both LSEEG and HSEEG performed bout 2 exercises of similar eccentrically biased exercises at 80% of HRR for 45 min. The training stimulus in the second bout was necessary for assessing muscle tissue adaptation induced by the acute exercise of bout 1. The 2-wk time interval was allowed for recovery from bout 1. The intensity of the exercise for each group was to determine the target heart rate (THR) calculated as a percentage of the HRR. THR was calculated using the Karvonen method (27). This is given by the relation:

$$\text{THR} = (\text{HR max} - \text{HR rest}) \times (\% \text{Intensity}) + \text{HR rest}$$

Where maximal heart rate (HR max) = $220 - \text{age}$, heart rate reserve (HRR) = $\text{HR max} - \text{HR rest}$, resting heart rate (HR rest) and percentage intensity (%Intensity) calculated as a percentage of the HRR. The target heart rates were determined during the familiarization session 2 days before the down-hill running exercises.

Muscle injury was induced by acute and repeated bouts of down-hill treadmill running. The Xenon treadmill (Okinawa, Japan) was declined at a gradient of 15° . The down-hill slope was obtained by placing a wooden pallet under the rear of the treadmill. This was obtained from angle of declination and the length of the treadmill (21). Heart rate was monitored with a HR monitor, Polar Accurex Plus, Polar Electro Oy, (Kempele, Finland) to ensure that the subjects exercised within the set target HRs. As an adjunct to HR in monitoring exercise intensity, rating of perceived exertion was used (9). The

speed of the treadmill was adjusted to the required target HRs. Blood pressure was measured using a standard mercury sphygmomanometer and stethoscope.

In order to minimize data variability, certain restrictions were placed on the subjects. They were told not to perform any exercise other than the physical activities associated with their activities of daily living. They were also instructed: (a) to abstain from massaging, stretching, and any form of treatment to the lower limbs; and (b) to refrain from taking any non-steroidal anti-inflammatory drugs (NSAIDs), nutritional supplements, alcohol, and caffeine before and during the experimental period.

Blood Sample Collection

Blood samples were collected before and at 1, 24, and 48 hrs after exercise in both the acute and repeated bouts. Five ml of venous blood was drawn from the antecubital vein by venipuncture. Each blood sample drawn was divided into two tubes: (1) one-half was collected into EDTA tubes for full blood count (FBC) analysis; and (2) the other half into Serum Separator Tubes for assays of serum markers of muscle injury. The time for blood sampling was fixed at 7:00 am after an overnight fast. Blood samples in Serum Separator Tubes were centrifuged at 4500 rpm for 10 min and the serum aliquoted into labeled eppendoff tubes for CK and LDH analyses. The samples were stored at a temperature of -20°C until analyses were completed.

Biochemical and Physical Markers of Muscle Injury and Adaptation

The serum CK activity was measured by the VITROS CK Slide method and the VITROS Chemistry Products Calibrator Kit 3 (UK). The serum LDH was measured by the VITROS LDH Slide method and VITROS Chemistry Products Calibrator Kit 3 (UK). Circulating levels of TWBC were measured as inflammatory markers with Sysmex Autoanalyser (Kobe, Japan). Perceived muscle soreness (SOR) was used to assess pain of bilateral quadriceps by Visual Analog Scale (VAS).

Statistical Analyses

The data were analyzed using Microsoft Excel and Statistical Package for Social Sciences (SPSS) version 16. Paired and unpaired *t*-tests were used to compare mean differences within and between the groups, respectively. A *P*-value of less than 0.05 was considered significant. Data are presented as means \pm standard deviation (mean \pm SD).

RESULTS

The serum levels of CK and LDH, TWBC as well as SOR were compared between LSEEG and HSEEG in the acute exercises as markers of muscle injury. The same markers were measured in the repeated bout exercises to assess muscle adaptation. Attenuations in these markers in the repeated bout showed the extent of muscle adaptation developed from the acute exercise-induced injury.

Table 1 shows the results of the characteristics of the subjects. There were no significant differences between LSEEG and HSEEG among the variables: age, height, BMI, SBP, DBP, and HR ($P>0.05$).

Table 1. Characteristics of the Subjects (LSEEG, n = 10; HSEEG, n = 10).

