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THE EFFECTS OF WHOLE-BODY VIBRATION ON MUSCLE RECOVERY AND PERFORMANCE

DISSERTATION

A Dissertation
presented in partial fulfillment of requirements
for the degree of Doctorate of Philosophy
in the Department of Health, Exercise Science, and
Recreation Management
The University of Mississippi

by

Nicole C. Dabbs

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ABSTRACT

Facilitating muscle recovery in trained individuals is essential, as it allows for a quicker return to activity without reduction in performance. Many proposed modalities have been studied but have not shown consistent effectiveness. A relatively new modality, whole-body vibration (WBV) has been shown to increase strength and power outcomes and recently has been shown to decrease perceived pain associated with muscle soreness. Therefore, the purpose of this study is to examine the effects of WBV following exercise induced muscle damage over a period of 72 hours in recreationally trained females. Participants were randomly selected into either the control group or the WBV group. There were three familiarization visits and four testing visits lasting about 45mins each. During every testing visit, all dependent variables were assessed 3 times (pre, post1, post2) in the following order: vertical jump, maximal voluntary isometric strength, interpolated-twitch, muscle activity, pressure pain threshold (PPT), range of motion (ROM), thigh circumference, and pain on movement. On visit 4, pre assessments were taken followed by 4, 40% front loaded, sets to repetition failure during split squats to induce muscle damage. This was followed immediately by WBV or control (rest) and the measurement of dependent variables. Following a 10 minute rest, measurements were reassessed. Visits 5-7 were replications of visit 4 with the exclusion of the damage protocol. Each dependent variable was measured by a 2x12 (group x time) mixed factor ANOVA. Significant ($p<0.05$) main effects for group were found for twitch torque up to 24hr post, with control being greater than WBV. No significant main effects for group

were found for all other variables. There were significant main effects for time from 0Pre to 24Pre and 48Pre in all PPT measures, active ROM, and muscle pain on movement. Significant ($p<0.05$) main effects for time were found for vertical jumping variables, indicating jumping performance declined following muscle damage. A significant ($p<0.05$) main effect for group was found for normalized peak EMG during jumping, indicating the control group exhibited greater muscle activity than the WBV group. Significant ($p<0.05$) main effects for time were found for muscle contractile properties, indicating a change in muscle contractile properties following muscle damage. These results indicate that WBV does not aid in alleviating muscle pain or symptoms, vertical jump performance and voluntary muscle contractile properties following exercise induced muscle damage with further research needed in clinical and/or athletic populations.

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1. **DOMS= delayed onset muscle soreness**
2. **EIMD= exercise induced muscle damage**
3. **WBV= whole-body vibration**
4. **PPT=pressure pain threshold**
5. **VM=vastus medialis**
6. **VL=vastus lateralis**
7. **RF=rectus femoris**
8. **aROM=active range of motion**
9. **pROM=passive range of motion**
10. **VJH= vertical jump height**
11. **PPO=peak power output**
12. **RFD= rate of force development**
13. **rGRF=relative ground reaction force**
14. **pEMG ratio= peak electromyography ratio**

15. %Act= percent activation

16. TT= twitch torque

17. VT= voluntary torque

18. TP= time to peak tension

19. HRT= half relaxation time

20. %Δ= percent change

CHAPTER I

INTRODUCTION

Delayed onset muscle soreness (DOMS) has been reported as an undesirable side effect of exercise due to its painful and debilitating effects on an individual (18). DOMS usually peaks at 24 to 48 hours following exercise (13,14) and presents symptoms such as tenderness, pain, swelling, and muscle stiffness (14). It has been suggested that these symptoms are related to an inflammatory process based on the lack of evidence of neural inhibition of damaged muscle (14) or changes in motor unit activation (17).

Repeated muscle contractions have been shown to cause muscle damage resulting in decreased force production, most evident in the eccentric phase (2,15). This muscle damage is evident as a disruption of the normal alignment of the skeletal muscle and disruption of the z-lines of sarcomeres (12,20). This process initiates the inflammatory process leading to muscle soreness. Production of prostaglandin E_2 which sensitizes type III and IV afferent fibers of muscle connective tissue, which are highly correlated with DOMS pain(7), has been observed at 24, 48 and 72 hours (12).

Eccentric exercises are commonly used as a component of strength-training programs and have been shown to elicit DOMS, potentially causing reduction in sport performance measures. Previous researchers have studied several ways to control or prevent DOMS (6). Most current modalities have not been shown to be consistently effective. These include, but are not limited to massage, cryotherapy, stretching, homeopathy, ultrasound, and electrical current (6).

Over the last decade, whole-body vibration (WBV) has increasingly been implemented with exercise due to the application of sinusoidal vibrations to the body showing positive effects on strength (10), power development (9), performance (8) and flexibility (11). Although the exact mechanism of how the body responds to the vibration

stimulus remains unclear, it has been suggested that it elicits neuromuscular facilitation (4,5). It has been previously shown that when vibration is directly placed on a tendon or muscle belly, vibration induces activity of the muscle spindle Ia fibers, mediated by monosynaptic and polysynaptic pathways (19). This increase in muscle activity elicits a tonic vibration reflex (TVR) arising from the vibratory stimulus. When WBV is implemented it is theorized that vibrations are transferred from the platform to specific muscle groups. Consequently, WBV stimulates the sensory receptors and afferent pathways, which may lead to a more efficient use of the stretch reflex, recruitment and synchronization of motor units (4). Although these mechanisms have been postulated, none have been clearly demonstrated in or after implementing WBV.

Recently, WBV has been suggested as a novel modality to reduce or control DOMS (1,3,16). Bakhtiary et al. 2007 found that vibration prior to eccentric loading may prevent and control DOMS with possible mechanisms of increased blood flow to facilitate recovery and regeneration and possible pain inhibition for a decrease in pain (3). Rhea et al. 2009, implemented WBV in combination with stretching and massage after strenuous exercises over a period of 72 hours, showing a decreased perceived pain in the WBV group (16). Aminan-Far et al. 2011 also showed a reduction in DOMS symptoms by measuring maximal isometric and isokinetic voluntary strength loss, creatine kinase, pain threshold and muscle soreness with WBV implanted prior to eccentric exercises (1).

Previous research has not investigated the effects of WBV post eccentric exercise on its effects on DOMS and recovery. Therefore, we hypothesize that applying WBV after eccentric exercises and subsequent days after may decrease DOMS symptoms while exploring the effectiveness of WBV in attenuating DOMS trained individuals.

Specific Aims:

Specific Aim 1:

To investigate the impact of whole-body vibration on pain rating, pressure pain threshold, range of motion, and thigh circumference following exercise induced muscle damage over time periods of 0, 24, 48, and 72 hours.

Specific Aim 2:

To investigate the impact of whole-body vibration on vertical jump height, peak power output, relative ground reaction forces, rate of force development, and normalized muscle activity in the vastus medialis during maximal vertical jump following exercise induced muscle damage over time periods of 0, 24, 48 and 72 hours.

Specific Aim 3:

To investigate the impact of whole-body vibration on maximal isometric force, percent muscle activation, twitch force, time to peak force, half relaxation time, and muscle activity on knee extensors following exercise induced muscle damage over time periods of 0, 24, 48 and 72 hours.

The following null hypotheses will be tested:

Ho_{1a}: There will be no difference in VAS responses between and within groups over time.

Ho_{1b}: There will be no difference in PPT responses between and within groups over time.

Ho_{2a}: There will be no difference in A-ROM responses between and within groups over time.

Ho_{2b}: There will be no difference in A-ROM responses between and within groups over time.

Ho_{3}: There will be no difference in thigh circumference responses between and within groups over time.

Ho_{4a}: There will be no difference in VJH responses between and within groups over time.

Ho_{4b}: There will be no difference in PPO during VJ responses between and within groups over time.

Ho_{4c}: There will be no difference in GRF during VJ responses between and within groups over time.

Ho_{4d}: There will be no difference in RFD during VJ responses between and within groups over time.

Ho_{5a}: There will be no difference in %Activation in ITT responses between and within groups over time.

Ho_{5b}: There will be no difference in time to peak in ITT responses between and within groups over time.

Ho_{5c}: There will be no difference in half relaxation time in ITT responses between and within groups over time.

Ho_{6a}: There will be no difference in mean and peak EMG amplitude in VMO in MVC responses between and within groups over time.

Ho_{6b}: There will be no difference in mean and peak EMG amplitude in TA in MVC responses between and within groups over time.

Ho_{6c}: There will be no difference in mean and peak EMG amplitude in MG in MVC responses between and within groups over time.

Ho_{7a}: There will be no difference in normalized mean and peak EMG amplitude in VMO in VJ responses between and within groups over time.

Ho_{7b}: There will be no difference in normalized mean and peak EMG amplitude in TA in VJ responses between and within groups over time.

Ho_{7c}: There will be no difference in normalized mean and peak EMG amplitude in MG in VJ responses between and within groups over time.

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CHAPTER II

LITERATURE REVIEW

Delayed Onset Muscle Soreness

Physiology and Mechanism

Delayed onset muscle soreness is an exercise induced skeletal muscle injury induced by eccentric muscle actions. Injury in the skeletal muscles often causes an undesirable tenderness, feelings of stiffness and pain. There have been a plethora of studies published examining exercise induced muscle injury, potential mechanisms and treatments all of which have determined a variety of markers of injury. This concern of muscle injury has been deemed of importance both in basic and clinical sciences and can relate on the functionality of human function and movement. Legislation, Omnibus Budget Reconciliation Acts of 1989 and 1990, have been passed due to the earlier act created by the Agency for Health Care Policy and Research (AHCPR) which allows for research in effectiveness of diagnostic, therapeutic and preventative health. These paved the path for two acts from the National Center for Medical Rehabilitation Research (NCMRR) under the National Health Institute (NIH), resulting in development of research on functional outcome measures tools (44,45,55). The importance on functional measures through these acts warranted medical significance for exercise-induced muscle injury (102). Muscle function has many factors that contribute to the ability to exert force which can be given over set of conditions: range of motion (ROM) or at a fixed length at a given velocity or at a given external load, at a given level of activation and over a given number of muscle actions. Therefore, it is critical that using the loss of muscle function, as a tool can be beneficial in assessing exercise induced muscle injury.

It is well established that repeated muscle actions have been reported to cause muscle injury resulting in a decrease force production due to the eccentric phase (6,84). Eccentrics can produce greater torques (37) with producing lower motor unit activation (37,60,72), this is thought to place a high mechanical stress on muscle fibers causing muscle damage (37). Crossbridges are detached during eccentric muscle actions mechanically with greater force and is stretched further than other muscle actions, resulting in damage (37). Damage caused by eccentric muscle actions can cause severe structural changes at the cellular level. Disruption of the sarcomeres is the first sign of damage and damage to the components of the excitation-contraction coupling system (5,75,110). Within the sarcomere, Z-lines in particular are disrupted (40,80) and are usually seen mostly in Type II muscle fibers due to their narrowest and weakest Z-lines, resulting in the process of muscle fiber degeneration (5,6,56,103) in the damaged muscle. The cellular disruption initiates the inflammatory response causing a transfer of fluid and cells to the affected muscles for the removal of damaged contractile proteins and byproducts (85). This inflammatory response from exercise induced muscle damage leads to DOMS, muscle stiffness, increased limb volume and circumference of damaged muscle, decreased ROM, decreased muscular strength, decreased power output and increased levels of creatine kinase (CK), blood lactate and hemoglobin in the blood (24,85). Each symptom caused from exercise induced muscle damage has its own time course before it returns to baseline.

Assessing Muscle Damage

When assessing exercised induced muscle injury, the most functional assessment examines maximal voluntary contraction (MVC) torque, since it is directly proportional

to force produced. Since torque is force times the moment arm, torque produced is joint angle dependent, where the length/tension relationship of that muscle is an important factor. Therefore, it is important to note torque production joint angles used when comparing studies, since at varied joint angles, the torque will change. The torque/velocity relationship plays a role in torque produced because torque is also velocity dependent. Because of these relationships with torque, the best way to assess torque is through an isometric MVC, controlling for joint angle and velocity (111).

There are a few disadvantages in measuring MVC torque as a functional measure following exercise induced muscle injury due to fatigue and motivation and pain. Since it is difficult to differentiate fatigue related decreases in torque from exercise induced muscle injury (39), fatigue may play a role during an MVC torque measurement or immediately after an injury protocol, which makes it difficult to be certain the cause of torque reduction. Motivation is a contributing factor during an MVC torque measurement with individuals, it has been argued whether or not all motor units are being recruited (43,97,104). Measurements of validity have been previous examined for MVC torque, having a relatively high reliability of an interclass correlation coefficients ≥ 0.85 (2,59). This supports the idea that the reduction in MVC torque from exercise induced muscle injury is consistent over the time course of degeneration and regeneration process in the muscle (111). It is well established that immediately after injury, MVC torque is down about 60% and continues to recover to baseline measures over a period of 1-2 weeks (23).

Along with MVC torque production, ROM is commonly used assessment tool for exercise induced muscle injury. One of the symptoms of muscle damage is increased fluid to the damaged muscle and using ROM to assess the amount of swelling in the

muscle is functionally important. ROM is joint specific and is a measure of the arc over that specific joint. The joint angle of each joint is a function of the amount of skin, subcutaneous tissue, tendon, articular capsule and bone properties and amount of musculature. ROM can be measured using a goniometer through active or passive ROM of each joint, where active ROM is how much the individual can move that joint and passive ROM is how much the investigator can move the joint through a ROM until pain occurs. Reliability of ROM following exercise induced muscle injury has been shown in the rehabilitative medicine field with an interclass correlation coefficient of .3-.9 and intra-rater reliability inter-class correlation coefficients ≥ 0.9 (42,89,96).

A more direct way of assessing exercise induced muscle injury is at the sarcomere level through needle biopsy, where a small sample is taken invasively from the damaged muscle. It is then examined through a light or electron microscope where disruption of sarcomeres and z-line streaming can be seen. Although direct measures are usually a more precise way of measurement, for muscle damage it can surface some problems. Since needle biopsies are only taken from a very small (10-50mg) area of the muscle, it is only a small representative of the whole muscle itself. Needle biopsies are usually only take from muscle, where usually more than one muscle is involved in a joint action, which has lead to speculations of how it can determine the damage of the movement. The histology of muscle fibers following exercise induced muscle injury has been shown to poorly correlate with functional measurements like MVC torque production. This is related to the difference in time course and magnitude of the damage, where abnormalities in muscle cross-sections are not evident until several days after injury, where MVC torque production is immediately decreased (41). Another drawback of

using needle biopsy for assessing muscle damage is that it has been shown that it increases blood CK levels (47), making it hard to differentiate the results of using CK as a damage marker.

Often when determining markers for muscle damage, blood levels of myofibril proteins like CK, locate dehyrogenase and myoglobin are measured. An increase in blood proteins has not been shown until at least 24 hours post injury which is inconsistent with functional measures (MVC torque production), resulting in a poor correlation with each other (22,47,78,79). However, a contradicting study shows that CK and myoglobin are significantly correlated with the reduction in MVC torque and ROM only at time points greater than 24 hours, anything prior variance in blood levels are too variable (90). There has also been evidence that repeated bouts of eccentric muscle actions, blood CK levels are elevated while contractile decrements are only minimally attenuated (78). There is evidence that there is extreme individual variability in blood levels of CK within an individual (22), which results in inconsistency as a marker for damage. Additionally, dissociation has been shown in histological signs of injury and blood levels of CK (38). There is not enough evidence to show that blood levels of myofiber proteins can reflect functional movement following exercise induced muscle injury.

A subjective method of assessing exercised induced muscle injury is through a scale of soreness or pain occurring the damaged muscle. The visual analog scale has commonly been used to rate the amount of soreness the individual is experiencing. Soreness has been shown to have low correlations with functional movements (50,70,91,99) due to time course and magnitude, since soreness doesn't appear until at

least 24 hours while peaking 24-72 hours post injury. Another method to assess pain caused by exercised induced muscle injury is through pressure pain threshold (PPT).

