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An Investigation of Exercise-Induced Hypoalgesia After Isometric and Cardiovascular Exercise

Abstract

Exercise-induced hypoalgesia is a well-established phenomenon in the literature. The underlying mechanisms responsible for this augmentation of pain perception are not completely understood. The specific mode and intensity of exercise that creates hypoalgesia remains equivocal. Therefore, the purpose of this study was to identify if any differences existed in the exercise-induced hypoalgesia of isometric gripping exercise (IGE) and treadmill exercise (TE). A repeated measures design was used to determine the differences in pain threshold between acute exposure to IGE and TE. Twelve healthy male volunteers served as our subjects. Subjects were tested on three different days under three different conditions (rest, IGE, TE). The order of the trials was randomized and applied force (AF) was used as the dependent variable. Applied force pain threshold (AFPT) was determined by a handheld dolorimeter used to apply progressive force and pain to the skin and muscles of the wrist flexors before and after exercise. Exercise induced hypoalgesia was found in both exercise conditions by comparing resting PPT values (6.23 ± 2.04) to those measured immediately after IGE (7.24 ± 1.61 ; $p = 0.0058$) or TE (8.03 ± 2.03 ; $p = 0.0001$). However, TE produced a larger (22.04 %) hypoanalgesic effect in comparison to isometric exercise (14.14 %). Both TE and IGE may have potential as methods of increasing one's pressure pain threshold. Further investigation into the specific causes of exercise-induced hypoalgesia is warranted.

Keywords

Hypoalgesia, Anaerobic Exercise, Aerobic Exercise, Pain Threshold

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AN INVESTIGATION OF EXERCISE-INDUCED HYPOALGESIA AFTER ISOMETRIC
AND CARDIOVASCULAR EXERCISE

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ABSTRACT

AN INVESTIGATION OF EXERCISE-INDUCED HYPOALGESIA AFTER ISOMETRIC AND CARDIOVASCULAR EXERCISE. **Daniel G. Drury, Kristin Stuempfle, Robin Shannon, James Miller.** **JEPonline.** 2004;7(4):1-5. Exercise-induced hypoalgesia is a well-established phenomenon in the literature. The underlying mechanisms responsible for this augmentation of pain perception are not completely understood. The specific mode and intensity of exercise that creates hypoalgesia remains equivocal. Therefore, the purpose of this study was to identify if any differences existed in the exercise-induced hypoalgesia of isometric gripping exercise (IGE) and treadmill exercise (TE). A repeated measures design was used to determine the differences in pain threshold between acute exposure to IGE and TE. Twelve healthy male volunteers served as our subjects. Subjects were tested on three different days under three different conditions (rest, IGE, TE). The order of the trials was randomized and applied force (AF) was used as the dependent variable. Applied force pain threshold (AFPT) was determined by a handheld dolorimeter used to apply progressive force and pain to the skin and muscles of the wrist flexors before and after exercise. Exercise induced hypoalgesia was found in both exercise conditions by comparing resting PPT values (6.23 ± 2.04) to those measured immediately after IGE (7.24 ± 1.61 ; $p = 0.0058$) or TE (8.03 ± 2.03 ; $p = 0.0001$). However, TE produced a larger (22.04 %) hypoanalgesic effect in comparison to isometric exercise (14.14 %). Both TE and IGE may have potential as methods of increasing one's pressure pain threshold. Further investigation into the specific causes of exercise-induced hypoalgesia is warranted.

Key Words: Hypoalgesia, Anaerobic Exercise, Aerobic Exercise, Pain Threshold

INTRODUCTION

Exercise-induced hypoalgesia is a phenomenon that has been observed repeatedly in humans (1-5) and in animals (6,7). Although the exact mechanism responsible for this augmentation of pain perception to exercise has not been determined (7), numerous researchers have produced a hypoalgesic response in a variety of exercise conditions (1,5,8-10). Unfortunately, the methodological diversity of this relatively small body of

literature has left major gaps in our understanding of what potentially could be an effective therapeutic intervention (7,10).

The nociceptive nerve endings that are found in our skin, joints, muscles, bone, and internal organs comprise an intricate network of pain perception controlled by our central nervous system. Pain comes in many forms and therefore we have a variety of nociceptors that can detect different types of stimuli (8). Since muscle afferent nerve pathways and pain afferent nerve pathways both converge on the dorsal horn of the spinal cord, some researchers have hypothesized that pain afferents may be susceptible to some form of selective inhibition (11). Others have hypothesized that endogenous opioid activation may be involved in exercise-induced hypoalgesia (2). Most likely, exercised-induced hypoalgesia is caused by a host of interrelated factors that may be partially controlled by the mode and intensity of exercise (7).