Variables	LSEEG (Mean ± SD)	HSEEG (Mean ± SD)	P-value
Age (yrs)	21.7 ± 1.9	21.1 ± 1.3	0.397
Weight (kg)	61.8 ± 6.3	63.2 ± 5.2	0.581
Height (m)	1.7 ± 0.1	1.7 ± 0.2	0.735
BMI (kg·m ⁻²)	22.4 ± 2.3	22.6 ± 2.1	0.812
SBP (mmHg)	117.6 ± 4.2	117.5 ± 4.4	0.959
DBP (mmHg)	75.5 ± 6.1	76.1 ± 5.0	0.795
HR (beats·min ⁻¹)	73.0 ± 8.3	72.0 ± 6.3	0.633

LSEEG - low stimulus eccentric exercise group, **HSEEG** - high stimulus eccentric exercise group; **HR** - resting heart rate, **BMI** - body mass index, **DBP** - diastolic blood pressure, **SBP** - systolic blood pressure. Data are presented as mean ± SD. The mean differences in age, weight, height, BMI, SBP, DBP, and HR between LSEEG and HSEEG were not significant ($P>0.05$).

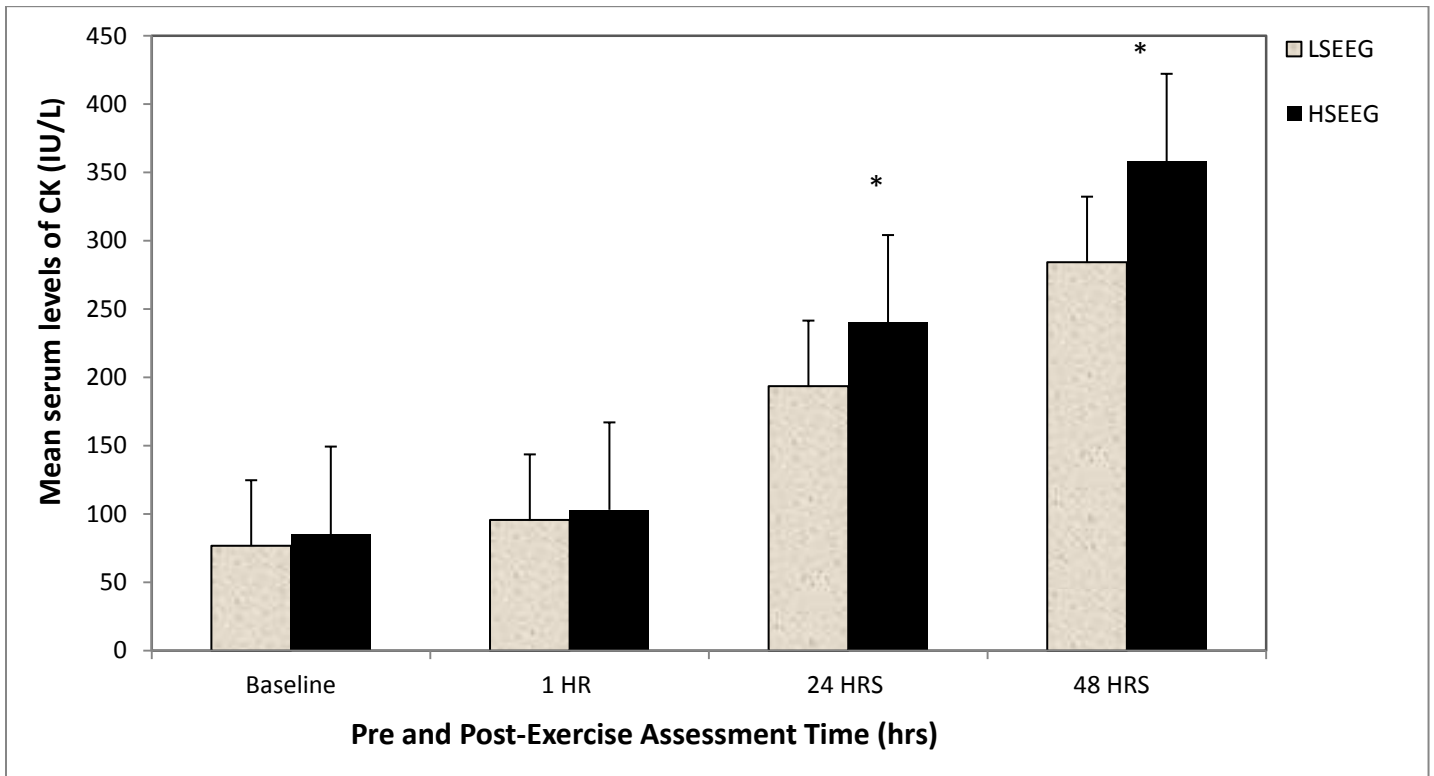


Figure 1: Comparing Mean Serum CK Levels between LSEEG and HSEEG in the Acute Exercises. The mean differences between LSEEG and HSEEG at baseline and 1 hr post-exercise were not significant ($P>0.05$). *Indicates CK significantly higher in HSEEG at 24 and 48 hrs post-exercise ($P=0.00018$; $P=0.0038$, respectively).

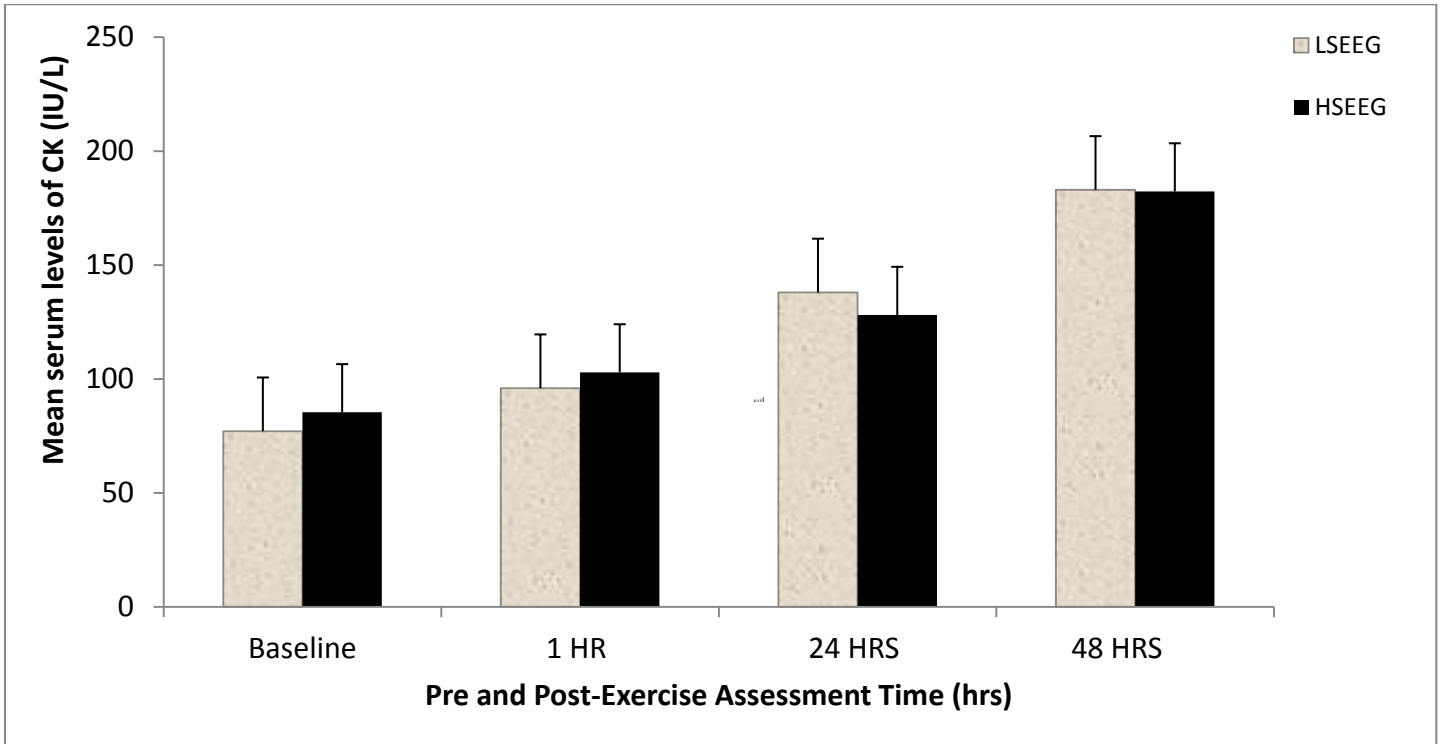


Figure 2: Comparing Mean Serum CK Levels between LSEEG and HSEEG in the Repeated Bout Exercises. There were no significant differences in means between LSEEG and HSEEG at baseline and 1, 24, and 48 hrs post-exercise ($P>0.05$).