Non-invasive imaging devices are used as assessments of exercise induced muscle injury, researchers have used magnetic resonance imaging (MRI) and computed tomography (CT) scans, ultrasound and ϒ-camera imaging. MRI and CT scans are typically utilized to measure increased volume in the localized muscle that was damaged. As expected, these do not correlated highly with functional measures (50,70,91) but gives more precise way of measuring increased volume however; abnormities have been detected and peaks from 3 to 6 days after damage (56,70,80,91,101).

Effects on Performance

During most performance activities the main goal is to generate the most amount of power output, however, with exercise induced muscle injury, power generation may be compromised. It has been shown that peak power output has an immediate reduction following eccentric muscle actions in knee extensors during isokinetic cycling (98) and during Wingate cycle test (19) while also continuing to reduce up to 2 days post injury. A decrease in power output has also been shown during 10 x 6s intermittent maximal sprints on a cycle ergometer after 10 sets of 10 plyometric jumps to induce damage (108). Vertical jump performance is peak power performance output, predicting measurement and can be compromised following exercise induced muscle damage. Studies have found a prolonged reduction in maximal force production, EMG activity, ground reaction forces, stretch reflex sensitivity, muscle and joint stiffness regulation and stretch shortening cycle (8,49); which all play a role in jumping performance. Vertical jump

performance with and without a countermovment have been shown to have an immediate and long-lasting reduction in performance up to 4 days post injury but are dependant on jump type (20). Squat jump had the most prolonged reduction in jump height compared to the countermovement jump and depth jump.

To produce maximal muscle force recruitment and activation of motor units is critical and is often reduced following exercise induced muscle injury. A decrease in muscle force output seen with electromyographic (EMG) activity has been shown (33,62) and indicated that a greater central activation is necessary to achieve a given sub maximal or maximal force. EMG can show in general if a muscle is more or less active but it can also be measured specifically using interpolated twitch technique (ITT) to measure percentage of activation of the motor units during voluntary contractions. It has been proposed that muscle activation is better determined by extrapolating the relationship between evoked and voluntary force to provide an estimate of true maximum force. In healthy individuals ITT has been shown that different muscles have different activation percentages, for example the quadriceps femoris activation is between 85-95% (46,51,58) compared to ankle plantar flexors that have a percent activation of 80-99% (100). ITT has also been shown in determining recovery by percent activation following exercise induced muscle injury (76,83).

Recovery Modalities

Previously, a variety of recovery modalities such as stretching, massage, cyrotherapy and ultrasound have been used to treat DOMS symptoms but have been shown to be inconsistent. The most commonly used modalities practiced are passive

stretching and massage but research is limited and conflicting. Some studies have investigated massage (105) and stretching (68) individually or in combinations of treatments such as warm-up, stretching and massage (92), warm underwater water jet massage (109) and ice massage (114). Alternative modality methods such electrical stimulation, light exercise (4), aerobic exercise (107) as hyperbaric oxygen therapy (73), acupuncture (66), and whole body vibration (87) have been examined as well but no one modality has been deemed to work more efficiently than another.

Whole-body Vibration

Physiology and Mechanism

Vibration is a mechanical stimulus elicited by oscillatory motions. These oscillatory motions are determined by the frequency and amplitude of the vibration exposure. The mechanism(s) in which whole-body vibration (WBV) occurs is still unclear in the literature but has been shown to have significant performance and clinical benefits. It has been previously suggested that enhanced performance following WBV may result from enhanced muscle spindle sensitivity and gamma activation, leading to increased motor unit recruitment and neuromuscular facilitation. If muscle spindle threshold is decreased, an increase in Ia afferent fibers would increase muscle activation through facilitation of homonymous alpha motor neurons (86). It has also been suggested that an increased neuromuscular activation inducing adaptations similar to resistance training (13,15,32). Suggesting, that specifically the Ia-afferent-mediated myotatic reflex contraction may be responsible for an increase in strength following WBV (32,88). When applying vibration directly to the muscle it has been speculated that tonic vibration reflex is induced (18,93). It seems possible that enhanced performance may be a result of

postactivation potentiation (PAP) caused by acute exposure of WBV. PAP is a condition that in which an exercise or modality is performed prior to performance to increase motor neuron excitability or phosphorylation of myosin light chains (35,48,77,106), enhancing performance. This motor neuron excitability has been measured indirectly with the H-reflex following WBV and had varying effects between individuals (7).

In clinical pain populations some potential mechanisms have been suggested that WBV inhibits pain receptors, allowing for individuals to be more tolerant to pain (87). It has also been suggested that vibration may have influence the activation of afferent input from sensory units in the muscle fibers and attenuated pain sensation associated with exercise or increased lymphatic blood flow and the removal of metabolic wastes (36,65,67). More research is warranted to determine mechanism(s) of the effects of WBV.

Performance

Enhancing performance in athletes and recreationally trained individuals has become increasingly important. Traditional techniques to train for sport performance are still prevalent (63,64,81) but an increasing number of options have been identified. Traditional training techniques such as strength training, plyometrics, and weightlifting may benefit from the inclusion of non-traditional techniques to further enhance performance (21,28,31,94).

One of the more recent nontraditional techniques is WBV, which has been shown to increase performance in upper and lower body muscular activity in both trained and untrained populations (12,17,25,26,29,34,54). Current research has shown that WBV

exposure at a moderate intensity is safe and effective in stimulating the neuromuscular system (21) and has been shown to induce non-voluntary muscle contractions (52), which may be beneficial in sport performance. WBV has also shown an increase in power production by facilitation of an explosive strength effort (12,53,82) leading to enhancement of performance via muscular strength and motor function (13,14). Also, sprinting and jumping performance has been shown to increase after bouts of WBV (3,11,17,28,30). This enhancement of acute performance is accomplished with little or no effort by the subject (88). In contrast, WBV has also been shown not to increase performance but to only have similar effects as traditional techniques (26,28,29,31,61).

WBV is increasingly being utilized as a warm-up for its potentiating effect prior to performance. Warm-up prior to performance is often recommended in order to prevent injury and to prepare the body for activity. WBV has been used as active passive warm-up instead of traditional active warm-up methods (27,28,88) due to its reported acute performance effects. The acute lower body neuromuscular activation from WBV (1,3) may be beneficial in many power sports.

WBV exposure variables such as frequency, amplitude, duration, and rest intervals need to be considered to optimize performance. Rest intervals following WBV have been shown to effect performance outcomes with too short a rest possibly over stimulating the neuromuscular system and too long a rest maybe allowing any effect to dissipate(3). Therefore, optimal rest intervals are crucial to the utilization of WBV to enhance sport performance. Previously researched, rest intervals following acute bouts of WBV have been used from immediately post to 10mins (3,11,17,26,28,71,95) and have demonstrated conflicting results. It has been shown that individuals optimize

performance at different rest individuals and are significantly greater than no WBV exposure (30).

Pain

WBV has also been researched in assisting with symptoms of exercise-induced pain. It has been shown that vibration applied to an unexercised muscle reduces the perceived level of pain from local pressure (112,113) with also showing reduced pain during muscle vibration in individuals suffering from muscle pain (69,112). These finding are consistent in supporting the gate control hypothesis (74), stating that afferent signals that are mediated by large myelinated fibers inhibit small pain fibers presynaptically in the dorsal horn of the spinal cord. However, it has been shown that after DOMS has set in (24hours)(57) perceived pain from local pressure increased with vibration and authors suggest it sensitizes nociceptors to the point where they become vibration responsive (74,112). The literature is sparse and conflicting on findings involving vibration and alleviation of pain, which warrants further investigation.

Whole Body Vibration and Muscle Recovery

WBV has recently been investigated as a muscle recovery modality following or prior to exercise induced muscle injury (4,9,10,16,65,87). As previously discussed there are a variety of measurements to assess exercise induced muscle injury, which can be utilized in determining the time course of muscle recovery. It has been suggested that WBV increases muscle spindle activity and muscle preactivation (lower firing threshold), which results in less disruption to excitation-contraction coupling (4,10,65). With an increase in muscle preactivation, theoretically a greater number of motor units and

muscle fibers would be recruited, which could lead to a reduction in myofibrillar stress during repeated muscle contractions, accelerating muscle recovery (14).

In a study with elbow flexors, investigators found that vibration treatment was effective for attenuation of DOMS, showing a decrease in soreness both over a time course of 7 days and acutely before and after vibration treatment. Researchers also found that vibration treatment was effective in ROM measurement, showing an increase in ROM over a time course of 7days. However, they did not find any effects on swelling, recovery of muscle strength and CK activity (65). In a few other studies, there were similar findings, lower perceived pain in the vibration treatment group compared to the control group were found (9,16,87), suggesting that WBV inhibited pain receptors and possibly stimulating blood flow to the musculature by increasing disposal of metabolic waste (36,67). There have been a few studies that administered the vibration treatment prior to the exercise induced muscle injury; these studies showed a decrease in soreness, isokinetic force, PPT, and plasma CK activity in the vibration treatment groups (4,10). Administering the WBV treatment prior to exercise induced muscle injury acts more as a protective mechanism, heightening sensitivity to the musculature, allowing for less amount of damage to occur (4). These studies have shown positive effects of WBV treatment with muscle recovery, however, more functional measures are needed to further investigate the use of WBV as a recovery modality. More specifically, effects on performance outcomes, forces and muscle activity would enable more understanding of mechanisms involving WBV.

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CHAPTER III

MANUSCRIPT 1

EFFECTS OF WHOLE BODY VIBRATION ON PAIN SENSIVITY FOLLOWING EXERCISE INDUCED MUSCLE DAMAGE

INTRODUCTION

Repeated eccentric muscle contractions have been shown to cause exercise induced muscle damage (EIMD) resulting in decreased force production (2,25). This muscle damage is evident as a disruption of the normal alignment of the skeletal muscle and disruption of the z-lines of sarcomeres (17,28). EMID presents symptoms such as tenderness, delayed onset muscle pain/soreness (DOMS), edema, and muscle stiffness (18,23). Pain and edema are thought to result from inflammation and the production of prostaglandin E_2 which sensitizes type III and IV afferent nocieptors (9,17). DOMS has been reported as an undesirable side effect of exercise due to its painful and debilitating effects on an individuals (27).

Previous research has studied several ways to control or prevent exercise induced muscle damage symptoms (8). Decreasing these symptoms in individuals is critical in many populations. In physically active individuals, decreasing swelling, stiffness and pain may allow for a quicker return to activity, potentially increasing specific performance measures over time. In clinically diagnosed pain individuals, decreasing muscle pain for any period of time is helpful for pain management and enabling activities of daily living. Most current modalities have not shown to be consistently effective in the treatment of symptoms, making it difficult to treat these individuals These treatment modalities include, but are not limited to massage, cryotherapy, stretching, homeopathy, ultrasound, and electrical current (8).

Whole-body vibration (WBV) is a mechanical stimulus elicited by oscillatory motions. These oscillatory motions are determined by the frequency and amplitude of the

vibration exposure. While the mechanism(s) remain unclear, exposure to WBV has been shown to increase neuromuscular activity with many positive results such as: muscle flexibility (16), strength (14), performance (10,11) and power output (5). Whole-body vibration has also been researched in assisting with symptoms of exercise-induced pain. Previous literature suggests that WBV increases muscle spindle activity and muscle pre-activation, which results in less disruption of excitation-contraction coupling (1,4,20), shown when WBV exposure was given prior to EMID, allowing less reduction in force compared to no vibration. It has been suggested that an increase in muscle pre-activation, theoretically increasing the number of motor units and muscle fibers recruited, could lead to an increased muscle recovery by decreasing myofibril stress during repeated muscle actions (6). This indicates that a decreased amount of force loss may occur following exercise induced muscle damage when WBV is utilized. It has also been suggested that WBV increases blood flow to the musculature (4), an increased blood flow to the muscle could increase removal of waste and delivery of nutrients, accelerating repair and remodeling in the muscle (13). Another proposed mechanisms in some clinical populations suggest that WBV inhibits pain receptors, allowing for a higher pain tolerance in patients with chronic pain (26). It is proposed that vibration receptors in the skin stimulate inhibitory interneuron's in the spinal cord, which in turn act to reduce the amount of pain signals transmitted (24). It was been suggested that gate control theory for pain perception and inhibition with vibration, has been suggested that vibration would shut down the pain message gate to the spinal column and brain and would be expected to increase pain threshold (24).

There have been conflicting findings in the literature involving muscle pain, pressure pain threshold (PPT), range of motion (ROM), and limb circumference measures (1,3,4,7,20,26,29) when utilizing WBV prior or following exercise induced muscle damage. A few studies have examined using WBV prior to EIMD, resulting in a less reduction in force (1,4), while others have examined vibration following EIMD as a recovery modality (3,7,20,26,29). Timing of when WBV is applied may have fundamental differences in the purpose of the WBV and the effect on EIMD symptoms. The type of vibration (direct or whole body) may contribute to the outcome of the applicability of using vibration as a treatment and each may contribute to specific populations. Thus, indicating inconclusive findings involving WBV and exercise induced muscle damage. The purpose of this investigation was to determine if WBV aids in managing symptoms of exercise induced muscle damage over a recovery period of 72 hours.

MATERIALS AND METHODS

Participants

Thirty recreationally trained females (age 21± 1.9yrs, height 165.69 ± 7.3cm, mass 58.69 ± 10.95kg) volunteered to participate in a 7-session protocol that was approved by the University's Institutional Review Board. Any participant with a recent history of lower body musculoskeletal or orthopedic injury or taking any medications that alter balance, musculoskeletal system, or central nervous system functions relating to posture and motor control were excluded from participating. Individuals taking prescription pain and/or psychiatric medications were also excluded. In addition, participants were screened by questionaire for potential risk factors to the exercise protocol (i.e. rhabdomyolysis, brusing easily, etc.). Partipants were asked to not perform any lower body exercise or take any pain medications 48hours prior to testing sessions and during all testing days and to keep all food and water intake consistent during testing sessions. Furthermore, partipants were scheduled to not be testing during menstral cycle to avoid failure to comply with above instructions.

Measures

Pressure Pain Threshold

Pressure pain threshold (PPT) was assessed in all 7 visits to the laboratory. Pain threshold was assessed in the left quadriceps while participants were seated comfortably on a padded table. A mark was placed on the rectus femoris at the mid-point between the patella and the proximal head of the femur (the mid-point between the knee and the hip),

as well as on the belly of the vastus medialis and vastus lateralis on the left thigh. Throughout the test participants were instructed to keep their quadriceps relaxed. The researcher placed a pressure algometer (Wagner Instruments, Greenwhich, CT USA) on each separate test site, and mechanical pressure was applied to the muscle in the following order: VM, VL, and RF. Three trials (VM, VL, RF) were performed with 20s between each trial. Participants were asked to indicate when the pressure transitioned from being "uncomfortable" to "faintly painful". The participant indicated this by saying "pain", and subsequently the researcher immediately removed the pressure stimulus. The corresponding force value was recorded. All three trials for each muscle were averaged for each participant.

Range of Motion

Range of motion (ROM) was measured to assess stiffness and mobility in the knee flexors during active and passive ROM with a goniometer. For reference, the mobile arm was placed along the lower leg and the fixed arm was placed along the upper leg; fully extended was defined as 0 degrees. Participants were placed in the prone position on a padded table. During active ROM measurements participants were asked to flex their right knee as much as possible. During passive ROM measurements, participants were asked to relax the knee flexors and the researcher passively flexed their right leg. If at any point it became painful in the musculature, participants were instructed say "pain" and measurements were stopped. If no pain was expressed, researchers stopped at the point of no further flexion.

Thigh Circumference

Thigh circumference was measured to assess localized inflammation to the right quadriceps at the distal end and mid point of the quadricep. The distal end was identified as the belly of the vastus medialis and the mid point of the quadriceps was identified by the mid-point between the anterior super iliac spine and the patella. Three measures were taken using a Gulick measuring tape (Ann Arbor, MI, USA) in centimeters at each site and then averaged.

