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# Whole Body Periodic Acceleration Reduces Levels of Delayed Onset Muscle Soreness After Eccentric Exercise

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# WHOLE BODY PERIODIC ACCELERATION REDUCES LEVELS OF DELAYED ONSET MUSCLE SORENESS AFTER ECCENTRIC EXERCISE

By

Daniel H. Serravite

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Several recovery strategies have been used, with limited effectiveness, to reduce the muscle discomfort or pain and the diminished muscle performance following a bout of unaccustomed physical activity, a condition known as delayed onset of muscle soreness (DOMS). Muscle damage in this condition is associated with mechanical disruption of the muscle and connective tissue and inflammation and increased oxidative stress. Low frequency, low intensity, whole body periodic acceleration (WBPA) that increases nitric oxide (NO) release from vascular endothelium into the circulation through increased pulsatile shear stress offers a potential solution. This is because endothelial derived nitric oxide has anti-inflammatory, antioxidant and anti-nociceptive properties. The purpose of this study was to examine the effects of WBPA on the pain and diminished muscle performance associated with DOMS induced by unaccustomed eccentric arm exercise in young male subjects. Seventeen active men, 23.4 ± 4.6 yr of age, made six visits to the research facility over a two-week period. On day one, the subject performed a 1RM elbow flexion test and was then randomly assigned to the WBPA or control group. Criterion measurements were taken on Day 2, prior to and immediately following

performance of the eccentric exercise protocol (10 sets of 10 repetitions using 120% of 1RM) and after the recovery period. During all subsequent sessions (24, 48, 72, and 96 h) these data were collected before the WBPA or passive recovery was provided. Variables including isometric strength (MVC), blood markers (CPK, MYO, IL-6, TNF-α and Uric Acid), soreness, pain, circumference, and range of motion (ROM) were examined in their relation to the recovery protocol. Significantly higher MVC values were seen for the WBPA group across the entire 96 h recovery period. Additionally, within group differences were seen in CPK, MYO, IL-6, soreness, pain, circumference, and ROM showing a smaller impact and more rapid recovery by the WBPA group. It was concluded the application of WBPA hastens recovery from DOMS after eccentric exercise. Given the lack of other potential mechanisms, these effects appear to be mediated by the increased NO release with WBPA.

## DEDICATION

I dedicate this dissertation to my sons, Luca and Nico, and my wife Jenny who supported me throughout the Ph.D. journey.

Thank you to my parents Humberto and Sara who gave up everything to provide me the best education and always supported me to pursue my dreams.

## ACKNOWLEDGEMENT

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# CHAPTER 1: INTRODUCTION

A delayed perception of skeletal muscle discomfort or pain that is termed delayed onset of muscle soreness (DOMS) is commonly experienced following a bout of unaccustomed physical activity (4, 14, 27). DOMS is usually associated with soreness, decreased strength, localized swelling, stiffness, and reduced range of motion (4, 14, 27). The soreness from DOMS usually follows an inverted U-shape curve over time in which the level of discomfort increases during the first 24 hours following the cessation of exercise, peaks between 24 to 72 hours, then subsides and eventually disappears by 5-7 days post-exercise (10, 13, 39). Further, the contour of the inverted U-shape may vary depending on the intensity, volume, and type of activity inducing DOMS (73). The decrement in muscle performance can parallel the course of the soreness or be independent of it (14). Eccentric exercises are the preferred procedure for evoking DOMS since they produce greater torque, causing greater reductions in strength (74) and generating more muscle damage than concentric or isometric contractions (48). This is especially true in sedentary or novice recreational athletes (49) performing unaccustomed eccentric exercises using small muscles, such as arm muscles (35), at high movement velocities (12).

A number of different mechanisms have been used to explain DOMS, and therefore, several methods have been employed to counteract the soreness of DOMS with varying degrees of success. These include ice water immersion, stretching, anti-inflammatory drugs, ultrasound, transcutaneous electric nerve stimulation (TENS), massage compression garments, acupuncture, hyperbaric oxygen therapy, oral antioxidants such as vitamins C and E, exercise (8, 14, 17, 21, 31, 33, 45). Whole body vibration (WBV) has been shown to reduce calf and gluteal pain and decrease

inflammation after downhill running (9) and when combined with stretching (58) produces a decrease in pain that ranges from 22-61% compared to stretching alone. These positive effects may be explained by the enhanced local blood flow immediately after vibration training (36) and/or by the potentiation of pain inhibition through increased proprioceptive feedback (41). These studies, however, did not measure recovery of performance.

Whole body periodic acceleration (WBPA), which produces low frequency, low intensity vibrations, increases nitric oxide (NO) release from vascular endothelium into the circulation through increased pulsatile shear stress. The latter is produced by the addition of small pulses superimposed upon the natural pulse; the number of pulses is a function of platform frequency (3, 34, 71). Since endothelial derived nitric oxide has anti-inflammatory, antioxidant and anti-nociceptive properties (1, 23, 28, 42), the purpose of this study is to determine whether WBPA could hasten the recovery from DOMS resulting from exposure of a sample of fit young volunteers to strenuous eccentric exercise by the elbow flexors.

# CHAPTER 2: METHODS

## Subjects

Seventeen healthy young men volunteered for this study. Their mean (± SD) age, height, and body weight was 23.4 ± 4.6 years, 176.0 ± 6.2 cm, and 79.1 ± 11.4 kg, respectively. A power analyses performed using muscle CPK as the dependent variable following massage indicated that ten subjects were required ($F(5, 10) = 3.32$, $p = 0.05$) (75). All volunteers provided written informed consent prior to participation in the study. The investigation was approved by the University of Miami's Institutional Review Board and conducted in conformity to the Declaration of Helsinki for Medical Research involving Human Subjects. Individuals who reported participation in competitive sports in the prior 12 months; cardiovascular, endocrine, or neuromuscular disorders; orthopedic problems that would limit or be aggravated by either isoinertial arm curl exercise or the performance of a standard isometric test; or other chronic medical conditions that might affect performance were excluded from participating in the study. In addition, subjects taking anti-inflammatory agents, nutritional supplements, or any other medications that could affect neuromuscular performance, as well as L-Arginine supplements, were not allowed to participate in this study. Subjects were required to avoid any formal physical activity, such as strength or endurance training, for 48 h before the initiation of the study and throughout its duration.

## Experimental Design and Procedures

Figure 1 presents a schematic of the study protocol. Participants were randomly assigned to a control or WBPA condition. For 2 weeks prior to data collection, and during the protocol period, subjects were instructed to continue their normal eating

habits, but to refrain from the use of dietary supplementation. Subjects were also instructed to abstain from exercise for 48 h before and for the duration of the study. Criterion measurements of pain, anthropometry, ROM, soreness, blood-borne markers, and isometric strength were taken at baseline and immediately, 24 h, 48 h, 72 h and 96 h after performing the lifting protocol.

On day 1, the participant completed a health status questionnaire (HSQ) to confirm eligibility. Upon acceptance into the study he completed the international physical activity questionnaire (19). A maximum 1-repetition strength test (1RM), performed using the National Strength and Conditioning Association protocol, was used to determine the maximum amount of weight that the participant could lift in a single repetition during an elbow flexion exercise using the dominant arm (5). The day concluded with the participant being familiarized with the lifting and WBPA protocols. The time required for the visit was approximately one hour. On day 2, prior to the participant's arrival, he was randomly assigned to the control or WBPA group. Criterion measurements were made before he performed the lifting protocol and after the treatment (WBPA or passive recovery). On days 3 through 5 criterion measurements were made first, followed by the treatment. During the last visit, on day 6, only the criterion measurements were made with no further treatment.