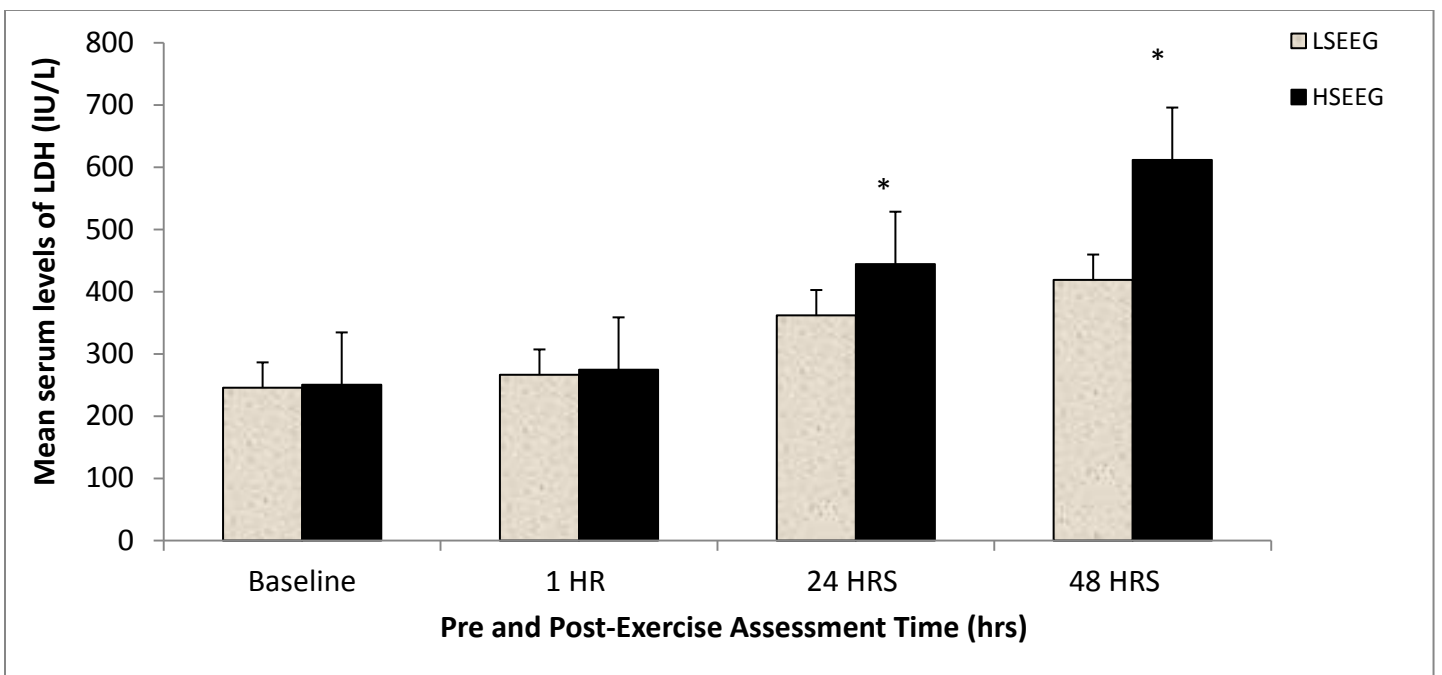


Figure 3: Comparing Mean Serum LDH Levels between LSEEG and HSEEG in the Acute Exercises. The mean differences between LSEEG and HSEEG at baseline and 1 hr post-exercise were not significant ($P>0.05$). *Indicates LDH significantly higher in HSEEG at 24 and 48 hrs post-exercise ($P<0.0001$; $P<0.0001$, respectively).