Muscle Pain During Movement

To assess DOMS, participants were asked to rate the intensity of the pain/hurt/soreness in their quadriceps during a body weight squat. A 10cm visual analog scale (VAS) was used to assess soreness. Participants were instructed to place a mark along the 10cm line that corresponded to the intensity of pain experienced during the squat. Anchors of "no pain" and "worst pain imaginable" were placed on the left and right end of the 10cm line, respectively.

Experimental Procedures

Participants came into the laboratory for three familiarization sessions prior to testing days, which included informed consent, anthropometrics, and familiarization with all protocols. Following three familiarization sessions, participants visited the laboratory for 4 consecutive days and were randomly assigned to the control or treatment (WBV) group. All participants were assessed for baseline PPT's, ROM, thigh circumference and muscle pain on movement in the quadriceps. After baseline measures were taken, participants performed an exercise induced muscle damage protocol, which consisted of split squats using a Jones Machine® by performing 4 sets to task failure on each leg with

a one-minute rest between sets. The Jones Machine® was front loaded with 40% of each participants body weight. During split squats, the back leg was placed on a bench for support with 90-degrees of flexion, allowing focus on single leg performance of the front leg. Researchers provided assistance on the concentric phase after the participants reached 90 degrees of flexion of the front knee on the exercising leg, allowing greater focus on the eccentric phase.

Immediately following the muscle damage protocol, participants in the control group performed 2 sets of body weight quarter squats on a flat surface for a 30s 1:1 work to rest ratio. Participants in the whole body vibration (WBV) group performed 2 sets of body weight quarter squats on the vibration plate. An AIRdaptive (Power Plate, Inc.) system was utilized for tri-axial vibration exposure. Vibration frequency was set at 30Hz with an amplitude of 2-4mm. Following treatment/control, participants were assessed for PPT's, ROM, thigh circumference and muscle pain on movement in the quadriceps. Participants then rested for 10min and all measures were reassessed. Participants were then asked to adhere to the restrictions of the study previously mentioned and to refrain from any other treatments (i.e. icing, stretching, heating).

Participants returned to the laboratory 24, 48, and 72 following muscle damage protocol to evaluate muscle pain on movement, PPT's, ROM, and thigh circumference. These sessions consisted of initial assessment of PPT's, ROM, thigh circumference and muscle pain on movement in the quadriceps followed immediately by treatment/control protocol. After treatment/control, measures were reassessed followed by 10min rest followed by a third set of measurements.

Reliability of the Measurements

Three days of measurements were obtained during familiarization sessions and a set of baseline measures on the first testing day of rectus femoris PPT. Measurement of reliability were quantified through the calculation of the intraclass correlation coefficient (ICC) with a 95% confidence interval. The ICC values over the four measurements for rectus femoris PPT were .92, respectively.

Data Analyses

To test changes in PPT's, ROM, thigh circumference, and muscle pain over time and between groups, a 12x2 (time by group) mixed factor analysis of variance (ANOVA) was conducted; time being 0Pre, 0Post1, 0Post2, 24Pre, 24Post1, 24Post2, 48Pre, 48Post1, 48Post2, 72Pre, 72Post1, and 72Post2 and group being control and WBV. If interactions occurred they were followed up with a one-way ANOVA's, if main effects were observed in the absence of an interaction they were followed up with a least significant difference (LSD) post-hoc analyses for pairwise difference.

All analyses were conducted using SPSS software (SPSS, Inc., Chicago, IL), when sphericity was violated; the Greenhouse-Geisser correction of degrees of freedom was used. Statistical significance was defined as p-value less than 0.05 and eta squared was calculated to determine effect sizes.

RESULTS

Pressure Pain Threshold

Vastus Medialis. No significant (F=1.16, P=0.33, η^2=.04) interaction of time by group was found for PPT in the VM. There was a significant main effect for time (F=5.62, P<0.001, η^2=.17) (Figure 1) but no significant (F=3.3, P=0.07, η^2=.11) main effect for group. VM PPT's significant main effects for time were that 0Pre and 0Post1 was greater at time points 24Pre and beyond; 0Post2 was greater than 24Pre-48Pre. (Figure Ia)

Vastus Lateralis. No significant (F= 2.1, P=0.07, η^2= .07) interactions of time by group was found for PPT in the VL. There was a significant (F= 7.05, P<0.001, η^2= .20) main for time but no significant (F= 2.30, P=0.14, η^2= .07) main effects for group. VL PPT's significant main effects for time were that 0Pre and 0Post1 is greater than 24Pre-48Post2; 0Post2 is greater than 24Pre, 24Post1, and 48Pre; 24Pre is less than 24Post2; 24Pre-48Post2 is less than 72Pre-72Post2 time points. (Figure Ib)

Rectus Femoris. No significant (F= 1.78, P=0.12, η^2= .06) interaction of time by group was found for PPT in the RF. There was a significant main effect for time (F= 4.09, P=0.002, η^2= .13) (Figure 3) but no significant main effect for group (F=2.21, P=0.14, η^2=.07) . RF PPT's significant main effects for time were that 0Pre and 0Post2 was greater than 24Pre and 48Pre; 0Post1 is greater than 24Pre, 24Post2-48Post1; 24Pre is less than 24Post1, 24Post2, 48Post2-72Post2; 24Post2-48Post1 is less than 72Pre-72Post2. (Figure Ic)

Figure Ia.

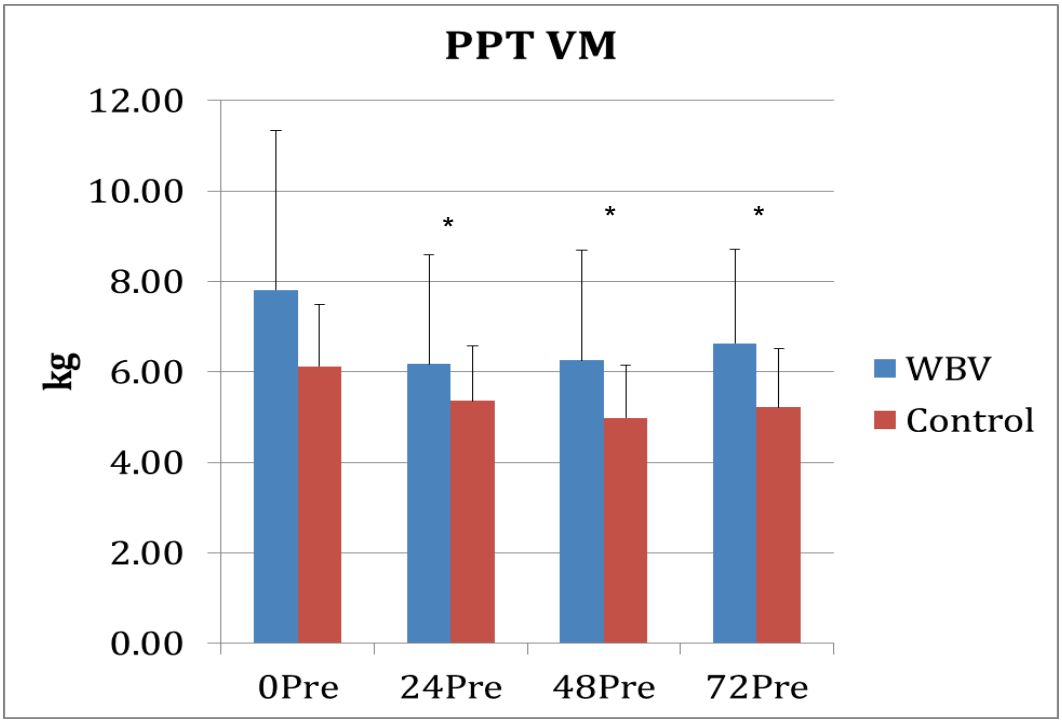

Figure Ib.

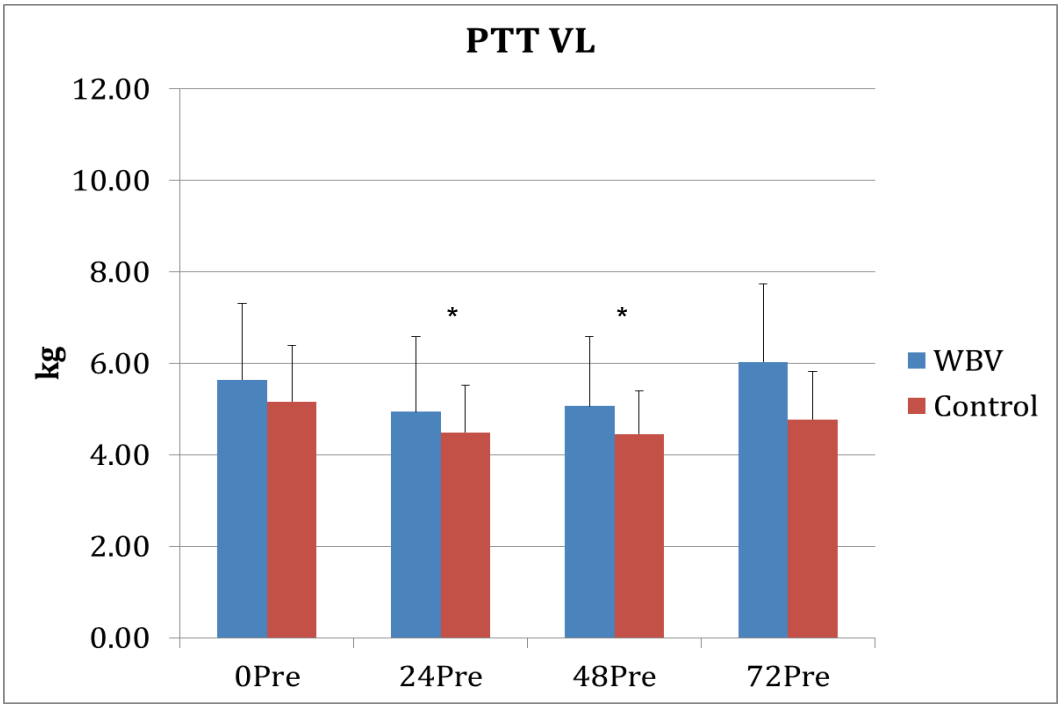

Figure Ic.

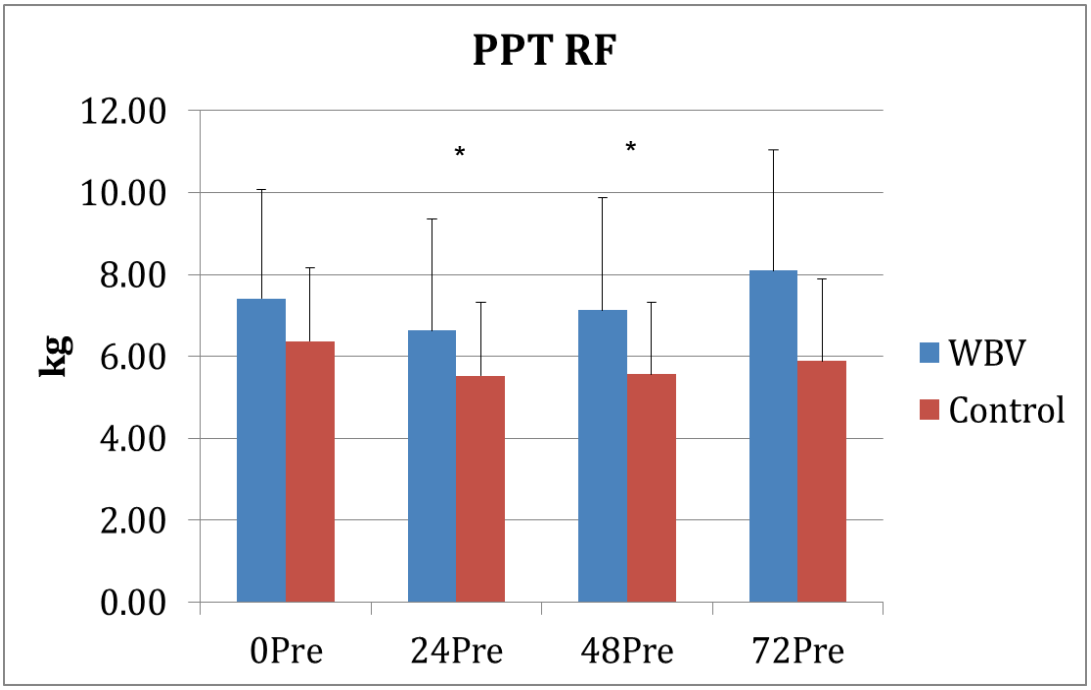

Figure I a-c. Means ± SDs for PPT between groups and across all time points following exercise induced muscle damage. Significant ($p<0.05$) main effects from 0Pre are indicated with *.

Range of Motion

Significant (F= 2.66, P=0.01, η^2= .08) interactions of time by group were found for aROM. This was followed up with a 12x1 repeated measures ANOVA for each group. Significant (F= 3.37, P<0.001, η^2= .18) main effect for WBV group was found for time. With 0Pre greater than 0Post1-48Post2; 24Pre is less than 24Post1; 24Pre, 24Post2, 48Pre, 48Post2 is less than 72Pre and 72Post2 in WBV group (Table 1). Significant (F= 2.54, P=0.03, η^2= .16) main effect for control group was found for time. With 0Pre-24Post1 is less than 48Pre and 72Pre; 24Post2 is less than 48Pre in control group (Table 1). No significant (F= 1.13, P=0.34, η^2= .03) interaction of time by group was found for pROM. No significant main effects for time (F= 1.89, P=0.77, η^2= .06) or group (F= .17, P=0.67, η^2= .006) were found for pROM (Table Ia).

Thigh Circumference

No significant (F= 1.95, P=0.15, η^2= .06) interaction of time by group was found for distal thigh circumference. No significant main effects for time (F= 1.44, P=0.24, η^2= .04) or group (F= 2.49, P=0.12, η^2= .08) were found for distal thigh circumference (Table 1). No significant (F= 1.61, P=0.21, η^2= .05) interaction of time by group was found for mid thigh circumference. No significant main effects for time (F= 1.40, P=0.25, η^2= .04) or group (F= .46, P=0.5, η^2= .01) were found for mid thigh circumference (Table Ia).

Dynamic Muscle Pain

No significant (F=.38, P=0.81, η^2=.014) interaction of time by group was found for muscle pain in the quadriceps. There was a significant main effect for time (F=44.93, P<0.001, η^2=.616) but no significant (F=.05, P=0.82, η^2=.002) main effect for group. Muscle pain main effects for time were that 0Pre is less than all other time points. 0Post1 and 0Post2 was less than time points 24Pre-72Post2; 24Pre- 24Post2 was less than 48Pre-72Post2; 48Pre-48Post2 is greater than 72Pre-Post2. (Table Ia)

Table Ia.

Table Ia. Means and SDs for active and passive ROM, thigh circumference of the VM and RF and muscle pain ratings between groups and across 72hours from exercise induced muscle damage.