**Exercise Protocol**

After performing the criterion measurement on day 2, the participant performed the lifting protocol consisting of 10 sets of 10 lengthening muscle contractions using a dumbbell loaded at 120% of his dominant elbow flexor 1 RM. The lifts were performed on a standard seated arm curl (preacher's) bench. The dumbbell was lowered from 75°

flexion to 180° (full extension) at a rate of 5 seconds per repetition controlled by the participant matching the sound of a metronome. When a participant showed difficulty in controlling the movement velocity, minimal spotting was provided by the investigator. After each eccentric movement, the participant had a 2s pause while the researcher lifted the dumbbell to the starting position to prepare the participant for the next repetition. The exercise protocol was followed by 30 min of passive recovery. Subsequent to the 30 min recovery, the participant underwent a 45-minute WBPA or a passive recovery bout depending on his group assignment.

**Whole Body Periodic Acceleration Protocol**

WBPA was provided using a motion platform that has the appearance of a single size bed with a mattress. The platform is driven by a digitally controlled servo motor assembly (Exer-Rest®, Non-Invasive Monitoring Systems, Inc) (see Figure 2). This apparatus has a handheld wireless controller that regulates its speed in cycles per minute, travel distance in mm and time of the treatment in minutes. The participant lies supine on the mattress and is coupled to the motion platform via sandals connected to a footboard. The platform moves 16 mm in a repetitive sinusoidal head-to-foot direction at 140 times per minute, thereby applying approximately ±0.22 g to the participant for the 45-minute WBPA period. These settings have been shown to release NO into the circulation of healthy adults and patients with inflammatory diseases (59).

**Criterion Measurements**

*Muscle Strength*

Each subject performed a unilateral isometric maximal voluntary contraction (MVC) using his dominant arm. During the testing, the elbow was positioned at two

separate angles, 90° (MVC90) and 150° (MVC150). Data were collected using a digital force gauge (Chatillon, DFS Series, Ametek, FL). The MVC testing protocol began with a warm-up consisting of two submaximal contractions (80% of perceived maximum) and one maximal contraction. The participant was then instructed to apply maximal force by pulling the handle of a load cell unit towards his body for five seconds. Strong verbal encouragement was provided by the investigator throughout the strength testing. Three maximal contractions were performed with a 2-minute rest period between trials. The peak force was recorded for each trial. The trial producing the highest force value was considered the MVC.

*Blood Measurements.*

Approximately 20 ml of whole blood were collected from the antecubital vein using a standard venipuncture technique at baseline and immediately, 24 h, 48 h, 72 h and 96 h after performing the lifting protocol. The 24 h, 48 h, 72 h and 96 h samples were taken prior to the WBPA or passive recovery sessions. The blood was collected into serum tubes and centrifuged for 15 min to obtain serum. Samples were stored at −80°C until analyzed for serum creatine kinase (CPK), myoglobin (MYO), IL-6 and TNF-α using a microplate reader (Thermo Multiskan Spectrum, Vantaa, Finland).

*Assay Procedure- CPK*: CPK was quantified using an EnzyChrom Creatine Kinase Assay Kit (ECPK-100; Bioassay Systems, CA). Each reaction well contained a mixture of 10 µL Substrate Solution, 100 µL Assay Buffer, and 1 µL Enzyme Mix. Subsequently, 10 µL samples were transferred into each well and 100 µL of reconstituted reagent were added. The contents of the plate were then mixed by tapping. The reaction was incubated at 37°C. CPK is fully activated within 10 min by glutathione provided in

the Substrate Solution. An OD340 nm reading was done at 10 min and again at 40 min. To calculate a sample's CPK activity, the following equation was used,

$$CK\ (U/L) = \frac{OD_{40min} - OD_{10min}}{OD_{CALIBRATOR} - OD_{H2O}} \times 100$$

Where $OD_{40min}$ and $OD_{10min}$ are OD340nm values at 40 min and 10 min for the sample; $OD_{CALIBRATOR}$ and $OD_{H2O}$ are OD340nm values of the Calibrator and water blank at 40 min.

*Assay Procedure- MYO.* Myoglobin concentration was determined using the MP Biomedicals myoglobin enzyme immunoassay ELISA kit (MP Biomedicals, NY). The Myoglobin ELISA test is a solid phase enzyme-linked immunosorbent assay. The assay system utilizes a unique monoclonal antibody directed against a distinct antigenic determinant on the myoglobin molecule. Mouse monoclonal anti-myoglobin antibody is used for solid phase immobilization on the microtiter wells and the antibody-enzyme (horseradish peroxidase) conjugate solution contains a goat anti-myoglobin antibody. The test sample was allowed to react simultaneously with the two antibodies resulting in the myoglobin molecules being sandwiched between the solid phase and enzyme-linked antibodies. After 45-min incubation at room temperature, the wells were washed with water to remove unbound labeled antibodies. A tetramethyl-benzidine (TMB) Reagent was then added and the sample was incubated for an additional 20 min resulting in the development of a blue color. The color development was stopped with the addition of a stop solution which changed the color to yellow. Absorbance was measured spectrophotometrically at 450 nm in a microplate reader.

*Assay Procedure- Il-6.* After bringing all reagents and samples to room temperature, 50 µl of Assay Diluent RD1-63 followed by 100 µL of standard, control, or

sample (previously 2-fold diluted) were added to each microplate. The plate was then covered and incubated for 2 h at room temperature. After incubation, the plate was aspirated and each well was washed 5 times with 400 µL of wash buffer. The last wash was aspirated and the plate was blotted using a clean paper towel. Then 200 µL of diluted porcine IL-6 conjugate was added to each well. The plate was covered with an adhesive strip and incubated for 2 h at room temperature and the washing procedure was repeated. Next, 120 µL of substrate solution was added to each well and incubated for 30 min at room temperature in a light-protected environment. Finally, 120 µL of stop solution were added to each well and the plate was gently tapped to ensure thorough mixing. The optical density was set 450 nm in a microplate reader.

*Assay Procedure- TNF-α.* The initial steps in the TNF-α assay were the same as described for the IL-6 assay. After aspiration and blotting with a clean paper towel, 100 µL of porcine TNF-α conjugate was added to each well. The plate was covered with a new adhesive strip and incubated for 2 h at room temperature. The washing procedure was then repeated. Next 100 µL of substrate solution was added to each well and the plates were incubated for 30 min at room temperature while protected from light. After this period, 100 µL of stop solution was added to each well and all wells were thoroughly mixed by gently tapping the plate. The optical density of the microplate reader was set at 450 nm.

*Muscle Soreness*

Muscle soreness was evaluated using a 100 mm visual analogue scale (VAS), with 0 mm indicating “no pain at all”, while 100 mm indicated “worst pain possible”. Subjects were asked to draw a vertical line on the scale representing the current intensity

of their pain under three different conditions; resting, after a palpating test, and during passive flexion and extension. With the subject in a seated position and the exercised arm resting on a table, the investigator palpated the biceps brachii at three sites: mid-belly and 3 cm above and below the mid belly, by applying firm pressure using the index and middle fingers. The participant was then asked to rate his level of soreness as the researcher extended and flexed his elbow joint while the participant relaxed his arm. All the soreness measurements were performed by the same investigator.