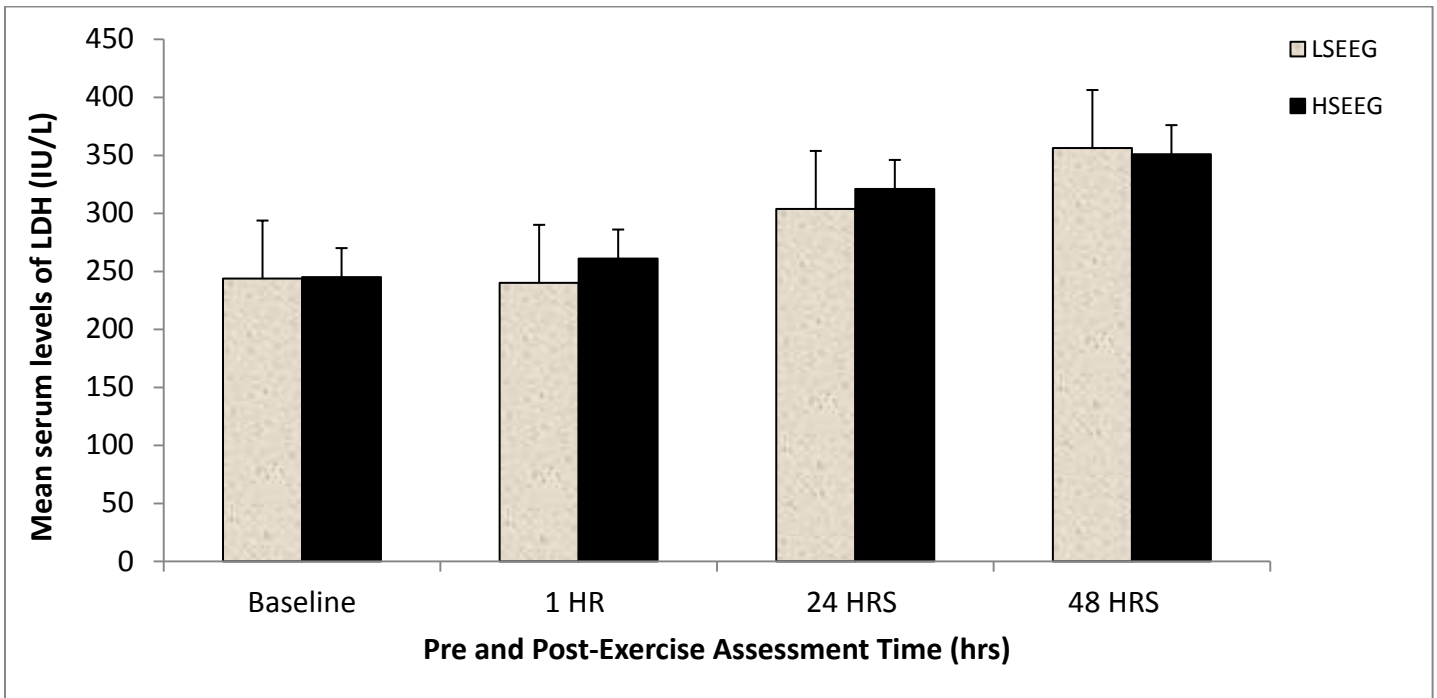


Figure 4: Comparing Mean Serum LDH Levels between LSEEG and HSEEG in the Repeated Bout. There were no significant differences in means between LSEEG and HSEEG at baseline and 1, 24, and 48 hrs post-exercise ($P>0.05$).

