INFLUENCE OF WHOLE-BODY VIBRATION ON DELAYED ONSET MUSCLE SORENESS

by

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ABSTRACT

Exercise induced muscle damage (EIMD) results in delayed onset muscle soreness (DOMS). Whole body vibration (WBV) may be a method that can be implemented to allow a subject suffering from DOMS to recover more quickly. The purpose of this study was to determine if WBV aids in managing symptoms of EIMD over a recovery period of 72 hours and to determine the effects of WBV on jumping performance following exercise-induced muscle damage. Measurements of performance like vertical jump height, peak-Z force, and pain pressure threshold were recorded. Twenty-seven recreationally trained females participated, and were damaged by performance of the eccentric portion of split squats. WBV was found to not be effective in the pain management of DOMS. Further research should be conducted, as literature shows some support for the management of DOMS symptoms via WBV. Inappropriate methodology for damage may have occurred in this study, damaging the subjects too much for WBV to be effective.

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CHAPTER I: INTRODUCTION

List of Abbreviations

DOMS	Delayed Onset Muscle Soreness
EIMD	Exercise Induced Muscle Damage
WBV	Whole Body Vibration
PPT	Pain Pressure Threshold
RF	Rectus Femoris
VJH	Vertical Jump Height
RFD	Rate of Force Development

Hypotheses

Ho_1: There will be no difference in PPT responses between and within groups over time.

Ho_2: There will be no difference in VJH responses between and within groups over time.

Ho_3: There will be no difference in peak Z force during VJ responses between and within groups over time.

Definitions

Concentric muscle action: a muscle shortening under tension

Contraction: a reduction in the distance between two ends of a muscle due to active shortening of the muscle

Eccentric muscle action: muscle lengthening under tension

Force: A vector quantity that describes the action of one body on another

Ground Reaction Force: An equal and opposite force that acts on the body in response to the force applied by the body to the ground

Length tension relationship: variation in force output of a muscle over a range of lengths due to different number of active sites available for cross-bridge formation

Muscle action: the development of muscle tension

Power: The rate of doing work; $P=W/t$

Introduction:

Repeated eccentric muscle contractions have been shown to cause exercise induced muscle damage (EIMD) resulting in decreased force production (5, 66). This muscle damage is evident as a disruption of the normal alignment of the skeletal muscle and disruption of the Z-lines of sarcomeres (40, 41, 76). EIMD results in symptoms such as delayed onset muscle soreness (DOMS), tenderness, edema, and muscle stiffness (43, 62). Inflammation results in pain, edema and decreased range of motion (ROM), force, and vertical jump (VJ) height. DOMS has been reported as an undesirable side effect of exercise due to its painful and debilitating effects on individuals (74). Peak DOMS usually occurs 24 to 48 hours following exercise (62).

Previous research has studied several ways to control or prevent EIMD symptoms (21). In physically active individuals, decreased swelling, stiffness and pain may allow a quicker return to activity. For individuals clinically diagnosed with pain, decreasing muscle pain for any period of time results in pain management and can enable activities of daily living. Treatment modalities such as massage, ultrasound, cryotherapy, and stretching have not been consistently effective in treatment of muscle pain (21).

Whole-body vibration (WBV) is a mechanical stimulus consisting of oscillatory motions. Frequency and amplitude are defining characteristics of the oscillatory motions of vibration exposure. The exact mechanism for WBV is still unknown but exposure to WBV has been shown to increase neuromuscular activity. There are many positive effects from the neuromuscular activity such as: joint flexibility (38), strength (30), performance (27, 29) and power output (13). Some research concerning how WBV affects EIMD symptoms has been conducted. Previous studies suggests that WBV results in less disruption of the excitation-contraction coupling due to increased muscle spindle activity and muscle pre-activation (4, 10, 55). When WBV exposure was given prior to EIMD, there was less reduction in force compared to control treatments of no vibration (4, 10, 55). It has been suggested that due to an increase in muscle pre-activation, theoretically, there should be an increase in the number of motor units and muscle fibers recruited. As a result, there could be a decrease in myofibril stress during repeated muscle actions, which leads to increased muscle recovery (14). This indicates that a decreased amount of force loss might occur when WBV is utilized prior to EIMD.

Increased blood flow to the muscle, leading to an increase in removal of waste and delivery of nutrients, may result from WBV (10). Increased blood flow could also accelerate repair and remodeling in the muscle (28). Another proposed mechanism is that WBV inhibits pain receptors, which allow for a higher pain tolerance in patients with chronic pain (68). It is proposed that vibration receptors in the skin stimulate inhibitory interneurons in the spinal cord, which act to reduce the amount of pain signals transmitted (64).

There are conflicting results in studies concerning WBV prior or immediately following EIMD and how it affects muscle pain, pressure pain threshold (PPT), range of motion (ROM) and limb circumference measurements (4, 9,10, 17, 55, 68, 84). A smaller reduction in force has been found when WBV is utilized prior to EIMD (4, 10).