*McGill Pain Questionnaire*

Muscle pain was assessed using the short-form of the McGill pain questionnaire (MPQ) (44). The short-form McGill pain questionnaire (SF-MPQ) contains 15 major descriptors (11 sensory; 4 affective) which are rated on an intensity scale from 0-3 (0 = none, 1 = mild, 2 = moderate and 3 = severe). Three pain scores were derived from the sum of the intensity rank values of the words chosen for sensory, affective, and total descriptors.

*Upper Arm Circumference*

Anthropometric measurements were made according to the International Society of Advancement in Kynanthropometry (ISAK). Upper arm circumference was assessed using a constant tension tape while the arm was maintained in a relaxed and hanging position at the participant's side. The measurement was taken at a point equidistant between the superior and lateral border of the acromion process and the proximal and lateral border of the head of the radius.

*Range of Motion (ROM)*

Passive range of motion (PROM) of the elbow joint was evaluated using a plastic goniometer while the participant lay supine on an examination table. ROM was defined as the difference between the extended and flexed angles. The lateral epicondyle of the humerus, the proximal apex of the deltoid muscle, and the styloid process of the radius were used as landmarks to measure elbow joint angles. The participant's arm was passively flexed and extended using a very low angular velocity from a full flexed position towards a full extension position.

**Statistical Procedures**

The responses of the dependent variables (VAS, pain, strength, ROM, circumference, CPK, MYO, IL-6 and TNF-$\alpha$) before and after exercise (immediately after, 24 h, 48 h, 72 h and 96 h after) were compared between conditions. A series of 6 (time point) x 2 (treatment group) repeated measure ANOVA were performed. If significant differences or interactions were found, LSD *post hoc* analyses were used to determine the sources of these differences. Analyses were performed using the statistical software package SPSS (version 17.0), with a significance level set *a priori* at $p < 0.05$.

Data from subjects who dropped out of the study were not included in the analyses unless one full trial was completed. Also, if a subject did not remain inactive during the testing period his data were not included in the analysis. All instruments were calibrated prior to each use. To increase intra- and inter-rater reliability, every attempt was made to use the same examiner for each test and a detailed examiner protocol with systematic instructions was provided and followed for each test throughout the investigative period.

## CHAPTER 3: RESULTS

No significant differences were found between the WBPA and control groups in physical characteristics or for baseline values for any of the criterion measures. Data are presented as means ± SD relative to pre-intervention values unless otherwise stated.

### Muscle Strength

Significant differences were seen between the WBPA and control groups in MVC90 and MVC150 from post test through 96 h ($p < 0.05$). The patterns of change in MVC90 and MVC150 for the WBPA and control groups also differed across the 96 h recovery; however, the patterns of change for both strength measures were consistent within experimental groups (see Figures 3 and 4). Both groups showed the greatest strength decreases immediately after exercise; however, the WBPA group recovered to baseline values by 48 h post-exercise, while MVC values for the control group remained depressed throughout the 96 h recovery period.

### Blood Measurements

*CPK*. As shown in Figure 5, significantly higher CPK values were seen for the controls compared to the WBPA group ($p < 0.05$). Both groups showed increases in CPK post-test values ($p < 0.05$). For the control group CPK levels remained elevated at 24 h and increased at 72 h and 96 h; however, only the increases at 24 h reached statistical significance ($p < 0.05$).

*MYO*. No significant differences between the WBPA and control groups were seen in MYO concentration. Both groups showed a peak at the post test time point (1.97 ± 0.99 and 2.43 ± 1.08 respectively) ($p < 0.05$) and returned to baseline during the 4 days of recovery (Fig 6).

*IL-6, TNF-α and Uric acid*. Changes in inflammatory markers are shown in Table 2. There were no significant differences between groups. When normalized to pretest value, however, IL-6 showed a significant increase post-test in the WBPA group (1.81 ± 0.83).

*Muscle Soreness*

Ratings of muscle soreness are shown in Figure 7. While there were no significant differences between groups, there were significant within group differences across the 4 recovery days. In the WBPA group, the rating of soreness peaked at 24 h (27.4 ± 18.0 mm) and was significantly different from pretest values from 24 h to 72 h post exercise ($p < 0.05$). In contrast, the control group soreness score peaked at 48 h (32.4 ± 28.8 mm) and was significantly elevated from posttest through the recovery period. Differences in ratings of muscle soreness after the palpating test, and during flexion and extension are shown in Table 3.

*McGill Pain Questionnaire*

Figure 8 summarizes the within group differences in the McGill pain scores since no significant differences between groups were seen. The WBPA and control groups showed significant increases from baseline at post-test (3.6 ± 3.8 and 5.6 ± 3.6 respectively). The WBPA scores remained high at the 24 and 48 h time points, while the control group scores were elevated above post-test scores for the entire 96 h testing period ($p < 0.05$). The sensory and affective dimensions of the pain questionnaire test are summarized in Table 3.

*Circumference*

Changes in upper arm circumference are shown in Figure 9. While no significant differences in circumference were seen at any point in the recovery period for the WBPA group, the control group had a significant increase in circumference at 24 h (33.4 ± 3.5 cm) that remained elevated at the 48 h test point ($p < 0.05$).

*Range of Motion*

As shown in Figure 10, there were no significant differences between groups during the recovery days; however, both groups showed significant reductions in ROM at 24 h (0.97 ± 0.03 and 0.96 ± 0.04 for the WBPA and control groups, respectively) ($p < 0.05$). The deficits in ROM for the control group remained significant through 72 h post-exercise.

## CHAPTER 4: DISCUSSION

The purpose of the present study was to examine the effectiveness of WBPA as a recovery method to treat DOMS resulting from a bout of unaccustomed eccentric elbow flexion exercise. The major finding was that application of WBPA during the recovery period after an eccentric exercise bout enhanced muscle performance and diminished the negative impacts of DOMS in a young, fit male population sample.

To our knowledge this is the first study to show a more rapid strength recovery using WBPA compared to passive recovery. In contrast, other interventions such as cryotherapy (24, 32), active recovery (43, 76), stretching (27), electrical stimulation (38, 43), massage (75), and NSAIDs (63, 69), hyperbaric oxygen therapy (45), acupuncture (33), and compression garments (21) have failed to show a significant positive impact on strength performance during recovery after eccentric exercise. However, contrast water therapy (CWT) was shown to enhance strength recovery at 24h, 48h and 72h when compared to passive recovery (72, 73). It should be noted that differences in damage protocol, targeted muscle and performance measures exist between Vaile et al and the present study. In comparison to the CWT protocol, a greater decrease in muscle performance was seeing immediately following the first treatment in both, control and WBPA groups, indicating a disparity in the damage protocol effectiveness. Although both damage protocols used similar intensities, a lower volume in a bilateral leg press rather than unilateral arm curl were used in Vaile et al. Additionally, the positive response in strength recovery with CWT, typically took 24h, while WBPA produced improvements immediately after exposure which lasted throughout the 96 h evaluation period.