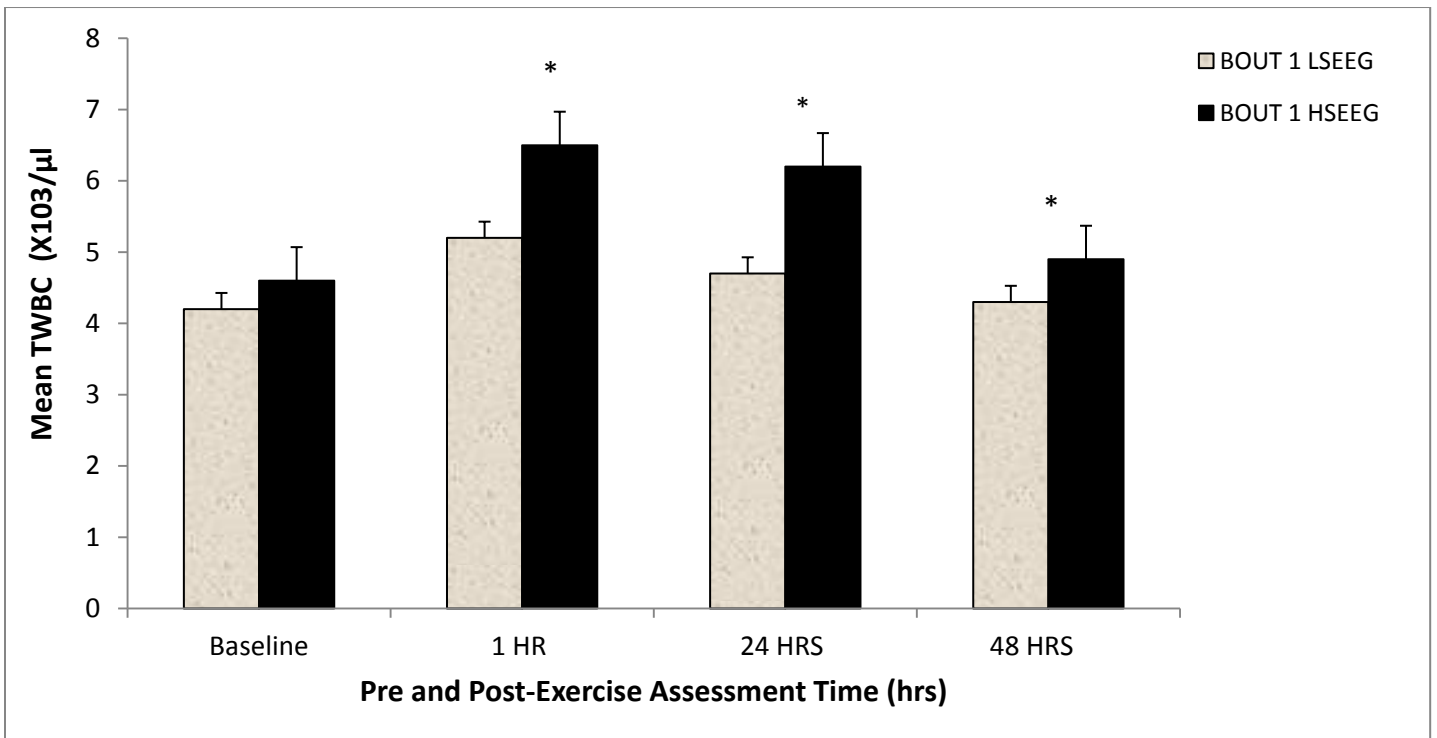


Figure 5: Comparing Mean TWBC between LSEEG and HSEEG in the Acute or Bout Exercises. *Indicates that TWBC was significantly higher in the HSEEG than the LSEEG at 1, 24, and 48 hrs post-exercises ($P<0.05$).