	0Pre	0Post1	0Post2	24Pre	24Post1	24Post2	48Pre	48Post1	48Post2	72Pre	72Post1	72Post2
ROM **												
aROM												
WBV	130.62 ± 4.30	128.93 ± 3.49 #	128.06 ± 4.34 #	127.25 ± 4.37 #	128.75 ± 4.17 #	127.75 ± 4.94 #	127.31 ± 4.92 #	128.43 ± 4.76 #	127.93 ± 5.20 #	129.93 ± 4.10	128.93 ± 4.97	129.5 ± 4.64
Control	130.71 ± 6.98	129.71 ± 7.12	129.85 ± 6.52	130.07 ± 7.26 #	129.85 ± 7.09	130.64 ± 5.13	132.78 ± 6.11 #	131.42 ± 6.23	130.85 ± 5.92	132.21 ± 7.17	132.21 ± 7.12	131.35 ± 5.94
pROM												
WBV	145 ± 5.44	142.62 ± 5.08	142.18 ± 5.31	143.62 ± 5.71	143.75 ± 4.78	143.06 ± 6.04	142.81 ± 7.05	143.62 ± 5.18	142.5 ± 5.57	144.62 ± 4.60	143.25 ± 5.20	144.87 ± 5.18
Control	142.85 ± 9.04	142.64 ± 7.42	143.14 ± 7.89	144.14 ± 7.54	145.28 ± 5.86	144.35 ± 6.91	144.35 ± 6.27	143.78 ± 5.88	144.14 ± 5.84	145.28 ± 6.08	146.35 ± 5.58	145.21 ± 5.51
Thigh Circumference												
Distal Thigh												
WBV	39.31 ± 3.48	39.64 ± 3.43	39.51 ± 3.51	39.42 ± 3.41	39.61 ± 3.34	39.58 ± 3.28	40.75 ± 5.56	39.84 ± 3.30	39.98 ± 3.43	39.55 ± 3.29	39.52 ± 3.33	39.51 ± 3.46
Control	41.46 ± 3.37	41.82 ± 3.12	41.65 ± 3.24	41.72 ± 3.32	41.76 ± 3.43	41.77 ± 3.45	41.54 ± 3.42	41.60 ± 3.31	41.61 ± 3.46	41.73 ± 3.38	41.69 ± 3.49	41.66 ± 3.71
Mid-Thigh												
WBV	52.20 ± 5.13	52.42 ± 5.29	52.31 ± 5.40	52.27 ± 5.27	52.03 ± 4.99	52.07 ± 4.75	52.31 ± 5.19	52.33 ± 5.07	52.20 ± 5.05	51.97 ± 4.89	52.02 ± 4.96	51.83 ± 4.84
Control	53.19 ± 3.45	53.55 ± 3.24	53.41 ± 3.09	53.34 ± 3.55	53.35 ± 3.36	53.36 ± 3.42	52.14 ± 5.75	53.38 ± 3.38	53.44 ± 3.43	53.41 ± 3.47	53.30 ± 3.53	53.16 ± 3.52
Pain on movement												
WBV	3.56 ± 5.33	22.28 ± 15.06 *	22.68 ± 12.04 *	43.68 ± 19.36 *	41.75 ± 21.25 *	43.75 ± 22.06 *	53.12 ± 19.63 *	48.62 ± 20.24 *	48.56 ± 17.96 *	34.43 ± 19.19 *	34.12 ± 19.82 *	31.87 ± 20.37
Control	4.42 ± 8.61	21.14 ± 13.35	22.35 ±14.10	39.21 ± 15.22	40.5 ± 17.37	38.78 ± 18.20	47.5 ± 23.62	49.92 ± 22.97	50 ± 24.88	33.14 ± 16.84	32.92 ± 19.08	33.57 ± 19.29

* Indicate significant main effects for time from 0Pre ($p<0.05$).** Indicate group by time interaction. # Indicate simple comparison to 0Pre.

DISCUSSION

The current study investigated the possible effects of WBV as a pain management and function modality following exercise induced muscle damage. This investigation found that four sets to failure split squats successfully induced muscle pain during movement and increased pain sensitivity to pressure stimuli. Whole-body vibration had no effects either acutely or on the day-to-day progression of symptoms, thus indicating that WBV was not effective in aiding in pain management in this study.

In clinical pain populations some potential mechanisms have been suggested that WBV inhibits pain receptors, allowing for individuals to be more tolerant to pain (26). It is proposed that vibration receptors in the skin stimulate inhibitory interneurons in the spinal cord, which in turn act to reduce the amount of pain signals transmitted to the brain (24). In gate control theory, pain perception and inhibition via vibration has been suggested to occur by vibration gating the afferent signal from nociceptors to the spinal column and brain, increasing pain threshold (24). It has been shown that vibration applied to an unexercised muscle reduces the perceived level of pain from local pressure (29,30) while also showing reduced pain during muscle vibration in individuals suffering from chronic muscle pain (22,29), supporting the gate control hypothesis (24). However, it has been shown that when DOMS is present (24hours) (19), perceived pain from local pressure increased with vibration. The authors suggest this was due to sensitization of nociceptors to the point where they become vibration responsive (24,29). In contrast to

these findings, the current investigation, no differences in muscle pain when WBV was applied were found.

The changes in muscle pain ratings during movement and PPT's observed in the present study are consistent with previous literature following exercise induced muscle damage (1,3,12,20,26). Some research shows group difference from WBV and control groups in muscle pain (1,20,26) indicating that WBV aids in reducing muscle pain after exercise induced muscle damage. Muscle damage protocols varied in these studies, some used 6 sets of 10 repetitions of eccentric only exercises on a isokinetic dynometer (1,20) in the elbow felxors (20) and knee flexors (1). Whereas, another study used a combination of resistance training, running and sprints to induced muscle damage (26). These studies also used different forms of vibration, Lau et. al used direct vibration from a handheld device (20) whereas, the other studies used WBV platforms (1,26). In the current investigation knee flexors were used during a lower body resistance training exercise with WBV platform, which may account for the difference in findings. These differences are important to note, since upper and lower body musculature may respond differently and different exposures of vibration may elicit different responses as well. Some research has shown a decrease in muscle pain rating they also showed that PPT's were not different with vibration treatment (7,20), which is consistent with the current investigations findings. The present finding that showed no differences in limb circumference between control and vibration groups and over time was consistent with previous research (1,20) following an eccentric only damage protocol. However, the findings of no differences in ROM between groups and over time is inconsistent with previous research that shows an faster increase to baseline in ROM in vibration group

(20). Conflicting results may be due to position in which ROM was measured and the stiffness and inflammation in the muscle may account for some difference in the present study.

It has also been suggested that vibration may influence the activation of afferent input from sensory units in the muscle fibers and attenuated pain sensation associated with exercise or increased lymphatic blood flow and the removal of metabolic wastes (15,20,21). An increase in blood flow to the musculature during WBV has been shown to occur (4), and indicates a removal of metabolic waste and increased nutrient delivery, accelerating repair and remodeling in the muscle (13). However, since this investigation did not measure muscle temperature and found no differences in exercise induced muscle damage symptoms between groups, the present investigators cannot conclude that WBV increased metabolic waste removal occurred in this investigation.

Previous research has studied several ways to control or prevent exercise induced muscle damage symptoms (8). Decreasing these symptoms in individuals is critical in many populations. In exercising, physically active individuals, decreasing swelling, stiffness and pain will allow for a quicker return to activity. In clinical pain individuals, decreasing muscle pain for any period of time is helpful for pain management and enabling activities of daily living. It may be plausible that WBV may be more effective for a generally healthy recreational individuals and direct vibration maybe more effect for clinically pain or injured individuals, however this has not yet been identified in the literature. Most current modalities have not been shown to be consistently effective, making it difficult to treat individuals with muscle pain, swelling, and stiffness. These include, but are not limited to massage, cryotherapy, stretching, homeopathy, ultrasound,

and electrical current (8). Recently, WBV has been explored as a potential modality in treating symptoms associated with exercise induced muscle damage. It is important to note that timing of when vibration is utilized may contribute to different findings in the literature, whether this be prior to muscle damage or after for treatment and needs to be further investigated. The literature is sparse and conflicting on findings involving vibration and alleviation muscle pain during movement. The current study contributes to the body of literature in this area and further research is warranted.

The present investigation provides a novel exercise in producing exercise induced muscle damage in the quadriceps that to our knowledge has previously not been established. As well, investigating muscle pain during movement, PPT's, ROM, and circumference in recreationally trained individuals on the lower body effects of alleviating pain with WBV has no previously been done. The research is consistent with other investigations supporting that our participants did experience exercise induced muscle damage in the quadriceps, allowing us to be confident that our findings with WBV exposure does not effectively aid in muscle pain management in health recreationally trained females. Future research should investigate a variety of populations (i.e. chronic and acute pain patients, recreationally trained males, and athletically trained population) for treatments in alleviating muscle pain.

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CHAPTER IV

MANUSCRIPT 2

EFFECTS OF WHOLE BODY VIBRATION ON VERTICAL JUMP PERFORMANCE FOLLOWING EXERCISE INDUCED MUSCLE DAMAGE IN WOMEN

INTRODUCTION

Resistance training is a common exercise and training tool to increase muscular strength, hypertrophy and endurance. While resistance training is essential for enhancing performance, it also causes temporary debilitations due to exercise induced muscle damage (EIMD). Delayed onset muscle soreness (DOMS) has been identified as painful and an undesirable side effects for individuals (37). Peak DOMS usually occurs 24 to 48 hours following exercise (23,29). EIMD presents as tenderness, pain, swelling, and muscle stiffness (29). It has been suggested that these symptoms are related to an inflammatory process based on a lack of evidence of neural inhibition of damaged muscle (29) or changes in motor unit activation (36). It has been well documented that EIMD occurs from repeated eccentric muscle actions, resulting in decreased force production (4,31). Evidence of disruption of the normal alignment of skeletal muscle and disruption of the z-lines of sarcomeres has been seen in damaged musculature (22,38). Muscle soreness has been shown to occur initially from the inflammatory process with production of prostaglandin E_2 which sensitizes type III and IV afferent fibers of muscle connective tissue, which are highly correlated with DOMS (18), observed at 24, 48 and 72 hours (22).

During most performance activities, the main goal is to maximize power output, however, with exercise induced muscle damage, power generation may be compromised. It has been shown that peak power output is immediately reduced following eccentric muscle actions in the knee extensors during isokinetic cycling (34) and a Wingate cycle test (14) while continuing to be reduced up to 2 days post injury. A decrease in power

output has also been shown during intermittent maximal sprints on a cycle ergometer after 10 sets of 10 plyometric jumps to induce damage (39). Vertical jump performance is related peak power output and could be compromised following exercise induced muscle damage. Studies have found a prolonged reduction in maximal force production, EMG activity, ground reaction forces, stretch reflex sensitivity, muscle and joint stiffness regulation and the stretch shortening cycle (5,25) following EIMD; which all play a role in jumping performance. Vertical jump performance with and without a countermovment have been shown to have immediate and long-lasting reductions in performance up to 4 days post injury but are dependant on jump type (15). Squat jumps have the most prolonged reduction in jump height compared to countermovement jump sand depth jumps.

Eccentric exercises are commonly used as a component of strength-training programs and have been shown to elicit DOMS, potentially causing reduction in sport performance. Previous researchers have studied several ways to control or prevent symptoms of EIMD (16). Most current modalities have not been shown to be consistently effective. These include, but are not limited to massage, cryotherapy, stretching, homeopathy, ultrasound, and electrical current (16). Recently, WBV has been suggested as a novel modality to reduce or control symptoms of EIMD (2,8,32). Bakhtiary et al. 2007 found that vibration prior to eccentric loading may prevent and control DOMS with possible mechanisms of increased blood flow to facilitate recovery and muscle regeneration and possible pain inhibition (8). Rhea et al. 2009, implemented WBV in combination with stretching and massage after strenuous exercise over a period of 72 hours, showing decreased pain perception in the WBV group (32). Aminan-Far et al.

2011 also showed a reduction in EIMD symptoms and maximal isometric and isokinetic voluntary strength loss, creatine kinase, pain threshold and muscle soreness with WBV performed prior to eccentric exercises (2). However, to our knowledge, no study has investigated the effects of WBV following exercise induced muscle damage in vertical jump performance measures. Therefore, the purpose of this investigation was to determine the effects of WBV on jumping performance following exercise induced muscle damage.

METHODS

Experimental Approach to the Problem

The aim of the current study was to investigate acute and chronic effects of WBV exposure on jumping performance following exercise-induced muscle damage. Therefore, this study used a mixed factor design testing a control and WBV group's vertical jumping performance prior to muscle damage and 3 days after.

Participants

Twenty-seven recreationally trained females (age 21 ± 2 yrs, height 172.38 ± 92.27 cm, mass 58.67 ± 11.53kg) volunteered to participate in a 7 session protocol and gave and signed informed consent that was approved by the University's Institutional Review Board. Recreationally trained individuals were defined as meeting American College of Sports Medicine recommendations for healthy living and did not exceed 5 lower body workouts a week on a regular basis in the last 6 months. Participant with a recent history of lower body musculoskeletal or orthopedic injury or taking medications that alter balance, musculoskeletal system, or central nervous system functions relating to posture and motor control were excluded from participating. Additionally, individuals taking prescription pain and/or psychiatric medications were excluded. All participants were screened by questionaire for potential risk factors to the exercise protocol (i.e. rhabdomyolysis, bruising easily, etc.). Partipants were asked to not perform any lower body exercise or take any pain medications 48hours prior to testing sessions and during all testing days and to keep all food and water intake consistent during testing sessions.

Furthermore, partipants were not scheduled for testing during their menstral cycle in order to avoid non-compliance with the instructions above.

Measures

Vertical Jump

Vertical jump performance was assessed on each visit to the laboratory using a combination of a Vertec® (Sports Imports,Columbus, OH, USA) free standing jump height measurement device and a Bertec® (Bertec Corp. Columbus, OH, USA) force platform sampling at 1080Hz. Participants were instructed to perform three maximal countermovement vertical jumps (CMVJ; 15s rest), with arm swing and were instructed to jump as quickly and high as possible. The Vertec was used as a visual target where participants could hit tabs indicating jump height. Vertical jump height was calculated by the difference between standing reach and maximum jump reach while peak power output (PPO) was calculated via the Sayers Equation (35). Relative peak ground reaction force (rGRF) was calculated from peak z-force (prior to landing) divided by body weight (Newtons/kg). Rate of force development (RFD) was derived from ΔForce/ΔTime over the first 200ms of the concentric phase of the vertical jump, beginning when force returned to body weight.

Electromygraphy

Bipolar surface electromyography (EMG) was recorded during maximal isometric voluntary contraction (MVIC) and maximal vertical jump on the left leg during each testing visit. Noraxon single electrodes (Noraxon USA Inc., Scottsdale, AZ, USA) were placed 3-5cm apart with a ground electrode on the head of the tibia. Proper skin

preparation included abrasion of the skin around the electrode site followed by cleansing with an alcohol swab. Data were recorded from the left vastus medialis (VM) using a Noraxon Telemyo 8-channel EMG system (Noraxon USA Inc., Scottsdale, AZ, USA) with a hardware band pass filter (10-500Hz). During isometric MVIC, EMG signals were recorded for 5s on a modified knee-extension/leg curl machine (Body Solid; model GLCE-365; Forest Park, IL). Participants were seated with the hip at 90º of flexion and with the knee fixed in a flexed position at a 60º angle below horizontal. A strap was used to secure their left ankle to the lever arm. Ratio of vertical jump peak EMG was performed by dividing vertical jump peak EMG by MVIC peak EMG.

DOMS Visual Analog Scale

To assess soreness in the quadriceps participants were asked to rate the intensity of their pain/hurt/soreness in their quadriceps during a body weight squat. A 100mm visual analog scale (VAS) was used to assess soreness. Participants were instructed to place a mark along the 100mm line that corresponded to their intensity of pain. Anchors of "no pain" and "worst pain imaginable" were placed on the left and right end of the 100mm line, respectively. Soreness was determined by measuring the interaction of the horizontal scale and the vertical line with a tape measure.

Procedures

During three familiarization sessions, participants read and signed informed consent, filled out screening questionnaires, were measured for anthropometrics performed all testing protocols. Following these familiarization sessions, participants visited the laboratory on 4 consecutive days and were randomly assigned to a control or

treatment (WBV) group. Prior to each pre-value measurement, all participants performed 2 sets of 15 meters of dynamic warm-ups including: jogs, gait swings, high knees, exaggerated lunges and Frankensteins. They were then assessed for pre-values on vertical jump performance, MVIC EMG and quadriceps muscle soreness. After pre-values were taken, they performed an exercise induced muscle damage protocol, which consisted of split squats using a Jones Machine® by performing 4 sets to momentary failure on each leg with a one-minute rest between sets. The Jones Machine® was front loaded with 40% of their body weight. During split squats, the rested back leg was placed on a bench for support with 90-degrees of flexion of the forward knee, allowing focus on single leg performance of the front leg. There was assistance on the concentric phase after they reached 90 degrees of flexion of the front knee on the exercising leg, allowing greater focus on the eccentric phase.