The mechanisms that may have contributed to the improved strength performance during the recovery period when applying WBPA are not clear. If reduced levels of structural damage are responsible for the increased force production seen with WBPA, the impact of NO should be considered. Although changes in intramuscular NO as a result of diffusion of NO from eNOS cannot readily be examined because of its rapid metabolism, its effects offer a plausible explanation for the increases in performance seen with WBPA since NO reduces muscular damage from inflammation and oxidative stress (6, 23, 25, 57).

Our results showing a significant difference in CPK concentration between the WBPA and control groups reflect to some extent those seen with other recovery methods. For example, in two separate studies using sport massage (64) and electro-membrane microcurrent therapy (37) CPK concentrations at 96 h after eccentric exercise were significantly lower than in controls. In contrast to our study, however, the use of microcurrent produced no improvement in strength recovery, while the sports massage study did not measure strength recovery. Additionally, while the results from studies using cryotherapy as a recovery modality are equivocal (24, 30, 32), those showing a positive impact (24, 32) reported reduced plasma CPK concentrations at 72 h post exercise, but failed to have a positive impact on strength. Finally, in contrast to our results, the use of light concentric exercise (76) or vitamin C supplementation (15) have produced no impact on CPK concentration or strength during recovery. Moreover, administration of vitamin C actually may be detrimental in DOMS (17).

The attenuated response of CPK in the WBPA group towards the end of the recovery period, along with improved recovery of strength, may indicate reductions in the levels of muscle damage with this intervention. However, it should be recognized CPK cannot be considered a direct measure of the degree of muscle damage due to the large variability in its response to eccentric exercise in similarly exercised individuals (16, 40, 52). While some recovery studies have shown CPK peaking within the first 24h after exercise (11, 22, 69), others have confirmed that the highest CPK concentration is found at day 4-5 of the recovery (37, 65). In addition, studies using different recovery methods have reported variations in the patterns of change in CPK concentration. In the current study, differences in CPK between the WBPA and control group may partially be explained by the NO release with WBPA (60); NO could modulate the CPK efflux by influencing the activation and accumulation of neutrophils (11, 65), as well as counteracting the appearance of reactive oxygen species (25) after eccentric exercise.

Along with CPK, MYO is commonly used to measure muscle damage. Even though there were no significant differences in MYO concentrations between groups, the earlier peak in MYO compared to CPK agrees with previous studies (72). Additionally, the increased MYO levels seen at 72 and 96 h post-exercise reflect the results reported by Beck et al (7) and Howatson et al (30) when providing a protease supplement or an ice massage, respectively. The differences in the time course of the appearance of MYO and CPK can be explained by MYO's smaller size and more direct route of delivery into the blood (62). Unlike MYO, due to its larger size CPK is delivered into the bloodstream via the lymphatic system which delays its appearance in the bloodstream following acute muscle damage.

Our current findings, which show significant increases in IL-6 by the WBPA group during the post-test assessment, have not been previously reported to our knowledge. Although IL-6 may be expected to increase with exercise (26), the lack of response in our controls is not without precedent (29). Results of previous studies indicate that the lack of significant increases in IL-6 concentration in the control group may have been due to the age and previous levels of conditioning of our participants (26, 53). Moreover, the timing of blood collection may blunt increases in IL-6 since peak concentrations have been shown between 6h to 12h post eccentric exercise (46, 70). In addition, our use of an exercise protocol involving a single arm may have limited the IL-6 response due to the low volume of muscle mass activated (24, 51).

The increase in IL-6 seen with WBPA may have been the result of NO increases at the muscle level. NO has been shown to upregulate the pretranslational signaling events leading to muscle IL-6 production (67). The increase in IL-6 can be considered a positive response since it has been proposed to stimulate the production of anti-inflammatory cytokines and may indirectly inhibit pro-inflammatory cytokines (50, 54-56, 66). Moreover, IL-6 also increases satellite cell proliferation and muscle regeneration (25). The lack of significant differences in systemic levels of TNF-$\alpha$ in our study reflects the results reported by other researchers who examined inflammatory markers following an acute bout of exercise.

Even though no significant differences in soreness and pain scores were seen between groups, the shorter durations of the soreness and pain responses in the WBPA group demonstrates an enhanced recovery pattern within this group. The positive impact of WBPA on pain is not without precedent; in a comparable study with

fibromyalgia patients, 45 minutes of WBPA produced significant reductions in pain within one to three treatments (61). The potential for NO to reduce inflammation and edema may explain, in part, the more rapid recovery from soreness and pain seen in the WBPA group due to reduced activation of the pain afferent fibers. The role of NO in pain relief has recently been confirmed by the work of Cunha et al (20), who found that the analgesic effects of morphine are achieved by stimulating a nNOS/NO/KATP channel antinociceptive pathway, this pathway appears to cause a hyperpolarization of nociceptive neurons, counteracting their enhanced excitability during the inflammatory process. Also, NO counteracts the effects of endothelin-1 that also plays a role in pain perception (28, 42).

The lack of significant increase in circumference in the WBPA compared to the increase at 24 and 48 h in controls is indicative of the capacity of WBPA to reduce the localized swelling commonly associated with DOMS. This positive impact is further supported by the more rapid recovery of ROM in the WBPA group compared to controls. The impact of WBPA on circumference and ROM may be explained by an increased muscle blood flow or a decrease in inflammation. While the possibility that localized blood flow served as a mechanism to reduce swelling and increase ROM in the present study is questionable given the results reported by Adams et al (2) showing no significant increase in muscle blood flow in pigs exposed to WBPA; however, muscle blood flow may still be considered a possible mechanism since these researchers reported a 158% increase (albeit not significant) in muscle blood flow with a WBPA exposure lasting 10 minutes rather than the 45 minutes used in the current study. The information supporting reductions in inflammation as a possible mechanism is supported by the positive impacts

of NO on skeletal muscle inflammation, due to reduced neutrophil-mediated lysis and decrease superoxide concentration (68).

## LIMITATIONS

A limitation of this study was the failure to quantify leukocytes during the recovery period. However, the quantification of cytokines provided a reliable indicator of inflammation. Additionally, the quantification of glutathione could further help to explain changes in some of the criterion measurement since it has been previously shown that subjects with low total plasma glutathione levels had a smaller plasma CPK and MYO response, and faster recovery from eccentric exercise compared with subjects with higher levels (40). Moreover, lack of hydration control during the recovery period should also be considered a limitation since hydration status can affect lymph flow and therefore impact the levels of CPK following exercise (62).

Finally controlling diet during the recovery period may have also provided a more stable testing environment (18), although the impact of nutrition on DOMS, as well as inflammation, is equivocal (8, 47).

## CONCLUSION

The use of WBPA as a recovery method after high-intensity eccentric resistance exercise improved strength recovery and had a positive impact on DOMS symptoms. These benefits were most likely the result of the enhanced release of NO through WBPA. Future research should investigate the effects of WBPA within skeletal muscle through quantification of inflammatory and oxidative stress markers as well as ultrastructural damage using electron microscopy. Finally, the use of WBPA as a pre-conditioning

method to reduce levels of DOMS and potentially increase subsequent cardiovascular and neuromuscular performance should be the subject of future investigations.