There are many variables concerning vibration that may contribute to the different effects observed when studying EIMD. The timing of when WBV is applied may contribute to fundamental differences in the effect on EIMD symptoms. The type of vibration, whether whole body or direct, may influence the applicability of using vibration as a treatment, and the best type of vibration may be specific to a population. One purpose of this investigation was to determine if WBV aids in managing symptoms of EIMD over a recovery period of 72 hours.

During most performance activities, the main goal is to maximize power output. Power is the product of force and velocity. Power output may be compromised when EIMD occurs. It has yet to be determined if reduction in force or velocity contributes more to the decrease in power output following EIMD, as most studies have only calculated power, and not the subcomponents. It has been shown that peak power output is immediately reduced following eccentric muscle actions while the power is continued to be reduced up to 2 days post injury. These studies involved the knee extensors during isokinetic cycling (72) and a Wingate cycle test (20). 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 (80). Vertical jump performance is related to peak power output and could be compromised following EIMD. Studies have found prolonged reduction in maximal force production, ground reaction forces, and muscle and joint stiffness regulation following EIMD, all of which affect jumping performance. VJ performance with and without a countermovement has been shown to have immediate and long-lasting reductions in performance up to 4 days post-injury but is dependent on jump type (19).

Eccentric exercises are commonly used as a component of strength-training programs and have been shown to elicit EIMD, potentially causing a reduction in sport performance. Recently, WBV has been suggested as a novel modality to reduce or control symptoms of EIMD (10, 68). One study, in 2007, found that vibration prior to eccentric exercises may prevent and control DOMS with possible mechanisms of increased blood flow to facilitate recovery, muscle regeneration, and possible pain inhibition (10). Another study, in 2009, implemented WBV in combination with stretching and massage after strenuous exercise over a period of 72 hours, and showed decreased pain perception (68). A study in 2011 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 (4). A secondary purpose of this investigation was to determine the effects of WBV on jumping performance following exercise-induced muscle damage. To our knowledge, no study has investigated these measures.

CHAPTER II: REVIEW OF LITERATURE

Exercise Induced Muscle Damage

Exercise Induced Muscle Damage (EIMD) can be categorized as either acute or delayed damage. Often, both types of damage occur together when unaccustomed exercise is performed. Acute damage is the damage seen immediately following the exercise and up to 24 hours post-damage. Delayed muscle damage can be most easily observed 24 hours through 72 hours following damage, although it can last up to 10 days (22).

There are two theories for the causation of the damage. The initial damage is thought to be due to the mechanical disruption of the fiber (63). Stretching of "weak" sarcomeres result in a reduction in myofilament overlap, which then affects the length-tension relationship. Damage occurs to the cell membrane, t-tubules, Z-lines and sarcoplasmic reticulum (54). Focal myofibrillar damage is observed immediately following a bout of eccentric contraction (61). This structural damage is associated with a decrease in force production. There are multiple suggestions for why there is a decrease in force, which are further considered below.

The immediate reduction may be attributed to a shift in the length-tension relationship. With the “popping sarcomere” hypothesis (66), the lengthening of active muscles doesn’t occur with uniform lengthening. Some sarcomeres over-extend (“popping”) beyond myofilament overlap and fail to re-interdigitate upon relaxation. The other sarcomeres must compensate with a shorter length, and a shift in the length-tension relationship. Thus, a longer muscle length is needed following eccentric exercise in order to achieve the same myofilament overlap as prior to damage (80).

“Streaming” or widening of the Z-lines is the most prominent visual indication of damage. The Z-disk provides an important structural linkage in the transmission of tension and contractile forces along the muscle fiber and has a role in sensing of muscle activity and signal transduction. The alterations to the Z-line can be associated with amorphous structures of the fiber, such as the sarcomere, Z-line, or even the alignment of A or I bands (87). This “streaming” is associated with decreased force production.

In injured muscles, there is impairment of calcium release from the sarcoplasmic reticulum. Eccentric contractions alter the structure of T-tubules. As damage to the sarcoplasmic reticulum increases, there is an increase in tetanic intracellular calcium. In the muscle, tetanus is a state of smooth, sustained contraction. Tetanus often occurs when a motor unit is maximally stimulated by its motor neuron, which occurs when there is a high frequency of stimuli. This increase in intracellular calcium is associated with increased protease activity, such as calpains (65).

Calcium is associated with calpains, which are proteolytic enzymes that initiate breakdown of myofibrils. Calpains have also been found to degrade Z-discs, and other proteins, such as desmin (81). Calpains ultimately cause sarcoplasmic reticulum damage, which results in a decrease in intracellular calcium, leading to decreased force production (65). Further study is required to more fully understand the relationship between calpains, and extent of muscle damage.