Immediately following the exercise induced muscle damage protocol, the control group performed 2 sets of body weight quarter squats on a flat surface for a 30s with a 1:1 work to rest ratio. The WBV group performed 2 sets of body weight quarter squats on the vibration plate. An AIRdaptive® (Power Plate, Inc.) system was utilized for tri-axial vibration exposure. Vibration frequency was set at 30Hz with an amplitude of 2-4mm. Following WBV or control, participants were assessed for vertical jump performance, MVIC and quadricep muscle soreness.

Participants returned to the laboratory 24, 48, and 72 hours following the exercise induced muscle damage protocol to evaluate jumping performance over time. These sessions consisted of a pre-value assessment of vertical jump performance, MVIC EMG

and muscle soreness in the quadriceps followed immediately by a WBV treatment or control protocol. After treatment/control, all measures were reassessed.

Reliability of the Measurements

Three days of measurements were obtained in familiarization sessions and a set of pre-values on the first testing day of vertical jump performance. Measurement reliability and precision were quantified through the intraclass correlation coefficient (ICC) with a 95% confidence interval. The ICC value for vertical jump was R=0.92.

Data Analyses

To test changes in VJH, PPO, RFD, rGRF, and EMG ratio over time and between treatment groups, we conducted a 2x8 (group x time) mixed factor analysis of variance (ANOVA). Groups were defined as WBV and control and time defined as Day0Pre, Day0Post, Day24Pre, Day24Post, Day48Pre, Day48Post, Day72Pre, and Day72 Post. If interactions occurred they were followed up with one-way ANOVAs while main effects were followed up with least significant difference (LSD) post-hoc analyses for pairwise differences. All analyses were conducted using SPSS software (SPSS 20, Inc., Chicago, IL). Statistical significance was determined as $P < 0.05$.

RESULTS

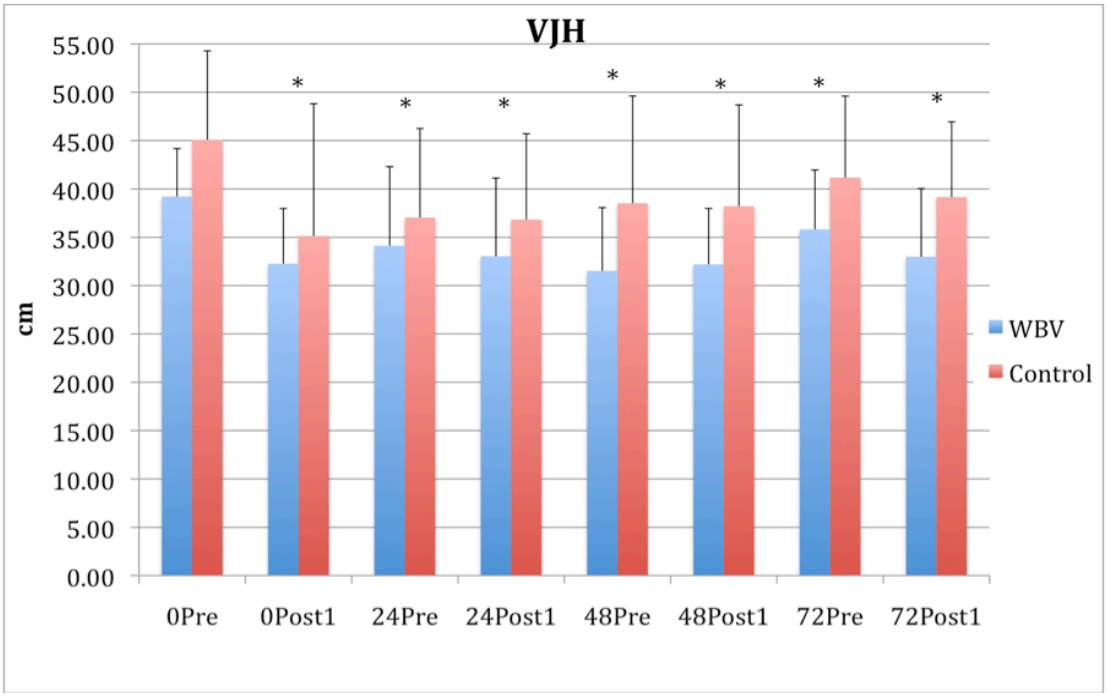
Vertical Jump

No significant ($p > 0.05$) interaction were found for either VJH or PPO. There was also no significant ($p > 0.05$) main effects for group but there were significant ($p < 0.001$) main effects for time. For VJH 0Pre was greater than all other time points and 72Pre was greater than 0Post, 24Pre, 24Post, 48Pre, 48Post, and 72 Post (Figure IIa). For PPO 0Pre was greater than all other time points and 72Pre was greater than 24Pre, 24Post, 48Pre, 48Post, and 72 Post (Table IIa).

No significant ($p > 0.05$) interaction was found for rGRF. There was a no significant ($p > 0.05$) main effect for group but there was a significant ($p < 0.001$) main effect for time. 0Pre was greater than all other time points, 48Pre was greater than 72Post, and 72Pre was greater than 72Post (Table IIa).

No significant ($p > 0.05$) interaction was found for RFD. There was no significant ($p > 0.05$) main effect for group but there was a significant ($p < 0.001$) main effect for time. 0Pre was greater than all other time points; 48Pre was less than 0Post, 24Pre, 24Post, 72Pre, and 72Post and 48Post were less than 72Pre and 72Post (Table IIa).

Figure IIa. Means and SDs for VJH between groups and across time following exercise induced muscle damage. Significant ($p<0.05$) differences from 0Pre are indicated with *.



EMG

No significant (p> 0.05) interactions or main effect for time were found for peak EMG ratio during the vertical jump. No significant (p> 0.05) main effect for group was found but a signficiant (p<0.05) main effect for time was found. With 0Pre being greater than 0Post1 and 24Pre (Table IIa)

Table IIa. The PPO, RFD, rGRF, and EMG ratio maximum values for each time point in each group.

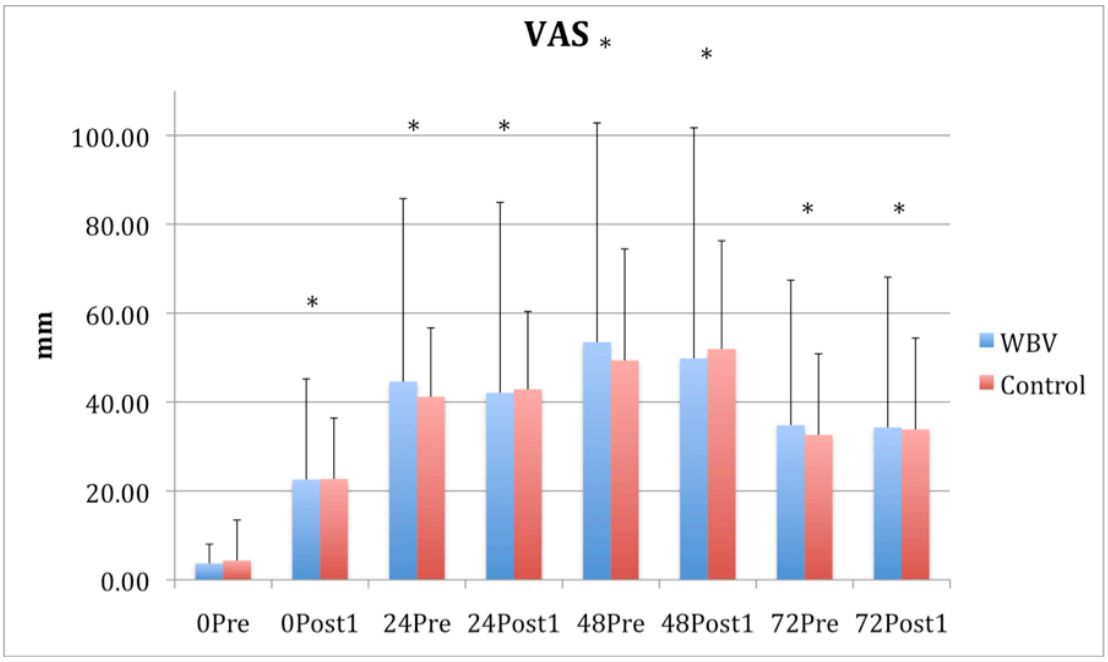
	0Pre		0Post1		24Pre		24Post1		48Pre		48Post1		72Pre		72Post1	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
PPO (W)																
WBV	2997.19	500.16	602.37	413.61	594.96	576.60	559.14	627.02	466.55	518.26	461.68	456.80	684.83	438.13	587.05	507.99
Control	3313.78	765.62	881.87	969.76 *	888.03	709.88 *	841.21	700.66 *	947.77	823.14 *	951.14	825.93 *	1131.02	685.02 *	1056.15	621.33 *
RFD (N/s)																
WBV	35290.56	9910.29	28309.99	12587.37 *	24571.26	11242.00 *	27657.40	11484.02 *	19666.22	9922.31 *	23030.32	11882.35 *	26144.95	14340.48 *	26360.40	12460.13 *
Control	39707.88	18315.46	26290.53	13245.30	27829.14	14039.02	26628.89	15136.31	24044.13	12000.85	26705.55	15809.33	33087.62	14155.71	31392.48	12979.35
rGRF (N/kg)																
WBV	239.21	27.72	219.63	29.18 *	217.54	35.37 *	209.30	30.20 *	220.99	31.48 *	218.86	34.02 *	219.63	20.81 *	211.20	26.85 *
Control	251.87	36.57	224.18	28.01	229.20	17.99	217.38	27.57	230.48	25.38	223.89	40.72	236.54	20.66	225.23	18.83
pEMG Ratio																
WBV	1.89	0.59	1.39	0.43 *	1.73	0.84 *	1.79	1.02	2.48	1.79	2.70	1.07	2.16	0.49	2.46	0.67
Control	3.03	2.69	1.35	0.55	2.13	0.82	1.83	1.25	2.46	1.20	2.22	0.88	2.02	0.69	1.95	0.93

PPO=Peak power output; RFD= rate of force development; rGRF= realtive ground reaction force; pEMG Ratio= peak vertical jump muscle activity/peak MVIC muscle activity. Values are given as means ± SD. *Significantly greater than 0Pre.

Visual Analog Scale

No significant ($p > 0.05$) interaction was found for soreness. There was no significant ($p > 0.05$) main effect for group but there was a significant ($p < 0.001$) main effect for time. 0Pre and 0Post were less than all time points. 24Pre were less than 48Pre, 48Post, 72Pre, and 72Post while 48Pre was greater than 72Pre and 72Post (Figure IIb).

Figure IIb. Means and SDs for VAS between groups and across time following exercise induced muscle damage. Significant ($p < 0.05$) differences from 0Pre are indicated with *.



DISCUSSION

This investigations aim was to determine the effect of WBV following exercise induced muscle damage on vertical jump performance. The exercise induced muscle damage protocol resulted in an immediate and prolonged detrimental affect on vertical jump performance, however, no differences were found between WBV and control groups. DOMS peaked at 48hours post injury, while the main performance findings were that VJH, PPO, RFD, and rGRF all had changes over time, indicative of decreased performance. Additionally, the normalized VM peak EMG during vertical jump exhibited no differences over time but there were differences between groups, with the control group having greater muscle activity when compared to the WBV group. To our knowledge, no previous research has investigated the effects of WBV on vertical jump performance following exercise induced muscle damage. Current research has either examined the effects of exercise induced muscle damage on vertical jump performance without WBV (6,15), the effects of WBV on vertical jump performance without muscle damage (1,9,10,11,13,17,19,20,27), or the effects of WBV on muscle recovery alone (2,7,8,12,28,32,41), which characterizes this investigation as novel in the performance and muscle recovery literature.

Previous research supports our findings that following exercise induced muscle damage vertical jump performance decreases immediately and up to 3 days after (6,15), irrespective of WBV treatment. In Byrne & Eston's investigation of vertical jump

performance following exercise induced muscle damage, they found decreases in squat jump height, depth jump height and counter-movement jump height following damage and for 3 days after (15). The present study extents their findings by measuring PPO, RFD or rGRF. Since the curret findings for PPO, RFD, and rGRF had similar trends as VJH, it may be expected that these trends would be similar in other jump performance studies. To our knowledge there are not any studies that have looked at PPO in vertical jump following exercise induced muscle damage however, it has been shown that PPO is immediately reduced following eccentric muscle actions in the knee extensors during isokinetic cycling (34) and during a Wingate cycle test (14) while continuing to be reduced up to 2 days post damage. A decrease in PPO has also been shown during intermittent maximal sprints (10x6s) on a cycle ergometer after 10 sets of 10 plyometric jumps to induce damage (39). These results are similar to the current results. Since PPO is a main predictor variable of athletic performance, it is important to limit any reductions in lower-body PPO.

In the current investigation, incorporating WBV as a recovery modality aimed at attenuating any reduction in performance was not successful as measured by VJH, PPO, RFD, and rGRF. Previous literature has shown mixed results when examining the effects of WBV on vertical jump performance. Some research has shown increases in VJH, PPO, RFD, and rGRF following WBV exposure (1,10,19,20), indicating neuromuscular facilitation or a potentiation effect. A recent study found increases in rGRF in recreationally trained individuals following WBV exposure compared to a control condition during a maximal vertical jump (20) and another study found no differences in RFD following WBV during isometric muscle actions (26). However, it appears that

when muscle is damaged it alters the effectiveness of WBV during vertical jumping performance. As previously mentioned, WBV has been researched as a recovery modality in the upper (28) and lower extremities (7,12,32), when measuring pain, force production, and clinical variables with different damage and vibration protocols but has not been investigated for vertical jump performance effects. These mixed results are most likely due to the use of varying damage protocols, vibration exposures, and extremities tested.

During the vertical jump in this investigation, VM muscle activity was measured using surface EMG and was normalized with VM peak EMG MVIC data for each participant. In the normalized VM peak EMG, there were no differences seen over time following muscle damage, however, the control group showed greater normalized muscle activity than the WBV group. Previous research has investigated WBV and EMG RMS in the quadriceps while squatting and found increased activation during WBV compared to no vibration (33). A study by Cormie et. al measured VJH and integrated EMG during vertical jumping and found increases in VJH immediately after WBV exposure compared to no vibration but no differences in integrated EMG between WBV and control (19). Our findings conflict with Cormie et. al, however their participants did not have muscle damage. Since exercise induced muscle damage changes the contractile properties of the muscle (3), this may account for the differences between our studies. Additionally, the current investigation used normalized peak EMG muscle activity where they used integrated EMG. Our findings suggest that WBV has a detrimental effect on peak normalized muscle activity in the VM during vertical jumping.

It is necessary to discuss how the potentiating mechanism of the stretch shortening cycle during a vertical jump attenuate the detrimental performance effects of exercise induced muscle damage. It has been suggested that excitation-contraction coupling is impaired following muscle damage (21), decreasing the release of calcium per action potential (40), leading to an inability to activate force-generating structures. It is proposed that after exercise induced muscle damage, a reduction in stretch reflex sensitivity and muscle stiffness occurs (24,30), which leads to decreased force potentiating mechanisms during the stretch shortening cycle. It is also suggested that exercise induced muscle damage induces modifications in pre-landing motor control, possibly brought on by central inhibition due to muscle soreness (24). Since the stretch shortening cycle is a key component in a countermovement vertical jump, this may help explain our findings of decreased in VJH, PPO, RFD, and rGRF following exercise induced muscle damage.

In conclusion, it appears that WBV has no effect on VJH, PPO, RFD, or rGRF following exercise induced muscle damage but has a detrimental effect on normalized VM peak EMG during vertical jumping. Utilizing WBV as a recovery modality has been shown to be ineffective in the current investigation. Future research should investigate a variety of WBV exposure times, frequencies, amplitudes, and rest intervals and their effects following exercise induced muscle damage. Different levels of soreness caused by exercise induced muscle damage should be examined to determine if the amount of soreness affects the results of WBV as a recovery modality. Additionally, trained athletes and males should also be examined with similar protocols to determine effects of different participant populations.