## FIGURES

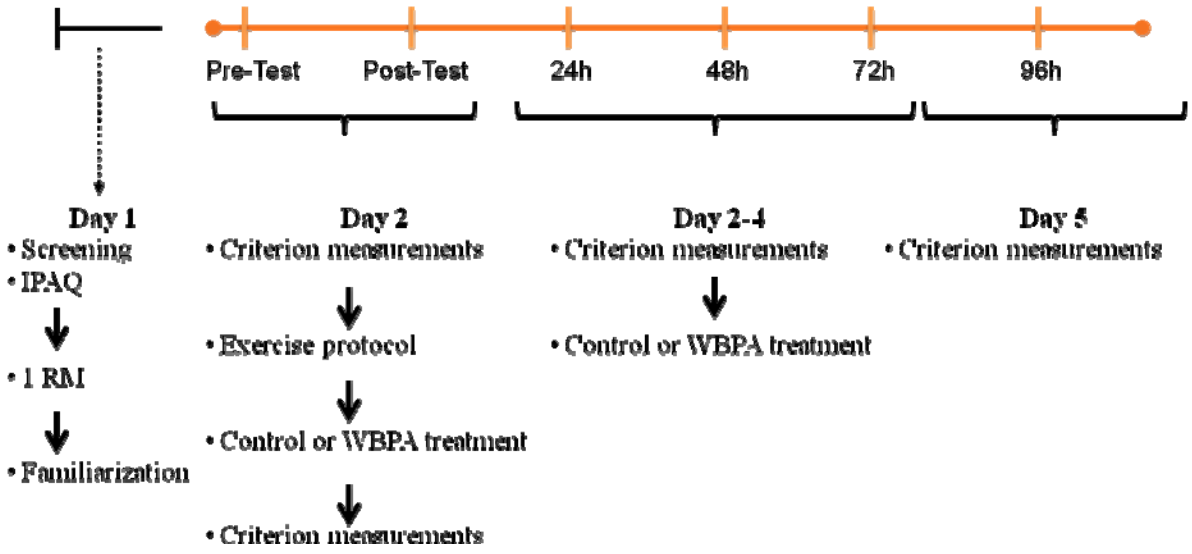


Figure 1. Study Design

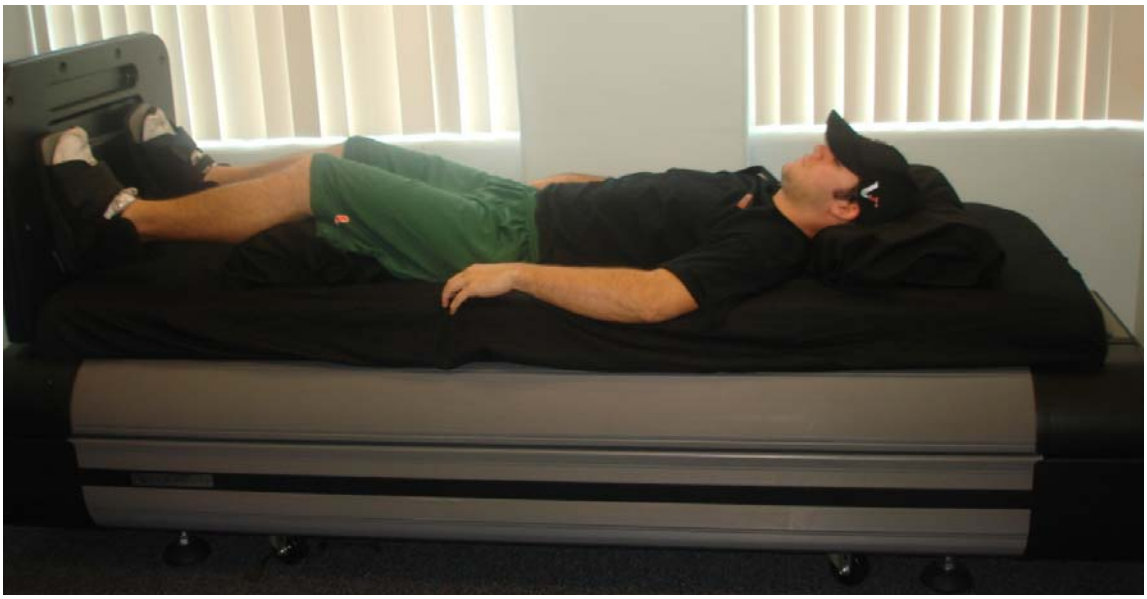

Figure 2. Subject during the whole body periodic acceleration treatment using the Exer-Rest AT motion platform.

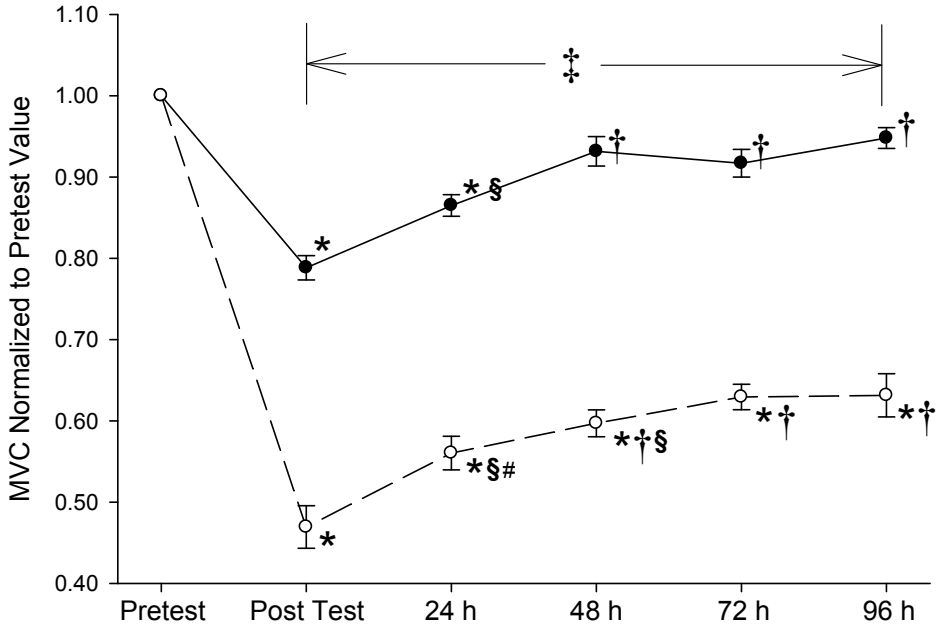


Figure 3. Maximal Voluntary Contraction scores at 90$^{o}$ elbow angle for the whole body periodic acceleration (●) and control (○) groups. ‡Significant difference between groups ($p < .05$). *Significantly different from pretest ($p < .05$). †Significantly different from post test ($p < .05$). #Significantly different from 48 hours ($p < .05$). §Significantly different from 72 hours ($p < .05$).