Following the mechanical trauma, there is an inflammatory response by the body. The inflammatory response occurs with the purpose of removing damaged debris from the injured area. Neutrophils destroy necrotic tissue via phagocytosis and work with macrophages found within the tissue. Infiltration of neutrophils occurs in the skeletal muscle 45 mins-2 hours after damage (63). Other inflammatory cells, such as macrophages and T-lymphocytes begin to flood the cell 6 hours or longer following exercises, and can be seen present in the muscles up to 9-14 days later (63). It is suggested that the presence of these cells at the later times is related to muscular repair, not removal of cellular debris.

The inflammatory response in a damaged muscle is responsible for decreased ROM, force, vertical jump height, percent activation and an increase in pain and swelling. To an extent, force production and ROM are directly related. As ROM is decreased, force production is also decreased. In the example of a squat-jump, with a decreased ROM, the individual isn't able to squat as deep before exploding up. In the length-tension curve, the curves for active and passive force overlap slightly. The maximum force created is at 125% resting length of the muscle.

The exact mechanism responsible for the decreased ROM requires further research. However, a decrease in ROM is a reliable indication of EIMD and can be observed immediately (81). Force loss immediately following eccentric exercise may be partially attributed to damage in the excitation-contraction coupling system (82). The delayed damage is due to the body's response to the initial inflammation, resulting in DOMS.

Delayed Onset Muscle Soreness

Physiology and Mechanism

Delayed onset muscle soreness occurs when unaccustomed exercise is performed, and is primarily associated with eccentric exercise. Eccentric exercise is the lengthening or stretching of active muscle fibers. Eccentric movements function to slow or stop the movement. When muscle damage occurs, DOMS along with stiffness, swelling, decreased range of motion and several other indicators of damage can be observed. DOMS generally lasts 24 to 72 hours and peaks in intensity at 24-48 hours.

Muscle injury results in a decrease of force production in the damaged muscle (66). Eccentric contractions create more force than concentric or isometric contractions, while having lower motor unit activation (37). Contractions detach the cross-bridges of muscle fibers, and in eccentric contractions, the detachment is much more forceful. This creates a substantial amount of mechanical stress on the muscle fibers resulting in damage (37).

Structural damage to the fibers is supported by research. Z line streaming occurs which is disruption of the myofibrils at the Z bands. Disruption of the striation pattern has also been recorded (40). Damage to the Z lines is more likely to occur in Type II fibers, due to their more narrow and thus weaker Z lines. Damage to the excitation-contraction coupling system also occurs (6). It is attributed to a reduction in Ca^{2+} released after eccentric contraction (65).

The inflammatory response is initiated due to damage at the cellular level of the muscle. This response causes fluid to accumulate in the affected muscle in order to remove damaged proteins and other byproducts. Lysosomes create an indigestible residue termed lipofuscin granules. There is an increased presence of the granules in the damaged muscles 72 hours post-exercises (41). The inflammatory response in a damaged muscle is responsible for DOMS, decreased ROM, increased plasma creatine kinase (CK), decreased power and muscular strength.

Assessing Muscle Damage

To assess muscle damage, there are several methods. Maximal voluntary contraction (MVC) torque is related to force, and is one of the more practical assessments. Torque is force times the moment arm, and is dependent on the length-tension curve of the muscle, velocity and joint angles. The best way to measure torque is to control for both joint angle and velocity, through an isometric MVC (81).

Fatigue, motivation, and pain are all disadvantages for using an isometric MVC to measure damage following eccentric induced damage. It can be difficult to differentiate decreases in torque due to muscle injury and those of fatigue (39). Motivation can also be a factor for MVC torque measurements, as there is a concern about whether or not all motor units have been recruited (77). Over the time course of muscle degeneration and regeneration, there is a consistent reduction in MVC torque

(81). Following muscular damage, MVC torque decreases about 60% and takes a period of 1-2 weeks to recover to baseline measures (73).

Range of Motion (ROM) can also be used to assess damage. ROM determines the amount of swelling, which occurs as fluid accumulates in the damaged cells. ROM is joint specific and is dependent on many factors including the amount of skin, musculature, subcutaneous tissue, and bone. Goniometer is the instrument that measures ROM and can be used in two measurements, active and passive ROM. Passive ROM is when the investigator moves the joint through the ROM until pain occurs. Active ROM is how far the individual can move the joint. Reliability of ROM following exercise-induced damage has shown to have a high level of correlation (42).

A needle biopsy, an invasive procedure removing a small (10-50 mg) section of the muscle, can be taken to more accurately measure muscle damage. The sample is examined through an electron or light microscope to look for damage of the Z lines and other structural features. Due to the small size of the biopsy, it is not depictive of the entire injured muscle. As well, biopsies are generally taken from only one muscle, while generally several muscles are injured. MVC torque production does not correlate strongly with needle biopsies, mainly due to the different time courses of damage. MVC torque production occurs immediately while damage at the sarcomere level can take several days to become evident (41). An increase in blood CK levels is associated with needle biopsies, making it difficult to differentiate CK levels due to exercise injury and the biopsy (46).