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CHAPTER V

MANUSCRIPT 3

EFFECTS OF WHOLE BODY VIBRATION ON MUSCLE CONTRACTILE PROPERTIES IN EXERCISE INDUCED MUSCLE DAMAGED FEMALES

INTRODUCTION

Over the last decade, whole-body vibration (WBV) has increasingly been implemented with exercise by applying oscillatory motions of a certain frequency and amplitude as a mean to improve performance. Research has shown positive effects on strength (13,14), power development (6,11), vertical jump height (11) and flexibility (17) following WBV. Although the exact mechanism of how the body responds to the vibration stimulus remains unclear, it has been suggested that it elicits neuromuscular facilitation (6,9). It has been previously shown that when vibration is directly applied to a tendon or muscle belly, vibration induces activity of the muscle spindle Ia fibers, mediated by monosynaptic and polysynaptic pathways (34). This increase in muscle spindle activity indicates a reflexive muscle contraction known as the tonic vibration reflex (TVR) arising from the direct vibratory stimulus. When WBV is implemented, it is theorized that vibrations are transferred from the platform to specific lower body muscle groups, especially ones that are in close contact with the platform. Consequently, WBV stimulates the sensory receptors and afferent pathways, which may lead to a more efficient use of the stretch reflex, recruitment and synchronization of motor units (6). Effects on muscle contractile properties have been examined following the use of vibration and have found mixed results. One study found no influence of WBV on peak force (PF), electro-mechanical delay (EMD), rate of force development, muscle electromygraphy, time to peak tension (TPT), and half relaxation time (HRT) during evoked twitch and voluntary contractions (20). Whereas, another study found no influence on direct vibration for twitch parameters (HRT, peak twitch, TPT, rate of

torque development, mean amplitude) but found differences in voluntary parameters (MVC and peak EMG), suggesting neural adaptations with an improvement of muscle activation (24).

Function of the neuromuscular system is critical in muscular performance; this may be important in sport performance or activities of daily living. In addition to maximal force output, percent activation of motor units, and EMG muscle activity are also major contributors to muscle function. Performance of resistance training for health and fitness benefits by athletes and clinical populations has gained in popularity in recent years. However, research has shown that repeated eccentric muscle contractions, which often occur during resistance training, may cause muscle damage resulting in decreased force production (2,29). This muscle damage is evident as a disruption of the normal alignment of the skeletal muscle and disruption of the z-lines of sarcomeres (18,35). This process initiates an inflammatory process and leads to delayed onset muscle soreness (DOMS) and edema in the damaged muscle. It is suggested that the loss of force may be due to voluntary activation, perhaps due to impairment of or damage to specific sites in the muscle. Impairment in the muscle may be due to the limited release (TPT) and/or reuptake (HRT) SR Ca^{++} process.

WBV has shown positive effects in assisting on exercise when applied prophylactically and therapeutically. Previous literature suggests that WBV increases muscle spindle activity, which results in less muscle fiber disruption to excitation-contraction coupling (1,4,25) when WBV is applied prior to muscle damage. It has been suggested that an increase in muscle pre-activation, theoretically increasing the number of motor units and muscle fibers recruited, could lead to an increased muscle recovery by

decreasing myofibril stress during repeated muscle actions (7). This indicates that a decreased amount of force loss may occur following exercise induced muscle damage when WBV is utilized. It has also been suggested that WBV increases blood flow to the musculature (4), which could accelerate repair and remodeling in the muscle (12). Another proposed mechanisms in some clinical populations suggest that WBV inhibits pain receptors, allowing for a higher pain tolerance when DOMS is experienced (30).

Literature involving WBV and muscle recovery following exercise induced muscle damage have conflicting results. Researchers have measured muscle pain, voluntary force, pressure pain threshold, creatin kinase levels, range of motion, and limb circumference measures (1,3,4,8,25,30,38) when utilizing WBV prior to or following exercise induced muscle damage and found mixed results. Thus, indicating inconclusive conclusions can be drawn involving WBV and exercise induced muscle damage. Furthermore, there are no studies that have examined the effects of WBV on muscle contractile properties following exercise induced muscle damage. The aim of this investigation was to determine the effect of whole-body vibration on muscle contractile properties following exercise induced muscle soreness in the quadriceps.

METHODOLOGY

Participants

Twenty-seven recreationally trained females (age 21 ± 2 yrs, height 172.38 ± 92.27 cm, mass 58.67 ± 11.53kg) volunteered to participate in a 7 session protocol and provided written, informed consent that was approved by the University's Institutional Review Board. Recreationally trained individuals were defined as meeting American College of Sports Medicine recommendations for healthy living and did not exceed 5 lower body workouts (aerobic and anaerobic) a week on a regular basis in the last 6 months. Participant with a recent history of lower body musculoskeletal or orthopedic injury or taking medications that alter balance, musculoskeletal system, or central nervous system functions relating to posture and motor control were excluded from participating. Additionally, individuals taking prescription pain and/or psychiatric medications were excluded. All participants were screened by questionaire for potential risk factors to the exercise protocol (i.e. rhabdomyolysis, bruising easily, etc.). Partipants were asked to not perform any lower body exercise or take any pain medications 48hours prior to testing sessions and during all testing days and to keep all food and water intake consistent during testing sessions. Furthermore, partipants were not scheduled for testing during their menstral cycle in order to avoid non-compliance with the instructions above.

Measures

Voluntary Force and Motor Unit Activation

An interpolated-twitch electrical stimulation protocol was employed to assess maximal voluntary isometric contraction (MVIC), the percentage of motor unit activation during MVIC (%ACT). Additionally, peak twitch torque (TT) in the relaxed muscle, the time to reach peak twitch torque (TP), and half relaxation time of twitch torque (HRT) were also assessed on all 7 visits to the laboratory. Knee extensor measurements were performed on a modified knee-extension/leg curl machine (Body Solid; model GLCE-365; Forest Park, IL). Participants were seated with the hip at 90º of flexion and knee fixed in a flexed position at an angle of 60º below horizontal. The lever arm of the machine was fixed to a force transducer (Transducer Techniques; model SBO-750, Temecula, CA) parallel to the line of pull and perpendicular to the lever arm, allowing for assessment of isometric torque. A strap was used to secure the participant's right ankle to the lever arm. Stimulation electrodes (7.5 cm X 10 cm; PALS Platinum; Fallbrook, CA) were placed on the skin over the distal vastus medialis and the proximal vastus lateralis to enable electrical stimulation of the quadricep. All electrode positions were marked with ink to ensure similar placement for subsequent days.

Prior to the initial assessment of MVIC, TT, %Act, TP, and HRT on each testing day, the stimulation current required to elicit a maximal torque value was determined by applying a series of brief electrical stimulations (paired pulses, consisting of two 0.2 ms pulses with an interpulse interval of 10 ms) to the knee extensors. Stimulation was applied using a constant current stimulator (model DS7AH; Digitimer, Hertfordshire, England) controlled by a computer using iWorx data acquisition software (iWorx System, Inc, Dover, NH, USA). Torque data was sampled at 5 kHz from the force transducer. The series of stimulations began with the current set at 40 mA and the current was

progressively increased by 20 mA until the measured torque plateaus. Each subsequent contraction was separated by 20s. The current eliciting the highest torque value was used to represent a supra-maximal stimulation current and was used for all subsequent stimulations applied that day. Next, participants performed a 3s MVIC with knee extensors. At 2.5s into the contraction a paired-pulse stimulation was applied, and the increase in torque over MVIC (interpolated-twitch torque; ITT) was assessed. At 2 and 4s after completion of the MVIC, additional paired-pulse stimulations was applied to the relaxed muscle. Peak TT was determined as the average of the two post-MVIC stimulations and be used in subsequent analyses. %Act was calculated as 100% x (1-ITT/TT). MVIC was determined as the peak torque during the 3s MVIC. TP was determined as the time from the onset of torque production to the time corresponding to peak twitch torque. HRT was determined as the time taken from peak twitch torque to reach 50% of baseline torque. Data from the two post-MVIC stimulations were averaged to determine TT, TP, and HRT and used for further analysis. Participants were given strong verbal encouragement during each effort, and three trials were performed, separated by 2 minutes rest. The two best trials were averaged and used as the criterion measures.

Electromyography

Bipolar surface electromyography (EMG) was recorded during MVIC on the left leg during testing visits to the laboratory. Noraxon Single Electrodes (Noraxon USA Inc., Scottsdale, AZ, USA) was placed 3-5cm apart at each location and a ground electrode on the head of the tibia. Proper skin preparation included abrasion of the skin around the electrode site followed by cleansing with an alcohol swab. Data was recorded from the

Vastus Medialis (VM) in the left leg using Noraxon Telemyo 8-channel EMG system (Noraxon USA Inc., Scottsdale, AZ, USA) with a hardware band pass filter (10-500Hz). During MVIC, EMG signals were collected on the left leg for 5s each on the same leg extension machine used for ITT. A strap was used to secure the participant's left ankle to the lever arm. Raw data was filtered using a 4-order Butterworth filter and mean and peak values were calculate and average over the 3 trials.

Procedures

During three familiarization sessions, participants read and signed informed consent, filled out screening questionnaires, were measured for anthropometrics performed all testing protocols. Following these familiarization sessions, participants visited the laboratory on 4 consecutive days and were randomly assigned to a control or treatment (WBV) group. Prior to each pre measurement, all participants performed 2 sets of 15 meters of dynamic warm-ups including: jogs, gait swings, high knees, exaggerated lunges and Frankensteins. They were then assessed for baseline values on %Act, VT, TT, TP, HRT, MVIC, and EMG in the quadriceps. After baseline values were taken, they performed an exercise induced muscle damage protocol, which consisted of split squats using a Jones Machine® by performing 4 sets to volitional failure on each leg. The Jones Machine® was front loaded with 40% of their body weight. During split squats, the rested back leg was placed on a bench for support with 90-degrees of flexion of the forward knee, allowing focus on single leg performance of the front leg. There was assistance on the concentric phase after they reached 90 degrees of flexion of the front knee on the exercising leg, allowing greater focus on the eccentric phase. Following

completion of each set, 1 min of rest was provided and participants switched legs. Exercise proceeded in this way until completed.

Immediately following the exercise induced muscle damage protocol, the control group performed 2 sets of body weight quarter squats on a flat surface for a 30s with a 1:1 (30s:30s) work to rest ratio. The WBV group performed 2 sets of body weight quarter squats on the vibration plate. An AIRdaptive® (Power Plate, Inc.) system was utilized for tri-axial vibration exposure. Vibration frequency was set at 30Hz with an amplitude of 2-4mm. Following completion of the treatment, participants were re-assessed for all measures. They then rested 10mins and were reassessed again.

Participants returned to the laboratory 24, 48, and 72 following the muscle damage protocol to evaluate measurements over time. Each sessions consisted of a baseline assessment of all variables and followed immediately by the treatment protocol. After WBV/control, measures were reassessed followed by 10min rest followed by a third set of measurements.

Data Analyses

To test changes in %Act, TT, TP, HRT, VT, and MVIC EMG over time and between treatment groups, a 12x2 (time by group) mixed factor analysis of variance (ANOVA) was conducted. Time being 0Pre, 0Post1, 0Post2, 24Pre, 24Post1, 24Post2, 48Pre, 48Post1, 48Post2, 72Pre, 72 Post1, and 72Post2 and group being control and whole body vibration. Also, three 3x2 (time by group) mixed factor ANOVA's were conducted on percent changes from 0Pre and 24Pre, 0Pre and 48Pre, and 0Pre and 72Pre. Eight 2x2 (time by group) mixed factor ANOVA's were conducted on the percent change

from each day comparing the pre measure to post1 and post2 of each respective day. If interactions occured they were followed up with one-way ANOVA's and any main effects were followed up with a least significant difference (LSD) post-hoc analyses for pairwise difference. All analyses were conducted using SPSS software 20 (SPSS, Inc., Chicago, IL). Statistical significance was defined as P value less than 0.05.

RESULTS

Raw values

%Activation. A significant ($p<0.05$) interaction for group by time was found. This was followed up with a 12x1 repeated measures ANOVA for each group. No significant ($p>0.05$) main effect for time was found for WBV group. A significant ($p<0.05$) main effect was found for time in the control group. With 0Pre being less than 0Post1 and 0Post2 and great than 48Post1. (Table1)

Twitch Torque. No significant ($p>0.05$) interaction of time by group was found. A significant main effect for group ($p=0.05$) and time ($p<0.001$) was found. With control group being greater than WBV group. For time, 0Pre values were greater than all other time points but 48Pre and 72Post1. (Table 1)

Volitional Torque. No significant ($p>0.05$) interaction for group by time were found. No significant ($p>0.05$) main effect for group was found but a significant ($p<0.05$) main effect for time was found. 0Pre was found to be greater than all other time points. (Table 1)

Time to Peak. No significant ($p>0.05$) interaction for group by time was found. No significant ($p>0.05$) main effect for group but a significant ($p<0.05$) main effect for time, with 0Pre being less than time points 0Post1- 24Pre. (Table 1)

Half Relaxation Time. No significant ($p>0.05$) interaction for group by time. No significant ($p>0.05$) main effect for group but a significant ($p<0.05$) main effect for time, with 0Pre being greater (longer) than time points from 0Post1-24Post2. (Table IIIa)

Table IIIa.

Table IIIa. Absolute means and SD's for %Activation, twitch torque, voluntary torque, time peak twitch torque, half relaxation time, mean MVIC EMG, and peak MVIC EMG between groups and across time.

	0Pre		0Post1		0Post2		24Pre		24Post1		24Post2		48Pre		48Post1		48Post2		72Pre		72Post1		72Post2	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
%Activation																								
WBV	70.74	15.27	74.22	10.26	74.23	20.32	77.16	19.72	74.85	17.78	77.74	20.68	73.94	17.63	69.66	21.98	71.57	21.46	70.06	17.50	73.60	22.10	70.87	18.14
Control	64.57	20.42	71.22	17.94 *	76.57	18.03 *	71.95	18.10	63.77	19.41	61.86	19.40	59.74	20.93	58.37	19.37 *	60.84	18.78	61.62	19.77	58.76	18.66	63.34	22.13
Twitch Torque (N/m)																								
WBV	88.26	18.97	54.20	19.93 *	47.26	19.26 #	69.00	18.96 #	64.63	18.50 #	65.03	18.73	81.51	20.26	78.82	19.90	75.36	21.67 #	78.76	15.71	80.19	18.34	75.12	18.78
Control	91.53	11.78	68.03	14.54 *	64.67	14.94 *	83.94	10.53 *	82.69	17.39 *	78.69	15.35 *	89.93	18.21 *	90.32	14.52 *	90.95	13.67 *	90.00	18.39 *	90.96	15.27 *	87.68	17.55 *
Voluntary Torque (N/m)																								
WBV	196.57	38.28	156.43	27.41	141.90	38.45	148.12	47.78	147.47	45.98	149.74	45.09	143.70	56.61	144.05	43.39 *	151.34	39.20	156.68	42.37	166.20	36.86	158.04	37.00 *
Control	172.37	42.47	144.28	46.47 *	142.98	39.04 *	137.95	26.12 *	132.27	34.87	141.67	38.41 *	132.28	33.60 *	133.81	33.19	141.25	33.50 *	155.93	32.76 *	152.73	30.57 *	172.27	49.06
Time to Peak Twitch Torque (ms)																								
WBV	40.51	5.23	43.47	5.57 *	42.82	5.93 *	44.42	5.12 *	41.79	6.13	42.82	7.45	42.68	6.69	40.45	6.36	42.21	6.44	41.59	8.07	39.91	7.72	41.76	8.44
Control	40.23	6.00	42.81	6.61	43.50	6.60	42.23	4.03	40.21	5.64	38.98	7.61	39.93	5.66	38.76	6.30	37.42	5.51	40.25	6.15	38.70	6.23	38.71	5.20
Half Relaxation Time (ms)																								
WBV	24.63	8.24	15.15	5.36 *	13.64	5.47 *	20.56	5.81 *	23.55	10.00 *	18.73	6.41 *	25.19	9.28	26.85	9.36	24.26	7.38	28.02	8.50	25.25	9.06	25.79	9.45
Control	25.51	6.90	14.57	5.31	12.46	3.40	21.73	7.18	22.02	9.43	22.96	9.20	25.54	8.76	25.02	10.27	23.43	10.39	26.22	12.37	24.18	8.60	24.99	8.09

# indicates significant group differences from WBV (p<0.05) and * indicates significant differences from 0Pre (p<0.05).

Mean EMG. No significant (p>0.05) interaction for group by time was found. No significant (p>0.05) main effect for group was found but a significant (p<0.05) main effect for time was found, with 0Pre being greater than 72Post1. (Figure IIIa)

Peak EMG. No significant (p>0.05) interaction for group by time was found. No significant (p>0.05) main effect for group was found but a significant (p<0.05) main effect for time was found. With 0Pre not being difference from any time point. (Figure IIIb)

Figure 6-7. Means and SD's for mean and peak EMG of VM between groups and across time following exercise induced muscle damage. Significant (p<0.05) group differences from WBV are indicated with #. Significant (p<0.05) main effects for time 0Pre are indicated with *.