Numerous researchers have reported hypoalgesia as a result of both isometric (1,11) and sub-maximal cardiovascular exercise conditions (5,10,12). However, we are unaware of any studies that have compared these modes of exercise within the same study while using applied force pain threshold as the dependent variable. Therefore, the purpose of this investigation was to determine if differences exist in the applied force pain threshold immediately after acute bouts of isometric or cardiovascular exercise.

MATERIALS AND METHODS

Twelve apparently healthy males were recruited for this study (age 20.50 ± 0.91). After reading and signing a document of informed consent, each subject completed a health history questionnaire and exercise activity survey. All documents and procedures were approved by the Institutional Review Board prior to data collection. Once admitted into the study, subjects had their height and weight measured using a stadiometer and a standard balance beam scale (Detecto, Webb City, MO). Body composition was then determined using Lange skin fold calipers (Beta Technology Corp., Cambridge, MD) and a three site formula previously described by Jackson and Pollock (13).

Each subject was required to visit the laboratory three separate times to determine his applied force pain threshold under three different conditions: rest, treadmill exercise (TE), and isometric gripping exercise (IGE). The order of these conditions was randomized. The pain threshold test was conducted with the subject seated with his dominant arm resting flat and extended on the prep-table in a supinated position. Prior to data collection, the subject was prepared by marking a small 1 cm circle on the muscle belly of the wrist flexors. Although this site varied slightly from subject to subject, the circle was made approximately 5-7 cm distally of the anticubital space and 1-2 cm medially from the centerline of the anterior forearm. This site was used for the administration of applied force and pain via the dolorimeter and (Baseline-EFFEGI, Italy). The hand-held dolorimeter was pressed into the muscle belly at a rate of approximately 1 kg of applied force per second, during which the arm was stabilized by a member of the research team. The subject was instructed to verbally indicate when the increasing applied pressure of the probe turned to pain.

This procedure was repeated 2 more times approximately 1 cm above and below the original marking and a mean of these three applied pressure measurements was used for analysis. The resultant 'applied force pain threshold' (AFPT) (kg) was retrieved from the dolorimeter and was expressed in units of applied force as the dependent variable for analysis.

Table 1. Bruce treadmill protocol.

Time (min:s)	Stage	Speed		Slope	METs
		(mi/hr)	(km/hr)	(%)	
0:00-3:00	1	1.7	2.7	10	5
3:00-6:00	2	2.5	4.0	12	7
6:00-9:00	3	3.4	5.5	14	10
9:00-12:00	4	4.2	6.8	16	13
12:00-15:00	5	5.0	8.0	18	16
15:00-18:00	6	5.5	8.0	18	16
18:00-21:00	7	6.0	9.7	22	22

*Adapted from Bruce RA & McDonough JR. Stress testing in screening for cardiovascular disease. Bulletin of New York Academy of Medicine, 1969;45:1288.

Condition 'A'- Rest

For the resting condition, the subject was asked to sit quietly in the laboratory for 7 minutes. AFPT scores were obtained 30 s after the rest period.

Condition 'B'- Isometric Exercise

Intermittent isometric contractions of the wrist flexors were achieved using a handheld isometric spring dynamometer (Lafayette Instrument Co., Lafayette IN). The subject was asked to squeeze maximally on the dynamometer every 2 s for one minute. The pace of activity was regulated using a metronome (Franz Electronic Metronome, New Haven, Conn). AFPT scores were determined 30 s after the last isometric contraction.

Condition 'C'- Treadmill Exercise

Subjects exercised on a treadmill using the Bruce protocol (Table 1). Subjects walked and/or ran on the treadmill until their heart rate (Polar Heart Rate Monitor) reached 65-75 % of their pre-determined heart rate reserve. The Karvonen method of determining the heart rate reserve was used to establish exercise intensity (14). This intensity has produced hypoanalgesic effects in several investigations (8). Once this level of activity was established, the subject was asked to continue exercise at that speed for seven additional minutes. AFPT scores were taken 30 s after exercise.

Statistical Analyses

Differences among the three conditions were determined using a repeated measures ANOVA. Post Hoc analysis of the ANOVA was achieved with a Scheffe test of mean differences. The a priori level of significance was set at $p \leq 0.05$. All data are reported as Mean \pm SD.

RESULTS

Anthropometric data for the subject's height, weight and % fat were 182.8 ± 4.1 cm, 85.30 ± 9.19 kg, and 16.54 ± 4.36 %, respectively. Differences among AFPT scores were found across all three conditions ($p = 0.0001$). These values represent an increase in pressure pain threshold of 22.04% (TE) and 14.14% (IGE) as compared to the resting scores. Post Hoc analysis revealed that the TE AFPT scores were significantly higher than both the IGE condition ($p = 0.034$) and the rest condition ($p = 0.0001$). In addition, IGE condition was significantly higher than the rest condition ($p = 0.0058$). A graphic depiction of these scores can be found in Figure 1.