Conflicting evidence exists for the accuracy of blood markers of muscular damage. One study shows a strong correlation between myoglobin and CK with ROM and MVC torque, only after 24 hours post damage (70). Blood proteins, such as myoglobin and CK have been shown to increase 24 hours after damage (23). This correlates poorly with the known fact that MVC decreases immediately. Individual variability of CK blood levels has also been revealed, which results in CK being an inconsistent injury marker (23). Thus, CK and other blood markers do not properly reflect exercise induced muscle damage.

The visual analog scale (VAS) is a subjective technique used to measure pain and soreness. With VAS, subjects rate the amount of soreness and pain currently experienced. Because of the 24-48 hour delay in DOMS, VAS correlates poorly with functional movements such as MVC torque (49). Another method to assess pain due to soreness is the pain pressure threshold (PPT).

Non-invasive imaging devices can also be used as assessments of exercise induced muscle injury. Devices such as ultrasound, computed tomography (CT) and magnetic resonance imaging (MRI) can be used. CT and MRI are typically used to measure increased volume of localized muscle damage. As with previous assessment tools, CT and MRI imaging poorly correlate with functional measurements due to the delayed onset (49). However, these methods do allow more precise measurements of the volume.

Effects on performance

Generating the greatest amount of power output possible is often the goal for performance activities. When exercise induced muscle damage occurs, changes in power production can occur. Peak power output has been shown to decrease immediately and persist for several days after eccentric exercise (20). A method to measure peak power performance is through the vertical jump, which can also decrease after muscle damage. Stretch-shortening cycle, ground reaction force, muscle and joint stiffness regulation, electromyographic (EMG) activity, and maximal force production have all been studied and found to have reduced measurements following muscle damage (7,8). There is an immediate reduction that can last up to 4 days post-damage for vertical jump performance, both with and without countermovement (19). The squat jump, when compared to the depth jump and countermovement jump, has the longest reduction in jump height (19).

Maximum muscle force recruitment and motor unit activation is reduced after exercise-induced damage. EMG activity showed a decline in muscle force output following damage (7), which suggests greater central activation is required to reach a maximal force or given submaximal force. EMG can be used to reveal if a muscle is more or less active, but is more useful when used to apply the interpolated twitch technique (ITT). ITT measures the percent activation of motor units during voluntary contractions. An estimate of true maximum force can be determined by deduction of the relationship between the evoked and voluntary force. Activation percentages, inferred by ITT, depend upon muscles tested. For instance, the ankle plantar flexors have activation ranging from 80-99% (75) while the quadriceps femoris activation is 85-95% (50).

Recovery Modalities

DOMS symptoms have been treated with recovery modalities such as stretching, massage, and ultrasound. The tested effectiveness of these treatments shows inconsistent results. The more widespread modalities used are stretching and massage. Not one modality studied has seemed to function more efficiently than another. Examined modalities, both alternative and customary, include: whole body vibration (68), aerobic exercise (78), light exercise (4), acupuncture (57), massage (79), stretching (59), and ice massage (85).

Whole-Body Vibration

Synaptic plasticity is the ability of synapses to either increase or decrease signal transduction efficiency in response to changes in their activity (47). Synaptic plasticity can be either short-term or long-term, often referred to as short and long-term potentiation. While synaptic plasticity is a term often applied to physiology and neuroscience, potentiation, specifically short-term, is a concept of importance when discussing WBV.

Potentiation is a heightened or improved state of readiness of muscles often associated with the enhancement of force, which is achieved after repetitive activation of skeletal muscle (45). There are two means of eliciting short-term potentiation: concurrent activation potentiation and post-activation potentiation. Concurrent activation potentiation occurs during the activation of a muscle. Post-activation potentiation follows activation of a muscle.

Concurrent Activation Potentiation

Concurrent activation potentiation (CAP) provides a potential ergogenic advantage, which is associated with simultaneous activation of muscles other than the prime synergists (32). A prime example of CAP is the Jendrassik maneuver where the subject cups and links the fingers of both hands in front of their chest and then pulls strongly outwards across the chest. A larger knee jerk reflex response along with other reflexes should be elicited when the subject is performing the Jendrassik maneuver (JM) (88). In one study, JM was associated with a larger H-reflex than either CAP, or CAP and JM combined (88). CAP works via activation and presynaptic modulation of the H-reflex, and cortical overflow. The H-reflex is elicited by selectively stimulating the sensory Ia fibers, and can demonstrate neural adaptation with training. Cortical overflow works when one part of the motor cortex is active, other areas of the motor cortex are affected, allowing for functional synergy.