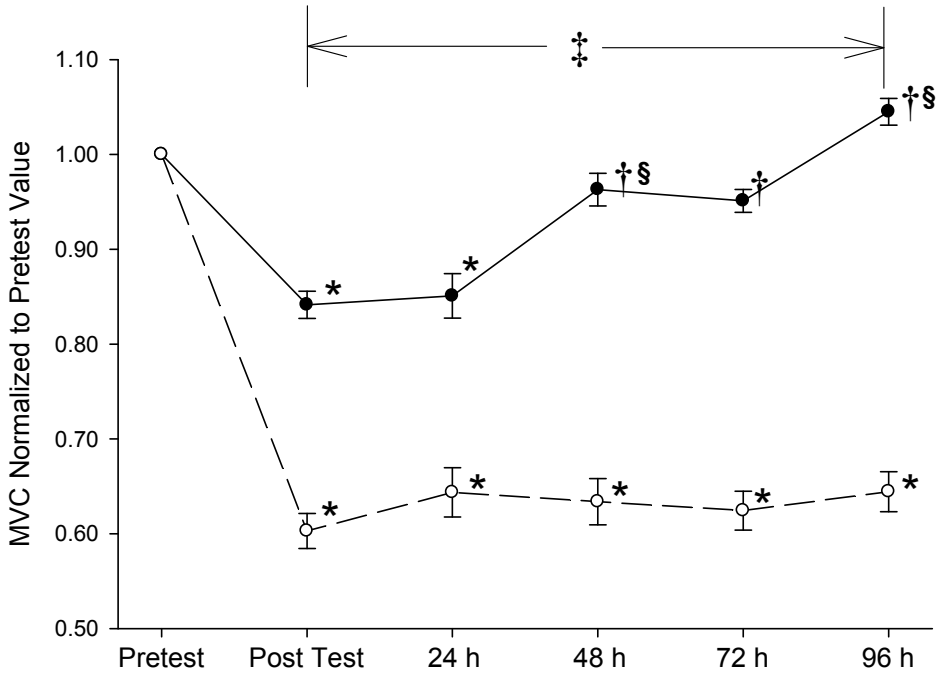


Figure 4. Maximal Voluntary Contraction scores at 150$^{o}$ elbow angle for the whole body periodic acceleration (●) and control (○) groups. ‡Significant difference between groups ($p < .05$). *Significantly different from pretest ($p < .05$). †Significantly different from post test ($p < .05$). §Significantly different from 24 hours ($p < .05$).

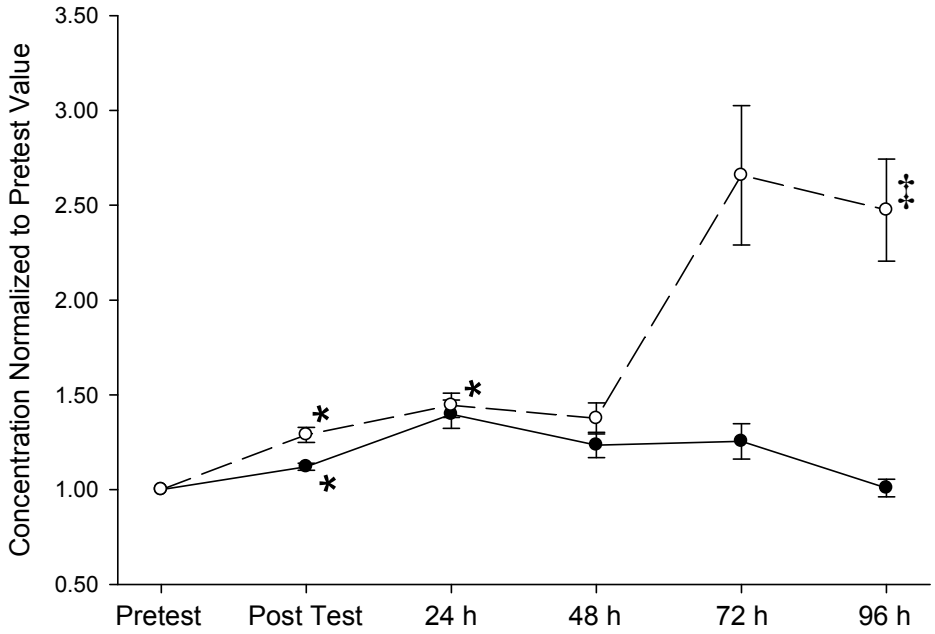


Figure 5. Concentration of Creatine Phosphokinase normalized to pretest values for the whole body periodic acceleration (●) and control (○) groups. ‡Significantly different from whole body periodic acceleration group ($p<.05$). *Significantly different from pretest ($p < .05$).

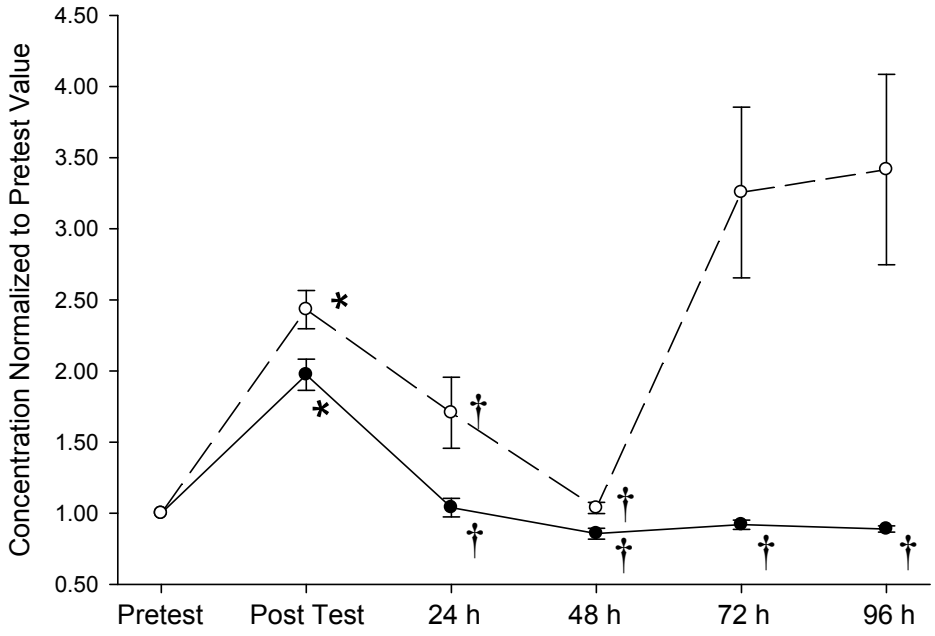


Figure 6. Concentration of myoglobin normalized to pretest values for the whole body periodic acceleration (●) and control (○) groups. *Significantly different from pretest ($p < .05$). †Significantly different from post test ($p < .05$)

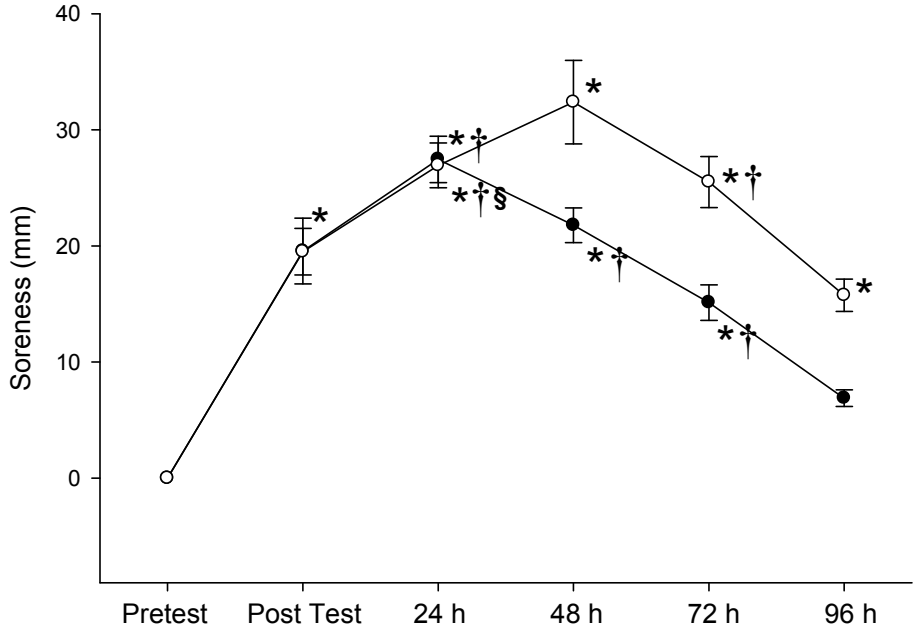


Figure 7. Muscle soreness using the visual analog scale for the whole body periodic acceleration (●) and control (○) groups. *Significantly different from pretest ($p < .05$). §Significantly different from 72 hours ($p < .05$). †Significantly different from 96 hours ($p < .05$).