Figure IIIa.

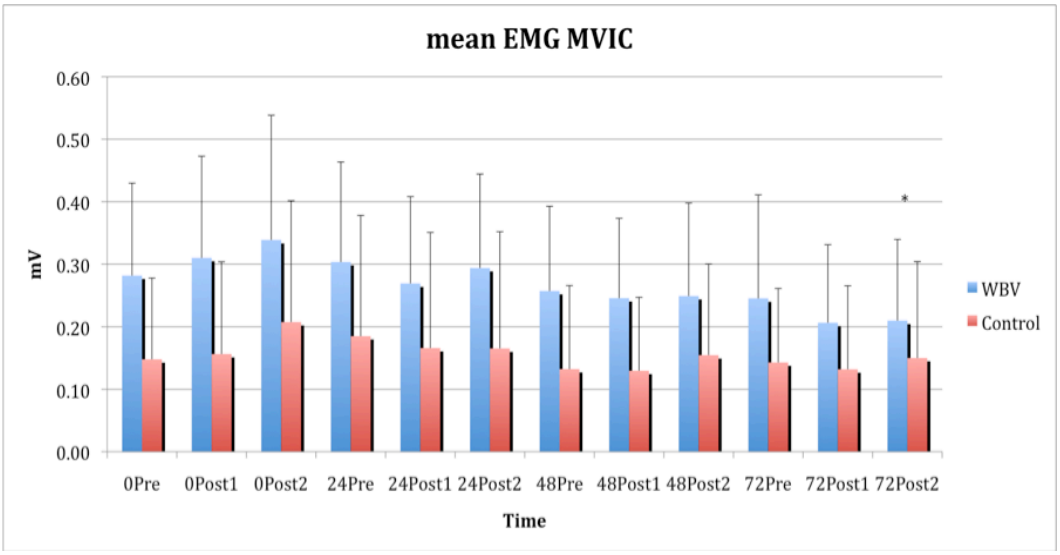
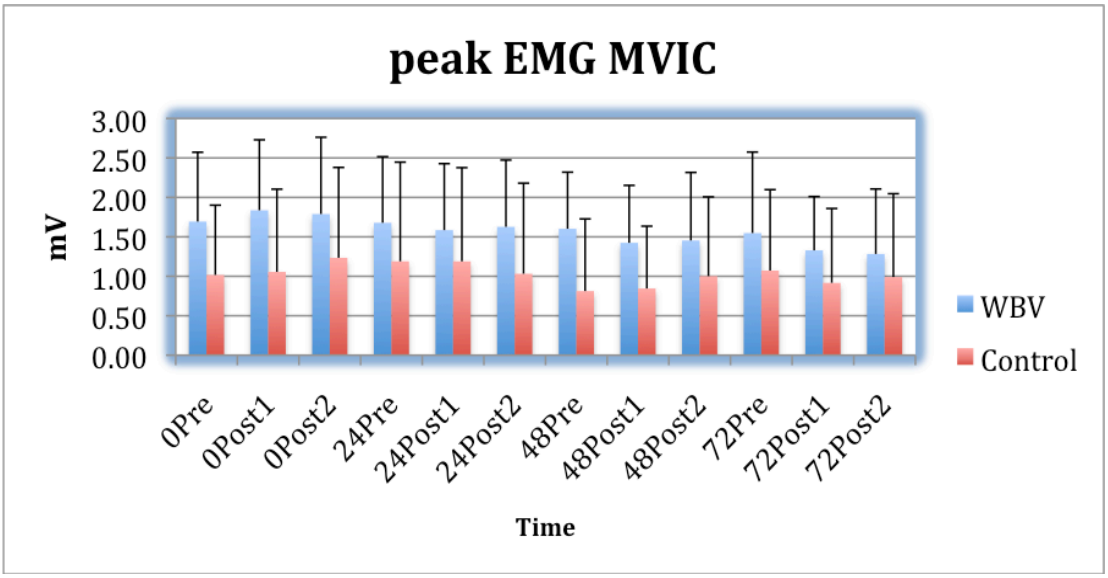


Figure IIIb.



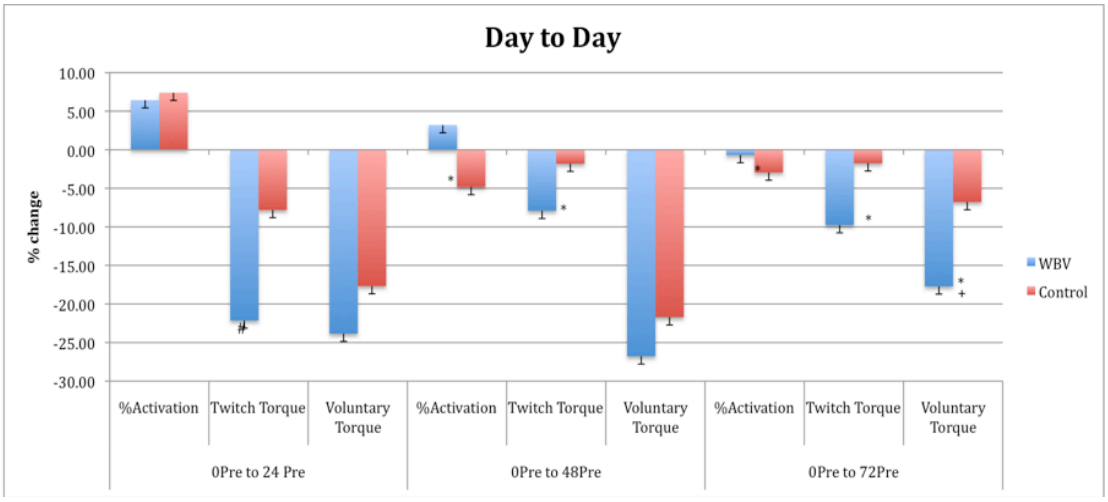
Changes from baseline to pre-values for each day

%Activation. No significant ($p>0.05$) interaction for group by time was found in %Δ pre's day to day. No significant ($p>0.05$) main effect for group was found but a significant ($p<0.05$) main effect for time was found for %Δ from day to day. With %Δ0Pre to 24Pre being great than %Δ0Pre to 48Pre and %Δ0Pre to 72Pre. (Figure IIIc)

Twitch Torque. No significant ($p>0.05$) interaction for group by time was found. A significant main effect for group ($p=0.02$) and time ($p=.001$) for TT. With control being greater than WBV group. For time, %Δ0Pre to 24Pre being less than %Δ0Pre to 48Pre and %Δ0Pre to 72Pre. (Figure IIIc)

Volitional Torque. No significant ($p>0.05$) interaction for group by time was found in %Δ pre's day to day. No significant ($p>0.05$) main effect for group was found but a significant ($p<0.05$) main effect for time was found for %Δ from day to day. With %Δ0Pre to 72Pre being greater than %Δ0Pre to 24Pre and %Δ0Pre to 48Pre. (Figure IIIc)

Figure IIIc. Means and SD's for %Δ of pre values day to day in %Act, TT, and VT between groups and across time following exercise induced muscle damage. Significant ($p<0.05$) group differences from WBV are indicated with #. Significant ($p<0.05$) main effects for time from %Δ0Pre toPost1 to all other time points are indicated with *.



Changes from pre-values to post measurement on each day

%Activation. No significant ($p>0.05$) interactions for group by time were found for %Δ within each day on all days. No significant ($p>0.05$) main effect for group or time was found for %Δ each day on all days. (Figures IIId-g)

Twitch Torque. A significant ($p<0.05$) interaction for group by time was found for day 48 and was followed up with a one-way ANOVA for each group, showing that control is greater than WBV group for %Δ48Pre to 48Post2 only. No significant ($p>0.05$) interactions for group by time were found were found for all other days. A significant ($p<0.05$) main effect was found for group and time on day 0. With control being greater than WBV group and was followed up with a one-way ANOVA for each time point, showing WBV was greater than control in %Δ0Pre to 0Post1 and %Δ0Pre to 0Post2. For time, %Δ0Pre to Post1 was greater than %Δ0Pre to Post2 . No significant ($p>0.05$) main effects for group or time were found for day 24. No significant ($p>0.05$) main effects for group were found but significant ($p<0.05$) main effects for time were found, with %Δ72Pre to Post1 being greater than %Δ72Pre to Post2 for day 72. (Figures IIId-g)

Volitional Torque. No significant ($p<0.05$) interaction for group by time was found for days 0, 24, and 48. Significant ($p<0.05$) interaction for group by time was found for day 72, this was followed up by a one-way ANOVA for each group indicating no significant ($p>0.05$) differences for both groups. No significant ($p>0.05$) main effect for group was found for all days. No significant ($p>0.05$) main effect for time was found for day 0 but significant ($p<0.05$) main effect for time was found in day 24 and 48. With %ΔPre to Post2 being greater than %ΔPre to Post1 for day 24 and 48. (Figures IIId-g)

Figure IIId-g. Means and SD's for %Δ of pre values to post values on each day for %Act, TT, and VT between groups and across time following exercise induced muscle damage. Significant ($p<0.05$) group differences from WBV are indicated with #. Significant ($p<0.05$) main effects for time from %ΔPre toPost1 to %ΔPre toPost2 are indicated with *.

Figure IIId.

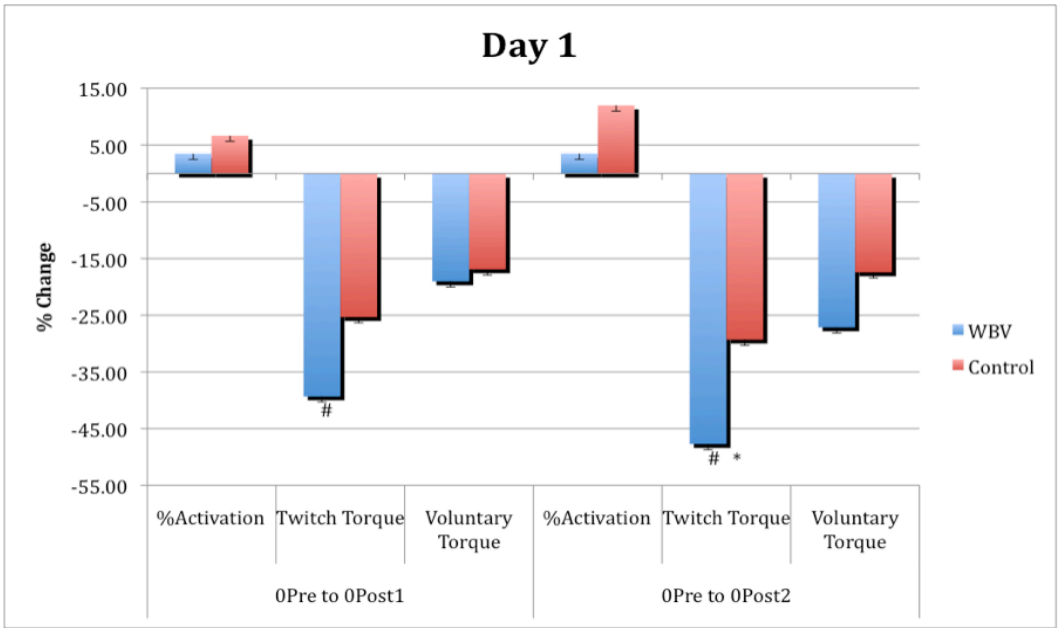


Figure IIIe.

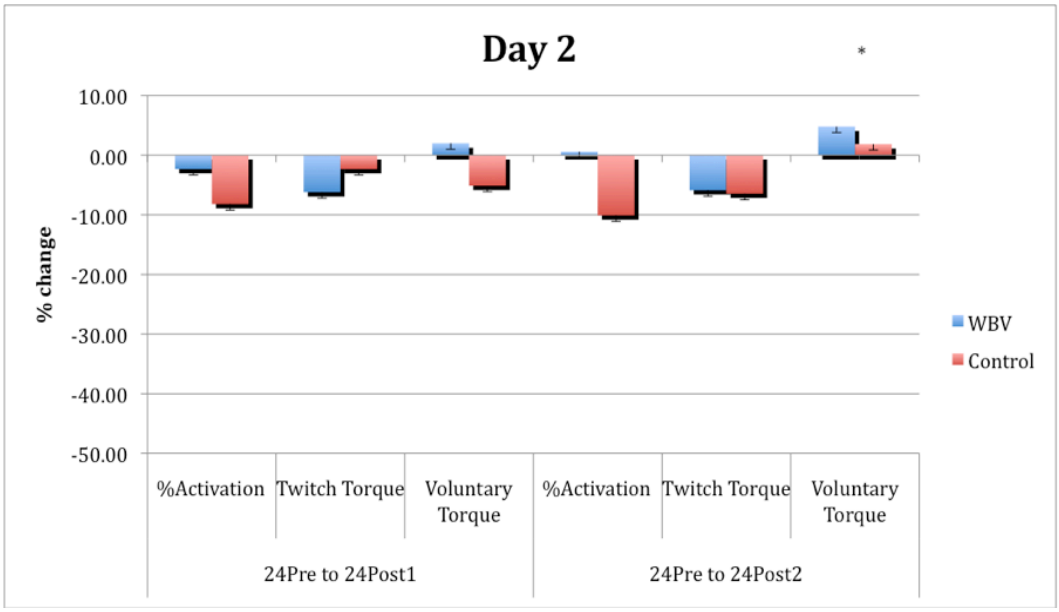


Figure IIIf.