Another example of CAP is clenching of the jaw during various exercises. Jaw clenching during CMJs showed a RFD that was 19.5% greater when compared to no clenching (33). Jaw clenching is a type of remote voluntary contraction (RVC), which is proposed to facilitate reflexes via the activation of muscles remote from the reflex (34). Research performed by Ebben yielded 14.6% and 14.8% higher isometric average torque and peak torque when compared to test conditions without RVC.

This study included both young women and men, who had previously participated in sports at a high school level or above. Remote voluntary contractions (RVC) conditions were measured and compared to baseline measurements of maximal measurement concentric isokinetic knee extensions and flexion. RVC conditions included maximal volitional jaw clenching on a mouthpiece, Valsalva maneuver and maximal bilateral hand gripping using hand dynamometers. Ebben et al. concluded that RVC increased the performance of several outcome variable assessed, which coincided with the concomitant increase in the EMG of the prime movers (34).

Post Activation Potentiation

Post activation potentiation (PAP) is a phenomenon by which muscular performance is enhanced as a result of previous contractions. PAP may be attributed to the phosphorylation of myosin regulatory light chains, which results in increased sensitivity of actin and myosin to calcium. This increased sensitivity may increase the rate and velocity of contraction, as myosin regulatory light chains are a protein responsible for contraction. The more responsive myosin-actin binding sites trigger events that lead to an enhanced muscle force output at the structural level, ultimately resulting in faster contraction rates and rates of tension development (31).

PAP results in an increase in alpha motor neuron excitability (33), which is measured via an increase in the H-reflex (5). PAP increases the efficiency and rate of impulses, leading to a greater recruitment of motor neurons. PAP also occurs at the spinal level, with an increase in the synaptic efficiency between Ia afferent terminals and alpha-motoneurons (47).

Post activation potential caused by the acute exposure of WBV may cause enhanced performance (25). WBV is being utilized as a warm-up for its PAP effect prior to performance. To prevent injury and prepare the body for activity a warm-up is often performed. As an alternative to traditional active warm-up methods, WBV is being utilized as an active passive warm-up method (24, 26, 69). Cormie used WBV as an active passive warm-up by applying 30 seconds of WBV prior to performing a jump.

For power sports, acute lower body WBV may be beneficial for activating the neuromuscular system (1, 2). Initial strength and power gains during weight training are attributable to neuromuscular facilitation. In one study, individuals performed bicep curls on a pulley system with vibration delivered through the cables. In elite and amateur athletes, a respective 10.4% and 7.9% increase in maximal power was measured (52). Neural factors include increased recruitment, synchronization, muscular coordination and proprioceptor response (2). These neural factors may improve with traditional weight training or WBV.

Methods of PAP

Complex Training

Complex training involves the incorporation of principles of PAP to induce long-term neuromuscular adaptations. Such an adaptation could be the rate of force development (RFD) in muscle performance. Complex training is performing heavy resistance exercises (HRE) prior to an explosive movement, which involves similar biomechanical characteristics. It is assumed that the HRE will cause a PAP reaction and increase the performance of the latter exercise (47). Prerequisite strength and the intensity of the load used in the weight training portion of the complex may be important in eliciting a complex training effect (34). When this complex training is continued for a long period, it is believed there will be long-term alterations in the ability of the muscle to produce power, as evident in performances such as vertical jump height. It is also believed to enhance short-term performance.

A common complex training pair is heavy back squats with vertical countermovement jumps (CMJs) (47). There are some studies supporting the idea of complex training. For instance, performance of 3-5 repetitions at 90-100% of 1 maximal voluntary isometric increase in a single leg-press position resulted in a 1.4 cm increase in the mean of eight CMJs (86). A correlation has been found linking a greater proportion of type II fibers to greater individual performance benefits due to PAP (45).

Whole-Body Vibration

Vibration is a mechanical stimulus consisting of oscillatory motions. Frequency and amplitude of the vibration are defining characteristics. The exact mechanism for whole-body vibration (WBV) is still unknown, although it is believed to be associated with PAP. WBV has been shown to have significant performance and clinical benefits. One suggestion is that the enhanced performance occurs from amplified muscle spindle sensitivity and gamma activation, which leads to an increase in motor unit recruitment and neuromuscular facilitation (67). Adaptations similar to resistance training have been observed, thought to be due to an increased neuromuscular activation (16). The increase in strength may be attributed to the Ia afferent mediated myotatic reflex contraction, which activate, via large alpha-motor neurons, mainly type II muscle fibers (69). The tonic reflex, a sustained muscular contraction, has also been observed when vibration is applied directly to the muscle (71).

Variables of WBV– frequency, amplitude, duration and rest intervals– can be adjusted to optimize performance. Optimal rest intervals are vital for the greatest enhancement of sport performance from WBV. If the rest period is too short, it is possible to over-stimulate the neuromuscular system while the positive effects of WBV may dissipate with too long of a rest (2). Rest intervals following acute bouts of WBV have been chiefly studied in intervals ranging from immediate to 10 minutes (2,12, 18, 25, 26). These studies have revealed conflicting results. It has been shown that for optimal enhancement of performance, highly individualized rest periods are necessary (27).