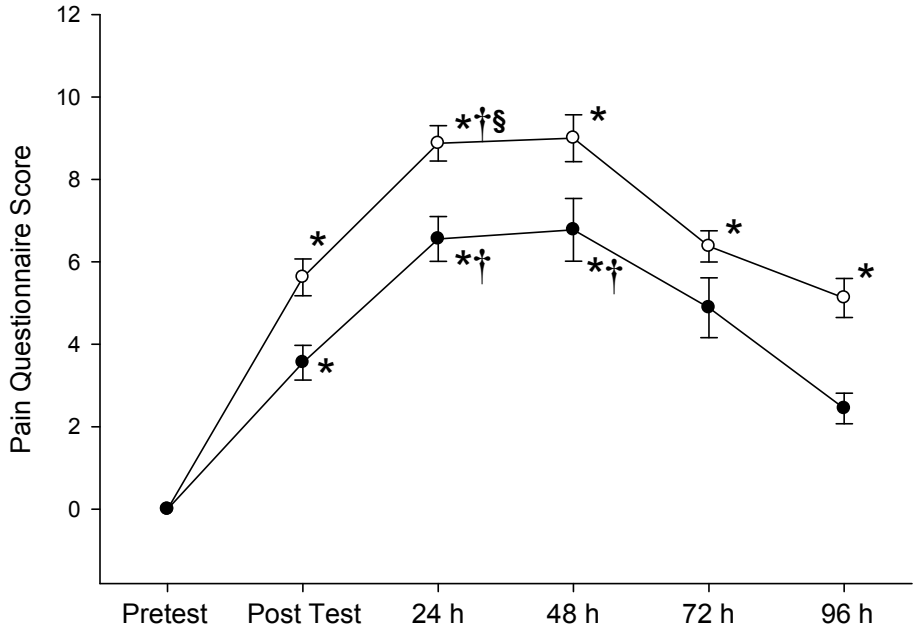


Figure 8. Pain Questionnaire score for the whole body periodic acceleration (●) and control (○) groups. *Significantly different from pretest ($p < .05$). §Significantly different from 72 hours ($p < .05$). †Significantly different from 96 hours ($p < .05$).

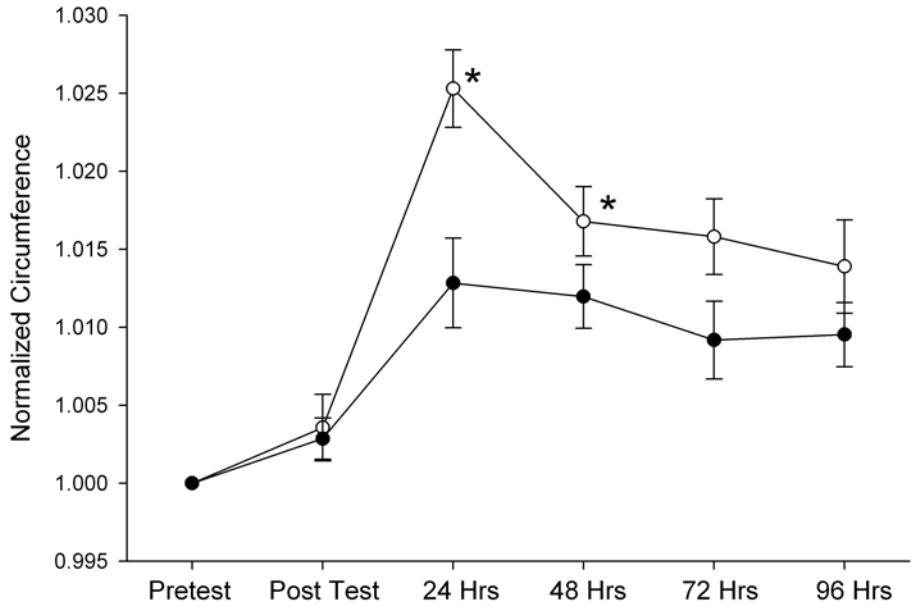


Figure 9. Circumference of the upper arm normalized to pretest values for the whole body periodic acceleration (●) and control (○) groups. *Significantly different from pretest ($p < .05$). †Significantly different from post test ($p < .05$).

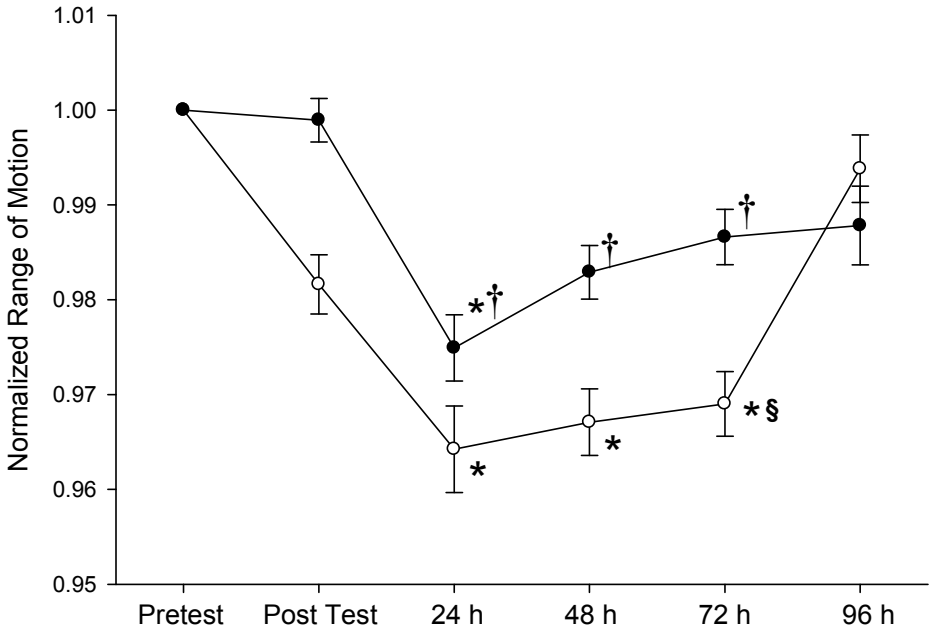


Figure 10. Range of motion of the elbow joint normalized to pretest values for the whole body periodic acceleration (●) and control (○) groups. *Significantly different from pretest ($p < .05$). †Significantly different from post test ($p < .05$). §Significantly different from 96 hours ($p < .05$).