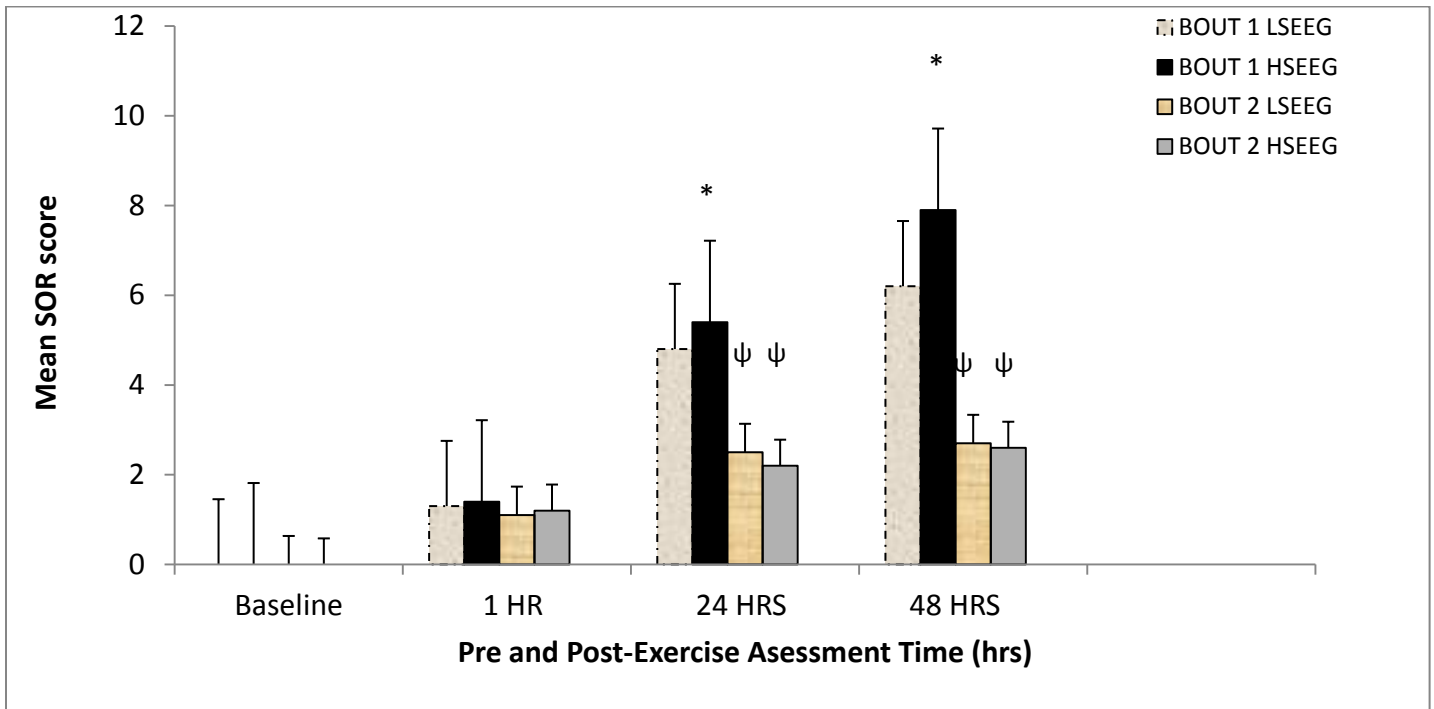


Figure 6: Comparing Mean SOR Score between LSEEG and HSEEG in the Acute (Bout 1) and Repeated Bout (Bout 2) Exercises. *Indicates that SOR was significantly higher in HSEEG at 24 and 48 hrs post-exercise in the acute bout ($P < 0.05$). Ψ Indicates that SOR was significantly lower in bout 2 than in bout 1 exercise in both LSSEG and HSEEG at 24 and 48 hrs post-exercise ($P < 0.05$); however, the difference between them was not significant ($P > 0.05$).

DISCUSSION

Acute exercise is associated with ultrastructural muscle injury that is mainly centered on the Z disk that anchors thin filaments and several intermediate filaments within the sarcomere (31). The injury from the acute exercise induces adaptation in the muscle tissues such that the muscle is more resistant to subsequent strenuous exercise (18). Despite the numerous studies on muscle injury and eccentric exercise, the question of whether low and high stimulus acute eccentric aerobic exercises elicit similar muscle adaptation has not been addressed. Thus, this study was designed to investigate an acute eccentric aerobic exercise stimulus that would induce minimal muscle injury while resulting in adequate skeletal muscle adaptation. The primary finding of this study was that the high stimulus acute eccentric aerobic exercise resulted in higher muscle injury than the low stimulus eccentric exercise. But, interestingly, the muscle tissue adaptation was similar in the repeated bout (bout 2) in both exercise groups.

Muscle Injury

The findings of this study show that muscle injury was generally high in the acute exercises in both LSEEG and HSEEG, but was higher in the HSEEG than the LSEEG. However, evidence of muscle injury was dramatically reduced in both exercise groups in the repeated bout. The results of this study are consistent with previous studies where there was increased evidence of muscle injury in the acute exercise bouts, but dramatic reduction in markers of muscle injury in the repeated bout of eccentric exercises (7,16,19,20,25,28,34). Several theories have been postulated as to the mechanism and why eccentric muscle contractions cause muscle injury. For example, eccentric exercise recruits fewer motor units that increases load per fiber ratio and causes mechanical injury to the muscle (4).