WBV, when the variables are suitably optimized for an individual, has the potential for performance and training applications in athletes. For athletes that have a short one-time performance (i.e. track (high jump, sprints)), WBV can be implemented immediately prior to performance. For athletes that have longer performances, training with WBV may be suitable. With conditioning, WBV can be used to train the muscles to produce greater peak power output, which can be carried over to future athletic performance. Increased proprioceptive abilities such as sprint speed, greater force production or even greater VJH can be attained with WBV training and utilized in athletic performances such as basketball.

Pain

WBV is also thought to inhibit pain receptors, allowing individuals to be more tolerant of pain (68). It has been suggested that vibration may influence the activation of afferent input from sensory units of the muscle fibers. It is also associated with the removal of metabolic wastes, increased lymphatic blood flow and attenuated pain sensation associated with exercise (36, 55, 58).

One theory proposed was that the activity in large diameter sensory fibers interacts with impulse transmissions in pain pathways, which alleviates pain (60). It is not certain what kind of receptive units are excited by the vibratory stimulus, but it is thought to be both superficial and deep cutaneous mechanoreceptors (60). The Pacinian corpuscles, which perceive deep pressure, such as vibrations, are primarily though to be affected by WBV. Vibratory stimulation is believed to depress the excitability of motoneurons innervating the antagonistic muscle via reciprocal inhibition, which reduces the perceived pain when vibration was applied directly to the antagonistic muscle (44). These findings are consistent with the gate control hypothesis (64), which states that afferent signals that are mediated by large myelinated fibers inhibit small pain fibers presynaptically in the dorsal horn of the spinal cord.

Research conducted concerning WBV relief in symptoms of exercise-induced pain reveals positive effects of reduced pain associated with both unexercised muscle (83, 84) and exercised muscle (60, 84). It has been shown that after DOMS has set in, 24 hours post-injury (53), perceived pain from local pressure increased with vibration. It was suggested that it sensitizes nociceptors to the point where they become vibration responsive (64, 83). It has also been proposed that activity in large diameter sensory fibers interacts with impulse transmission in pain pathways. Activation of Pacinian corpuscles may contribute to reduced pain, as when moderate pressure and cushion were applied along with vibration a more effective pain reduction was observed

(60). Additional research is needed, as there are conflicting conclusions involving vibration and the alleviation of pain.

Performance

It is important to enhance performance in both athletes and recreationally trained athletes. Greater power production is an essential portion of athletic performance. Studies have shown a significant increase in average power, maximal power, and mechanical power following bouts of vibration by facilitation of an explosive strength effort (13). Including non-traditional techniques, such as WBV, may enhance traditional techniques such as strength training, plyometrics, and weightlifting (26, 30). Plyometric training is associated with an increase in jump height and may be associated with an increase in sprinting speed (3). It appears that when training, improvements in performance measurements are more likely to occur when biomechanically and metabolically specific movements are employed (3). Increased performance in both upper and lower body muscular activity has been described with WBV, and can be found with both trained and untrained individuals (13, 18, 24, 25).

WBV exposure has been shown to be safe at a moderate intensity. WBV has been associated with a variety of positive effects including the following: stimulating the neuromuscular system (24), inducing non-voluntary muscle contractions (51) and increasing power production when connected with an explosive strength effort (13, 52). The latter leads to an enhancement of performance via motor function and muscular strength (14).

Sprinting and jumping performance has also increased after bouts of WBV (2, 12, 18, 26). Cormie et al. had moderately resistance-trained men serve as subjects in a study which measured power and height differences during CMJ and isometric squats when vibration was applied. Each subject served as his own control for both exercises. The primary finding of the study was that the jump height was significantly increased following vibration, although there was not a significant difference in other variables measured in the study.

Bosco et al. did a small-scale study with female volleyball players who played on the national level and measured average force, power and velocity of a single leg press with and without a vibration treatment. Each female served as her own control, with one leg being assigned the vibration and the other serving as the control. It was found that there was a significant change in the Velocity-Force and Power-Force relationships, and the Velocity-Force and Power-Force curves were shifted to the right. The improvements in performance only lasted about 10 minutes (14).

No or little effort is required by the subjects to achieve the enhanced acute performance (69). Contrasting studies do exist, with WBV being shown to not increase performance and only having the effectiveness similar to traditional training techniques (24, 25, 26, 30, 54).

It appears that there may be a higher sensitivity of muscle receptors and the CNS of trained athletes when compared to those untrained. This has resulted in greater increases in maximal power when vibration is applied to trained individuals (51). It is also known that stretched muscles are more sensitive to vibrations and contract more strongly (44). These differences and others still yet to be identified may contribute to conflicting results concerning benefits of training with vibration.