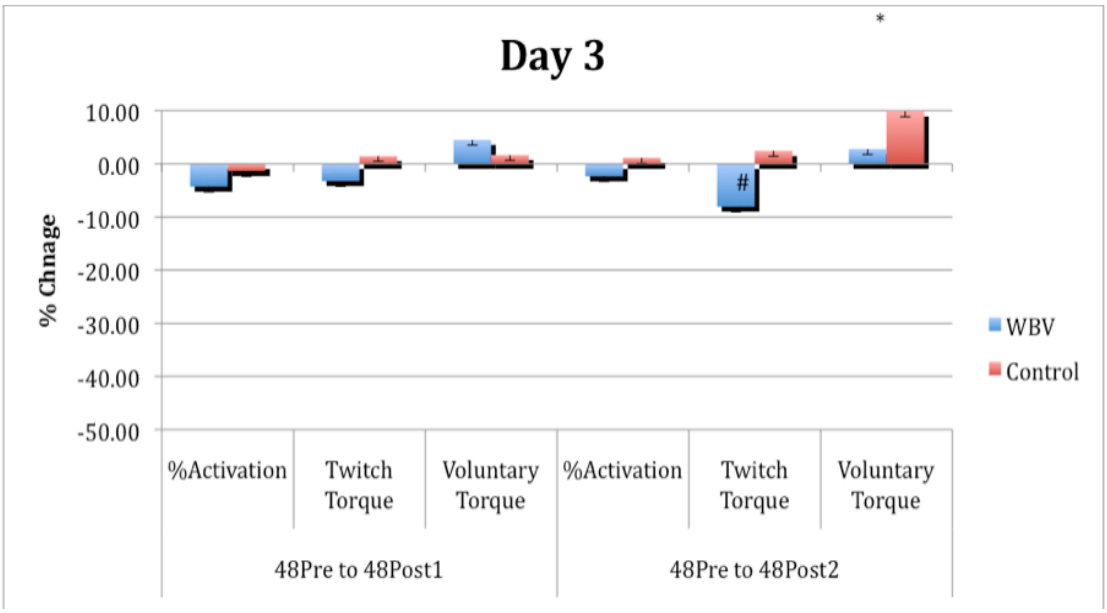
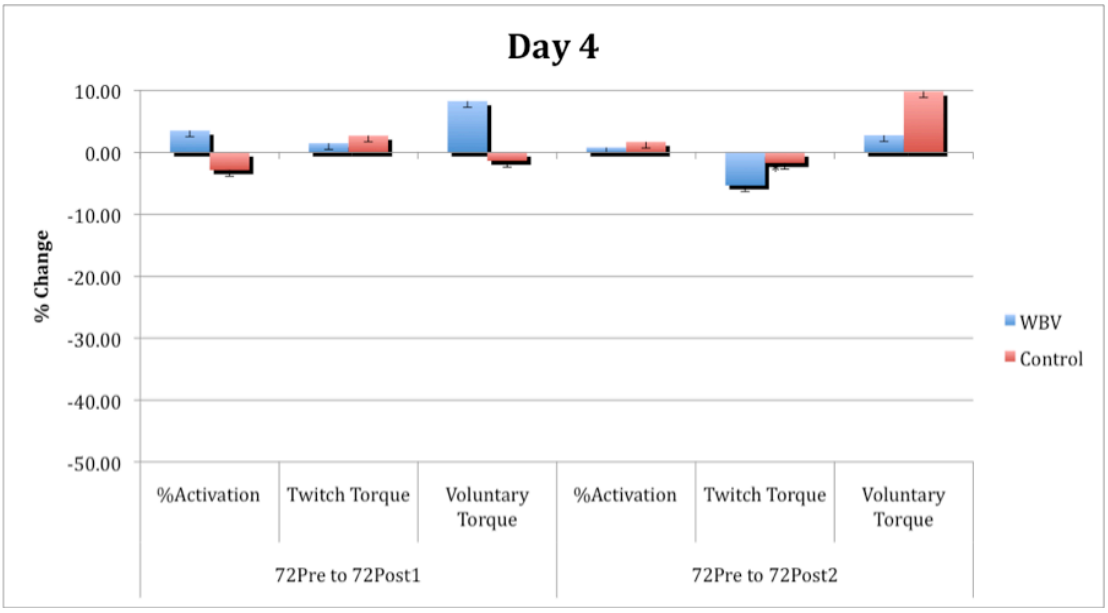

Day 3
*
#
10.00
0.00
-10.00
-20.00
-30.00
-40.00
-50.00
% Chnage
WBV
Control
%Activation
Twitch Torque
Voluntary Torque
%Activation
Twitch Torque
Voluntary Torque
48Pre to 48Post1
48Pre to 48Post2

Figure IIIg.


Day 4
10.00
0.00
-10.00
-20.00
-30.00
-40.00
-50.00
% Change
WBV
Control
%Activation
Twitch Torque
Voluntary Torque
%Activation
Twitch Torque
Voluntary Torque
72Pre to 72Post1
72Pre to 72Post2

DISCUSSION

The purpose of the present study was to determine if whole-body vibration altered muscular strength, motor-unit activation, and twitch contractile properties of skeletal muscle in the days following exercise-induced muscle damage. WBV has been shown to potentially be an effective treatment modality for both pain and strength loss following EIMD (1,3,25,30) and to lead to acute improvements in voluntary strength and muscle activation in the absence of EIMD (10,14). In contrast to previous findings, and despite the plausible benefits of WBV on muscular function, the effects of WBV on muscle strength, activation, and contractile properties were not differ from control. Voluntary torque and twitch torque both decreased immediately after the eccentric exercise protocol and remained lowered throughout the study (72hours later). This finding is consistent with previous literature on strength loss following EIMD (23,33). WBV was found to have no effects on the day-to-day progression (measured across 0Pre, 24Pre, 48Pre, and 72Pre) of VT compared to control. Motor unit activation was also unaffected by WBV over the 72 hours of the study. Previous research has examined muscle activity with and without WBV in non-damaged muscle and the literature is conflicting. Some researchers found that WBV had no influence on EMG during a MVIC in the knee extensors (20,21,27), which is consistent with the current investigation, where others have found WBV increased muscle activity during MVIC (24). There is some previous research on muscle activity following exercise induced muscle damage, showing an increase in the rectus femoris 2, 3 and 10 days post exercise induced muscle soreness (15). It has been suggested in several studies that during isolated muscle preparations in mice, there is a

dissociation in EMG activity from force production during electrically stimulated isometric muscle actions following series of eccentric muscle actions (22,36,37).

WBV has been shown to improve voluntary force production and percent activation in uninjured muscle, perhaps due to use of the stretch reflex, recruitment and synchronization of motor units (6). It is unclear whether the lack of an effect in the present study was due to the effects of the damage *pre se* (i.e. damaged muscle would not respond in the same manner to WBV as undamaged muscle) or to differences in the vibration protocol and muscle(s) examined. A previous study did show faster recovery of voluntary force following EIMD when vibration was applied prior to the eccentric exercise protocol, but no measures of motor-unit activation making it difficult to attribute a mechanism to the observed effect. However, when examining %Act following EIMD, a study has shown that there are no changes in %Act over time (19) where other studies have suggested that full voluntary activation can be achieved following muscle damage (28,31,32). Thus, indicating the immediate and prolonged decrease in force production following muscle damage is not due to a reduction in voluntary activation it is caused by peripheral mechanisms (at or distal to the neuromuscular junction). Future studies in this area are clearly needed.

Previous studies have shown no effects of WBV on TT in undamaged muscle (20,24). Additionally, research examining the effects of WBV and contractile properties in non-damaged muscles found no differences in TP following vibration (20,24), which is consistent with the current investigation. Changes in TP and HRT would represent alterations in the peripheral (muscle) contractile apparatus associated with excitation-contraction coupling (5). Calcium handling efflux from the SR (22), as well as calcium

re-uptake into the SR (27,41). In the current study, time to peak tension in the twitch increased immediately and up to 24hrs post exercise induced muscle damage but no influences of WBV were seen. These results indicate that following muscle damage, it took longer to reach peak twitch up to 24hr post, which has been suggested to occur due to a reduced amount of Ca^{++} release from the SR, occurring from excitation-contraction coupling failure, reducing force. The reduced release of Ca^{++} occurs from the SR Ca^{++} channels open with depolarization and close rapidly once the membrane is repolarized, allowing a small release of Ca^{++} (16). Previous research examining the effects of WBV and contractile properties in non-damaged muscles found no differences in TP following vibration (20,24), which is consistent with the current investigation. Other research examining effects of muscle contractile properties following exercise induced muscle damage, with no vibration, found no difference in TP after 48hr post (23), where another study found decrease TP immediately post muscle damage (33). Both of these studies do not support our findings in the current study. The current investigation found a decreased half relaxation time following exercise induced muscle damage immediately and up to 24hrs post. There were no influences of WBV on HRT in the current study. Previous researchers investigated WBV and muscle contractile properties found no differences with WBV on HRT (20,24), which is consistent with the current investigation. Researchers examining effect of muscle contractile properties following exercise-induced muscle damage have found that HRT has decreased immediately post (33), supporting the current investigation findings and another study found that it increased 48hr after muscle damage (23). If there is an increase in HRT following muscle damage it may possibly due to the limited SR Ca^{++} reuptake process. This is caused by a depression in

the Ca uptake by the SR and has been attributed to either a reduction in Ca^{++} stimulated ATPase activity (26,39) or to mechanical damage of the SR induced by contractions of stretched muscle fibers (5). Our findings, in conjunction with previous literature indicate that EIMD leads to alterations in muscle contractile function, likely due to impaired calcium kinetics and that WBV does not alter peripheral contractile function in either undamaged or damaged muscle.

When observing within day effects, the aim here was to tease out if WBV changed acutely and/or 10min after exposure. Acutely, VT at 24 and 48 hours following EIMD was found to increase at the post2 measure on both days. Since there were not any group differences, this could not be attributed to the treatment but it may be due to a warm-up effect. The warm-up effect has been shown to prepare the muscle with a heightened state of readiness. An interesting finding in the current study was that in the control group only, %Act increased immediately and 10min following muscle damage. This is indicating that immediately following muscle damage the ability to recruit motor units, that were capable of cross bridge cycling, increased compared to baseline measures. When observing the percent change from day to day and within each day, %Act showed no differences between groups or over time. In mean EMG at 24hours, pre being greater than post1 measure. There were no within day differences for peak EMG.

For TT, on damage day and at 72 hours following EIMD, acute effects were seen. On damage day, the percent change from pre to post1 was greater than the percent change from pre to post2 measure. This may be indicative of fatigue from the damage protocol during the post1 measurements. However, there was a detrimental effect with WBV on TT 10min after WBV following the damage protocol and 24hr following. These findings

in TT are unexpected and due to its inconsistency over time make it difficult to conclude any reasoning as to why a decrease in TT occurred. However it may indicate that WBV exacerbated the decline in force during these time points; suggesting WBV does not aid in recovery and actually may contribute to the neuromuscular functional declines caused by muscle damage. This may be caused by peripheral fatigue in which WBV is impairing excitation-contraction coupling, causing a decline in force, indicated by the results in TT. In TP, at 24 hours pre values were greater than post1 and post2. For HRT, on damage day, post1 values was greater than post2 values, this may also be due to fatigue, indicating that it took longer for the muscle to relax. Acute effects have not previously been examined during this type of investigation.

In conclusion, WBV only had a negative effect on TT following exercise induced muscle damage and all other muscle contractile properties investigated in this study were not influenced by WBV and did not facilitate neuromuscular function. Torque variables were decreased following muscle damage where percent activation and EMG were increased immediately after muscle damage and up to 24hr post. The literature in muscle contractile properties is limited and contradicting. Further research is warranted in the effects of WBV on muscle contractile properties following exercise induced muscle damage.

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VITA

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EDUCATION

Doctor of Philosophy

University of Mississippi, University, MS

Dept of Health, Exercise Science, & Recreation Mgt

Major Area: Health & Kinesiology

Concentration: Biomechanics

August 2013

Dissertation: The Effect of Whole-body Vibration on Muscle Recovery and Performance

Master of Science

California State University, Fullerton, Fullerton, CA

Dept of Kinesiology

Major Area: Kinesiology

Concentration: Strength and Conditioning

May 2010

Thesis: The Effect of Whole-body Vibration Warm-up on Bat Speed

Bachelor of Science **California State University, Fullerton**, Fullerton, CA

Dept of Kinesiology

Major Area: Kinesiology

Concentration: Strength and Conditioning

May 2008

RELEVANT WORK EXPERIENCE

August, 2011 – Present **Graduate Research Assistant**

Applied Biomechanics Laboratory

Department of Health, Exercise Science, & Recreation Management

Appointment: Applied Biomechanics Laboratory Student Director

- Equipment Management
- Research Design & Implementation
- Data Collection & Analysis

January, 2011 - Present **Graduate Teaching Assistant**

Department of Health, Exercise Science, & Recreation Management

University of Mississippi

- ES 456, Exercise Testing and Prescription
- ES 447, Biomechanics Lab
- ES 349, Exercise Physiology Lab
- HP 203, CPR and First Aid
- HP 191, Personal and Community Health
- EL 151, Weight Lifting

- EL 147, Tennis

July, 2011 **Graduate Assistant**

Health Advisor for General Electric Aviation Plant

University of Mississippi

- Health Assessments
- Group and Individual Exercise Prescription

August, 2010-January 2011 **Personal Trainer**

Campus Recreation Center

University of Mississippi

August 2009-April, 2010 **CPR, AED and First Aid Lay Responder Instructor**

Student Recreation Center

California State University, Fullerton

May, 2008 – August, 2010 **Fitness Instructor**

Employee Wellness Center

California State University, Fullerton

August, 2008-August, 2010 **Graduate Assistant**

Health Fitness Testing

California State University, Fullerton

August, 2008 – August, 2010 **Teaching Assistant**

Dept of Kinesiology

California State University, Fullerton

- KNES 100, Physical Conditioning
- KNES 117A, Bowling

Research Experience

August 2010 – Present

University of Mississippi Applied Biomechanics Laboratory

Student Director (2011 – Present)

- *Kinetics and Kinematics of Slip Trials in Firefighters (3-D analysis-Vicon)*
- Three-Dimensional Examination of the Influence of Differently Weighted Warm-up Bats on Swing Kinematics *(3-D analysis-Vicon*), ***PI***
- *The Effects of Whole-Body Vibration on Rest Intervals in Jumping Performance,* ***PI***
- *The Effect of a TMJ Device on Athletic Performance Measures*
- The Acute Effects of Whole-Body Vibration on Functional Stability Measures in Older Women.
- The Influence of Body Composition on Selected Jump Performance Measures in Varsity Collegiate Female Varsity Athletes.
- The Effect of Boot Type on Postural Control
- The Effect of DOMS on Gait Kinematics during VO2 Max Assessments

2009 – 2010

California State University, Fullerton Fitness Assessment Laboratory

Student Director (2009 – 2010)

- Organizing data collection
- Mentoring interns on administering a variety of fitness assessments
- Data Entry
- Analyzing and interpreting reports

2008 – 2010

California State University, Fullerton Center for Sport Performance

Research Assistant (2008 – 2010)

- The Effect of Time on Navicular Height with Low-Dye Arch Taping
- Influence of Rest Duration Following a Potentiating Stimulus on Muscular Power
- Relationship Between Heart Rate Recovery values and Body Composition in children and adolescents
- Physiological Profile of Mixed Martial Artists
- Effect of whole-body vibration warm-up on bat speed
- Optimal Elastic Cord Assistance to Enhance Vertical Jump Performance
- Effect of Different Rest Intervals Following Whole-Body Vibration on Vertical Jump Performance

PEER-REVIEWED JOURNAL ARTICLES

1. Allen CR, **Dabbs NC**, Garner JC. The Acute effect of commercially available performance mouthpiece on Strength and Power Assessments. Journal of Strength and Conditioning Research. (in review)

2. Abe T., **Dabbs N.C**., Nahar V.K., Ford M.A., Bass M.A., & Loftin M. Relationship between Dual-Energy X-Ray Absorptiometry-Derived Appendicular Lean Tissue Mass and Total Body Skeletal Muscle Mass Estimated by Ultrasound. International Journal of Clinical Medicine. 4, 283-286, 2013.

3. **Dabbs NC,** Tran TT, Garner JC, Brown LE. A Brief Review: Utilizing whole-body vibration to increase acute power and vertical jump performance. Strength and Conditioning Journal. 34(5):78-84, 2012

4. Tran TT, Brown LE, Coburn JW, Lynn SK, **Dabbs NC**. Effects of assisted jumping on vertical jump parameters. Current Sports Medicine Reports. 11(3):155-159, 2012.

5. **Dabbs NC**, Muñoz CX, Tran TT, Brown LE, Bottaro M. Effect of different rest intervals following whole-body vibration on vertical jump performance. Journal of Strength and Conditioning Research. 25(3): 662-667, 2011

6. Tran TT, Brown LE, Coburn JW, Lynn SK, **Dabbs NC**, Schick MG, Schick EE, Khamoui AV, Uribe BP, Noffal, GJ. Optimal elastic cord assistance to enhance vertical jump performance. Journal of Strength and Conditioning Research. 25(12):3472-3478, 2011.

7. Schick MG, Brown LE, Coburn JW, Beam WC, Schick EE, **Dabbs NC**. Physiological profile of mixed martial artists. Medicina Sportiva. 14(4): 182-187, 2010

8. Dabbs NC, Brown LE, Coburn JW, Lynn SK, Biagini MS, Tran TT. Effect of whole-body vibration warm-up on bat speed. Journal of Strength and Conditioning Research. 24(9):2296-2299, 2010

9. Jo E, Judelson DA, Brown LE, Coburn JW, **Dabbs N**. Influence of Rest Duration Following a Potentiating Stimulus on Muscular Power. Journal of Strength and Conditioning. 24(2):343-347,2010.