In addition, the initial unequal resting length of the sarcomeres causes more tension on the shorter sarcomeres (33). As a result, there is more stress on the shorter sarcomeres during an eccentric contraction causing them to elongate relatively further than the longer sarcomeres (3). The increased stress on the shorter sarcomeres causes them to “pop” and this popping is what causes the muscle injury (33). Therefore, the higher the amount of eccentric muscle work, the higher the injury induced in the muscle.

Muscle Adaptation

The reduced levels of markers of muscle injury connotes muscle adaptation process in the repeated bout which was stimulated by the acute exercises that made muscle tissues more resistant to injurious or potentially damaging exercises. Brown and colleagues (12) observed that similar amounts of protection were afforded to individuals performing 10 maximal voluntary contractions of the knee extensors as was afforded to those who performed 50 maximal voluntary contractions. The few maximal eccentric contractions resulted in attenuation of symptoms or markers of muscle injury when a higher amount of eccentric muscle work was performed 3 wks later. In a similar study, Clarkson et al. (17) reported that an acute bout of 24 maximal eccentric repetitions reduced plasma CK activity, strength loss, and DOMS when a 70-repetition bout was performed 2 wks later.

Also, Paddon-Jones and Abernethy (39) demonstrated that acute adaptation to low volume eccentric exercise can occur in the absence of significant muscle damage and that exposure to a small number of non-damaging eccentric contractions can significantly improve recovery after a subsequent damaging eccentric bout. The outcome of the present study supports these findings, given that LSEEG and HSEEG developed similar adaptations even though both groups were subjected to different eccentric exercise stimuli. In fact, the present study appears to answer the question as to whether or not the acute exercise bout has to elicit serious symptoms of muscle injury in order to develop protection associated with muscle adaptation. This is because both low and high stimulus groups developed similar muscle adaptation.

The reasons for adaptation have been postulated to be due to changes in motor unit recruitment during the repeated bout that limits the extent of muscle injury and the improvement in neural adaptation in the repeated bout where the muscle becomes more efficient in the recruitment of motor units (19,41). In addition, the increase in number of sarcomeres in series may result in the muscle being more resistant to the damaging effects of muscle lengthening (18). Hence, muscle appears to benefit from the additional sarcomeres in series by increasing contractile velocity and by increasing its extensibility and force produced at longer muscle lengths without sustaining similar subcellular damage for an equal stimulus of exercise (14).

This study demonstrates that acute eccentric aerobic exercises do not have to elicit overt muscle injury before stimulating adaptation in muscle tissues. That is, small or adequate levels of eccentric muscle work are capable of producing the repeated bout effect. Therefore, for acute eccentric aerobic exercise to induce physiologic adaptation to muscle-damaging exercises, a stimulus of 50% of HRR for 30 min may be considered as a threshold stimulus to produce physiological adaptation in muscle tissues.

Since both the low and the high stimulus exercise groups developed similar adaptation, there is no need to overexert in the acute exercise bout to cause muscle tissue adaptation. The exercise stimuli employed in this study were based on the American College of Sports Medicine's (ACSM) Position Stand on developing and maintaining cardiorespiratory fitness (2,3). Starting novel exercises at a low to moderate stimulus is very necessary for gaining improvements in fitness and health (3). While this can be encouraging to exercising at low stimulus exercises because of reduced muscle injury from

the acute exercise and possible improved training effects such as adaptation or repeated bout effect (3), performing high stimulus exercises in the acute bout may be discouraging and inappropriate due to concomitant overt exercise-induced muscle injury.

CONCLUSIONS

This study showed that both low and high stimulus eccentric exercises induced similar muscle adaptations, although muscle injury was higher in the high stimulus eccentric exercise group in the acute exercise. Acute exercises of eccentrically-biased muscle contractions or activities should not necessarily elicit overt or serious muscle injury in order to develop muscle adaptation. Exercise at 50% of HHR for 30 min may be considered as a threshold stimulus for acute eccentric aerobic exercises or eccentrically-biased aerobic exercises. It is recommend that further studies should be done to find out whether or not acute exercises of sedentary individuals should be eccentrically biased in order to develop muscle adaptation against subsequent potentially damaging exercises.

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