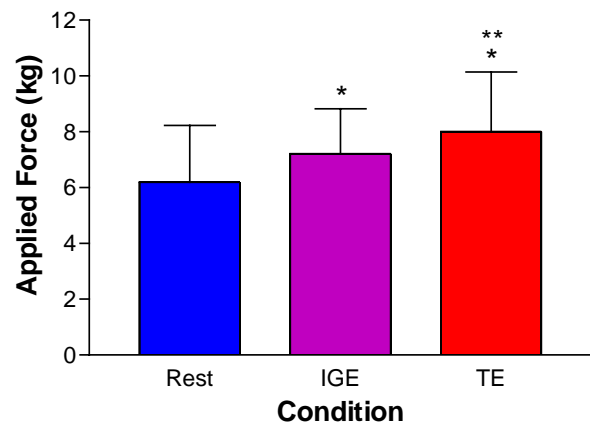


Figure 1. Applied force pain scores under three conditions.
* = < 0.05 from Rest; ** = < 0.05 from Rest and IGE

DISCUSSION AND CONCLUSIONS

The primary finding of this investigation is that sub-maximal TE produced a greater increase in AFPS as compared to IGE. More specifically, repeated isometric contractions of the muscles under the test administration site did not improve the pressure pain threshold as much as TE. This finding suggests that systemic alterations that accompany sub-maximal large muscle group exercise seem to have a greater influence on exercise-induced hypoalgesia in comparison to the local and systemic effects of repeated isometric contractions of muscles prior to the administration of applied force and pain.

Previous investigations of isometric exercise and its effects on exercise-induced hypoalgesia have been equivocal (15). Koltyn and coworkers (15) tested 15 males and 16 females under two isometric exercise conditions. Subjects squeezed a hand dynamometer for two minutes at two different intensities (40-50% & 100% of MVC). These researchers concluded that both men and women experienced hypoalgesia after maximal isometric gripping exercise, but the men did not experience effects after the sub-maximal condition. Our

findings are in agreement with those of Koltyn (15) in that our subjects also experienced a decrease in pain sensitivity after maximal isometric contractions. Although our subjects completed intermittent maximal isometric contractions for only one minute and our method of introducing pain was different, the resultant decrease in pain sensitivity was similar.

In a similar study by Kosek and Ekholm (11), subjects were asked to maintain a sub-maximal (21% of MVC) isometric contraction of the quadriceps until exhaustion. After finding an increase in pressure pain thresholds both during and after exercise, these researchers have hypothesized that the sympathetic response to isometric exercise may trigger a concomitant release of adrenal enkaphalins (11). Indeed, others have reported a link between exercise induced catecholamine release and increases in circulating hormones including beta-endorphins, and adrenocorticotropin (2,4,16). With regards to the current investigation, the sympathetic response of the weight-bearing TE may have produced a greater hormonal response as compared to the response caused by the IGE. Although isometric handgrip has been used extensively by researchers as a method of inducing sympathetic activity, the amount of muscle mass and metabolic activity involved during isometric gripping is far less than the metabolic activity required for dynamic exercise at 65-75% of one's target heart rate reserve. Because both exercise conditions produced a hypoanalgesic effect and the size of the response was different between the conditions, the hypoalgesia we found may be the result of varying degrees of the same physiological response dictated by the relative amount of tissue required for each activity. Other investigators have reported that exercise-induced hypoalgesia has correlated well with several exercise related variables such as heart rate (2,12), blood pressure (2,12) and exercise intensity (4).

An investigation very similar to the one reported here was conducted by Paalasmaa and coworkers (17) on both isometric (70% of MVC for 2 minutes) and progressive cycling exercise (100, 150, 200, 250 Watts) of the lower body. These researchers measured skin sensitivity through the administration of thermal and electrical stimuli. The results of this investigation revealed that isometric exercise did not alter heat pain thresholds or sensitivity to tactile stimuli, while the cycling exercise produced intensity dependent attenuation of both heat and tactile stimuli (17). Investigations of exercise induced hypoalgesia that have used thermal sensitivity as a dependent variable have been criticized due to the confounding element of metabolic heat production that is inherent with physical exertion (7). In the current investigation, we induced pain through applied pressure and therefore avoided this methodological pitfall. However, our findings were similar in that cardiovascular exercise was identified in both studies as being a superior mode for creating hypoalgesia.

In summary, the existence of exercise-induced hypoalgesia is well established in the literature (7). However, the underlying mechanisms at work remain theoretical and un-established. The data presented here provide some insight into the conditions in which exercise can produce a decrease in pain pressure threshold in young men. Future investigations in this area should consider a dose-response model to determine if exercise intensity is indeed correlated with hypoalgesia or if a critical intensity threshold must be reached to experience the effects. Researchers also may want to consider attempting to identify the specific hormonal markers associated with the exercise related sympathetic release of hormones that may affect adrenal enkaphalins and consequently pain perception.

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