Whole-Body Vibration and Recovery

Recently, there have been several studies concerning WBV as a muscle recovery modality prior or following muscle injury (4, 10, 55, 68). A variety of measurements can be used to assess exercise induced muscle damage, which can be used in determining the time course of muscle recovery. WBV has been proposed to increase muscle spindle activity and muscle pre-activation, which is a lower firing threshold. Less disruption in the excitation-contraction coupling (4,10, 55) is the result. A larger number of motor units and muscle fibers would be recruited following the muscle pre-activation. This could lead to a reduction in myofibrillar stress during repeated muscle contractions, accelerating the muscle recovery (14).

In a particular study with elbow flexors, investigators discovered that vibration was effective for lessening DOMS. There was a decrease in soreness both immediately before and after vibration, and over a time course of seven days. Furthermore, an increase in ROM measurement over a time course of seven days was recorded. The vibration treatment had no effect on CK activity, swelling, and recovery of muscle strength (55). Similar results were recorded in other studies, with a lower perceived pain in the vibration treatment group compared to the control group, which proposes that WBV inhibits pain receptors (68). It was suggested that WBV stimulates blood flow to the musculature by increasing the disposal of metabolic waste (58). A decrease in soreness, isokinetic force, PPT, and plasma CK activity was detected when vibration was administered prior to muscle damage (4,10). When WBV is performed prior to muscle damage, it acts as more of a protective mechanism. This occurs due to a heightened sensitivity of the musculature, which allows for lesser amount of damage to occur (4). Although positive effects of WBV as a recovery mechanism have been observed, more investigation is needed. There are many different ways that studies regarding WBV as a recovery mechanism for DOMS have been structured. Differences include selection of subjects (untrained, recreationally trained and elite athletes) and whether vibration is included pre- or post damage. To our knowledge, there have not been any previously published studies concerning recreationally trained athletes and WBV applied as a mechanism for recovery post-damage.

CHAPTER III: METHODS

Participants

Thirty recreationally trained females (age 21 ± 1.9 yrs., height 165.69 ± 7.3 cm, mass 58.69 ± 10.95 kg) volunteered and twenty-seven females completed 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 was excluded. Any participant taking any medications that alter balance, musculoskeletal system, or central nervous system functions relating to posture and motor control or those taking prescription pain and/or psychiatric medication were also excluded. Participants were screened by questionnaire for potential risk factors to this exercise protocol such as bruising easily, rhabdomyolysis etc. Participants were asked abstain from lower body exercise and from pain medication 48 hours prior to testing sessions and during all testing days. Furthermore, participants were asked to keep all food and water intake consistent during the study. To avoid failure of the above requests, participants were not scheduled for testing during their menstrual cycle.

Measures

Pressure Pain Threshold (PPT)

PPT was assessed in all 7 visits, and assessed in the left quadriceps while participants were seated comfortably on a table, with the feet dangling over the edge and not touching the ground. To ensure consistency, marks were made on the belly of the rectus femoris (RF), at the mid-point between the patella and the proximal head of the femur. The participants were instructed to keep the quadriceps relaxed while the researcher placed a pressure algometer (Wagner Instruments, Greenwich, CT USA) on each test site. Mechanical pressure was applied to the muscle in the following order: VM, VL, RF during three trials and 20 seconds between each trial. Participants were asked to indicate when the pressure transitioned from being "uncomfortable" to "painful." The researcher immediately removed the pressure stimulus when the participant said "pain." The corresponding force value was recorded and all three trials for each muscle were averaged for each participant.

Vertical Jump (VJ)

VJ 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 1080 Hz. The Vertec® device was used solely to measure the VJH, while the Bertec® plate collected data concerning force, which was later used to calculate GRF_Z. Participants were instructed to perform three maximal CMVJ, with 15 s rest between, 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. VJ height was calculated by the difference between maximum jump reach and standing reach. Peak Power Output (PPO) was calculated via the Sayers Equation [PAPw = (60.7 x jump height (cm)) + (45.3 x body mass (kg)) – 2055] (Sayers, cross validation).

Using the force plate, GRF_Z and $rGRF_Z$ were calculated. Ground Reaction Force (GRF) is the force exerted by the ground on a body in contact with it. The subscript Z denotes that the GRF was measured in the vertical direction, in the Z plane.

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 the three-familiarization sessions, participants visited the laboratory for four consecutive days and were randomly assigned to the control or WBV treatment 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. All participants were assessed for baseline PPT's. After baseline measures were taken, participants performed an exercise induced muscle damage protocol. The protocol consisted of split squats using a Jones Machine® and 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 participant's body weight. During split squats, the back leg was placed on a bench for support with 90-degrees of flexion, which allowed focus on single-leg performance of the front leg. Researchers provided assistance on the concentric phase after the participants reached 90-degrees 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 two sets of body weight quarter squats on a flat surface, with 30 seconds of squats paired with 30 seconds of rest. Participants in the treatment 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 30 Hz with amplitude of 2-4 mm. Following treatment or control, participants were assessed for PPT's and VJ. Participants then rested for 10 minutes 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.