## TABLES

**Table 1. Physical Characteristics of Subjects**

| Variable | Control *(n=8)* | WBPA *(n=9)* |
|---|---|---|
| **Age (yr)** | 24.3 ± 6.5 | 22.6 ± 2.3 |
| **Height (cm)** | 175.8 ± 5.4 | 176.2 ± 7.2 |
| **Mass (kg)** | 80.7 ± 14.0 | 77.8 ± 9.2 |
| **Body mass index** | 26.1 ± 4.1 | 25.1 ± 3.2 |
| **1RM** | 28.4 ± 3.5 | 31.1 ± 4.9 |

Values are means ± SD

**Table 2. Blood Markers**
Values are means ± SD; n Control = 7, n WBPA=9.
*Significantly different from Pre ($p < 0.05$). †Sig different from 96 h ($p < 0.05$).

| Variable | Group | | Pre | 1 h | 24 h | 48 h | 72 h | 96 h |
|---|---|---|---|---|---|---|---|---|
| **IL-6** | *Control* | *Abs* | 1.14 ± 0.55 | 1.82 ± 1.14 | 1.14 ± 0.54 | 0.91 ± 0.30 | 1.53 ± 0.66 | 0.73 ± 0.19 |
| | | *Norm* | 1.00 ± 0.00 | 1.60 ± 1.60 | 1.01 ± 0.54 | 0.92 ± 0.44 | 1.55 ± 0.81† | 0.74 ± 0.29 |
| | *WBPA* | *Abs* | 0.60 ± 0.35 | 1.06 ± 0.83 | 1.34 ± 1.62 | 1.25 ± 1.03 | 1.01 ± 1.24 | 0.98 ± 0.76 |
| | | *Norm* | 1.00 ± 0.00 | 1.81 ± 0.83* | 3.10 ± 4.38 | 2.69 ± 2.66 | 2.73 ± 4.03 | 2.97 ± 4.22 |
| **TNF-α** | *Control* | *Abs* | 0.00 ± 0.00 | 0.02 ± 0.04 | 0.00 ± 0.00 | 0.03 ± 0.07 | 0.00 ± 0.00 | 0.00 ± 0.00 |
| | *WBPA* | *Abs* | 0.00 ± 0.00 | 0.15 ± 0.29 | 0.09 ± 0.26 | 0.25 ± 0.51 | 0.00 ± 0.00 | 0.44 ± 1.24 |
| **Uric Acid** | *Control* | *Abs* | 8.92 ± 1.68 | 9.35 ± 2.48 | 9.22 ± 1.79 | 9.25 ± 1.61 | 9.39 ± 2.12 | 9.97 ± 2.45 |
| | | *Norm* | 1.00 ± 0.00 | 1.04 ± 0.10 | 1.05 ± 0.17 | 1.07 ± 0.26 | 1.09 ± 0.34 | 1.14 ± 0.27 |
| | *WBPA* | *Abs* | 9.53 ± 1.24 | 9.24 ± 1.44 | 9.31 ± 1.54 | 9.63 ± 1.60 | 9.31 ± 1.53 | 9.29 ± 1.37 |
| | | *Norm* | 1.00 ± 0.00 | 0.97 ± 0.09 | 0.98 ± 0.15 | 1.01 ± 0.13 | 0.98 ± 0.17 | 0.98 ± 0.15 |

**Table 3. Soreness and Pain Markers**
Values are means ± SD; n Control = 8, n WBPA=9.
* Significantly different from Pre ($p < 0.05$). §Sig different from 1 h ($p < 0.05$). #Sig different from 24 h. $p < 0.05$. ‡Sig different from 72 h ($p < 0.05$). †Sig different from 96 h ($p < 0.05$)

| Variable | Group | Pre | 1 h | 24 h | 48 h | 72 h | 96 h |
|---|---|---|---|---|---|---|---|
| **Soreness Flexion** | *Control* | 0.00 ± 0.00 | 2.50 ± 4.44 | 13.25± 18.06§ | 19.13 ± 22.47§ | 12.13 ± 14.11§ | 8.38 ± 10.53 |
| *(mm)* | *WBPA* | 0.00 ± 0.00 | 1.44 ± 2.19 | 11.22 ± 9.76*§ | 18.06 ± 16.36*§ | 12.56 ± 8.69*§ | 9.00 ± 11.27 |
| **Soreness Extension** | *Control* | 0.00 ± 0.00 | 3.50 ± 6.05 | 18.50± 18.84*§ | 21.63 ± 20.40*§ | 19.25 ± 22.95* | 13.75 ± 17.07 |
| *(mm)* | *WBPA* | 0.00 ± 0.00 | 1.83 ± 1.90* | 17.33 ± 14.34*§ | 22.11 ± 17.07*§† | 23.11 ± 18.60*§† | 10.89 ± 14.32 |
| **PPT Mid** | *Control* | 0.00 ± 0.00 | 4.00 ± 4.99 | 10.88 ± 11.39*§ | 13.75 ± 13.82*§† | 10.13 ± 11.56*† | 5.88 ± 6.77* |
| *(mm)* | *WBPA* | 0.00 ± 0.00 | 1.33 ± 1.58 | 13.22 ± 13.69*§† | 15.89 ± 19.16* | 15.00 ± 23.85 | 5.56 ± 8.60 |
| **PPT Above** | *Control* | 0.00 ± 0.00 | 2.63 ± 3.74 | 4.13 ± 6.10*† | 8.50 ± 9.40* | 4.75 ± 5.87 | 2.13 ± 3.00 |
| *(mm)* | *WBPA* | 0.00 ± 0.00 | 1.33 ± 1.80 | 4.89 ± 4.99*† | 7.33 ± 10.81 | 4.33 ± 6.10 | 1.22 ± 1.79 |
| **PPT Bellow** | *Control* | 0.00 ± 0.00 | 1.75 ± 2.87 | 15.25 ± 21.31 | 21.56 ± 24.01*# | 16.31 ± 20.55*#§ | 10.88 ± 10.11*§ |
| *(mm)* | *WBPA* | 0.00 ± 0.00 | 2.11 ± 2.57 | 12.00 ± 12.47*§ | 21.33 ± 23.65*§ | 16.44 ± 23.05 | 9.11 ± 12.28 |
| **PQT Sensory** | *Control* | 0.00 ± 0.00 | 4.24 ± 3.24* | 7.13 ± 2.75*‡† | 7.75 ± 3.81* | 5.38 ± 3.02* | 4.75 ± 3.58* |
| | *WBPA* | 0.00 ± 0.00 | 2.33 ± 3.57 | 5.67 ± 3.64*‡† | 5.56 ± 5.55*† | 4.11 ± 4.99*† | 2.22 ± 3.03 |
| **PQT Affective** | *Control* | 0.00 ± 0.00 | 1.38 ± 1.06*† | 1.75 ± 1.04*† | 1.25 ± 1.16* | 1.00 ± 0.93*† | 0.38 ± 0.74 |
| | *WBPA* | 0.00 ± 0.00 | 1.22 ± 1.09* | 0.89 ± 1.62 | 1.22 ± 1.39*† | 0.78 ± 1.72 | 0.22 ± 0.44 |

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