Participants returned to the laboratory 24,48, and 72 hours following muscle damage protocol to evaluate muscle pain on movement and VJ performance. These sessions consisted of initial assessment of PPT's and VJ followed immediately by the treatment or control protocol. After the treatment or control protocol, all measurements were re-taken, followed by a 10-minute rest period and a third set of measurements.

Reliability of Measurements

Three days of measurements were obtained during familiarization sessions and a set of baseline measures on the first testing day for rectus femoris PPT and vertical jump performance. Measurements 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 and for VJ performance were 0.92, respectively.

Data Analyses

To test changes in PPT's over time and between groups a 12x2 (time by group) mixed factor analysis of variance (ANOVA) was conducted. Time was 0Pre, 0Post1, 0Post2, 24 Pre, 24Post1, 24Post2, 48Pre, 48Post1, 48Post2, 72Pre,72Post1, and 72Post2 and the groups were WBV and control. 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 least significance difference (LSD) post-hoc analyses for pairwise difference.

To test changes in VJH and peak Z over time and between treatment groups, a 8x2 (time by group) mixed factor analysis of variance (ANOVA) was conducted. Groups were defined as WBV and control. Time was defined as Day0Pre, Day0Post, Day24Pre, Day24Post, Day48Pre, Day48Post, Day72Pre, and Day72Post. Similar to PPT analysis, interactions were followed up with one-way ANOVAs while main effects were followed up with LSD post-hoc analyses for pairwise differences. Again, all analyses were conducted using SPSS software (SPSS 21,IBM, Rochester, NY) and statistical significance was determined as a p-value less than 0.05.

All analyses were conducted using SPSS software (SPSS 21,IBM, Rochester, NY), when sphericity was violated. The Greenhouse-Geisser correction of degrees of freedom was used. Statistical significance was defined as a p-value less than 0.05 and eta squared was calculated to determine effect sizes.

CHAPTER IV: RESULTS

Vertical Jump Height

No significant (p>0.05) interaction was found for VJH. There were no significant (p>0.05) main effects for group but there were significant (p=0.001) main effects for time.

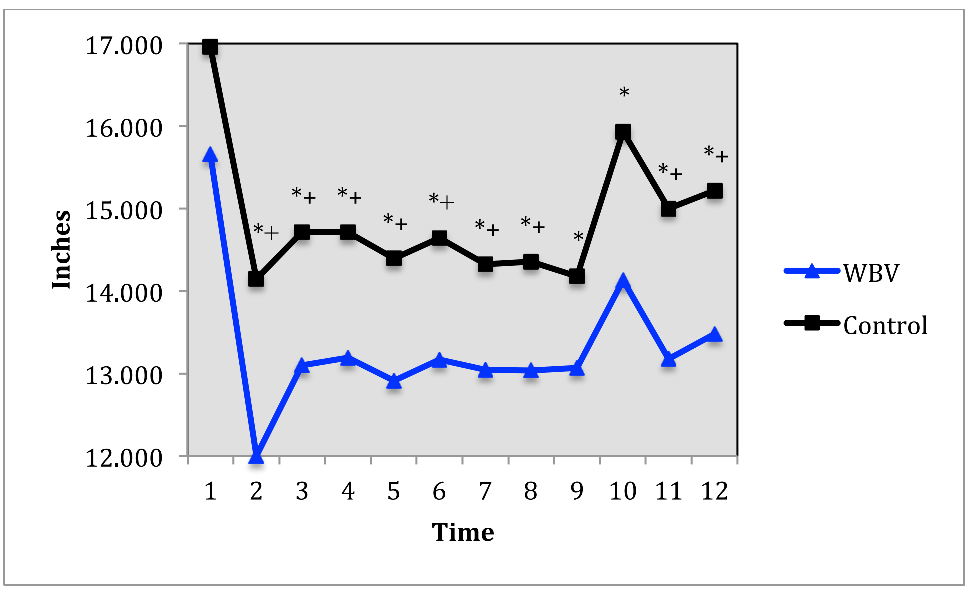

Figure I. Estimated Mean VJH. Means of VJH between groups and across time following exercise induced muscle damage. For VJH, 0Pre was greater than all other time points, and 72Pre was greater than 0Post, 24Pre, 24Post, 48Pre, 48 Post and 72Post.

Significant (p<0.05) differences from 0Pre are indicated with *. Significant (p<0.05) differences from 72Pre are indicated with +

Peak Z Force

No significant (p> 0.05) interaction was found for peak Z force. There was no significant (p>0.05) main effect for group but there was a significant (p <0.001) main effect for time.

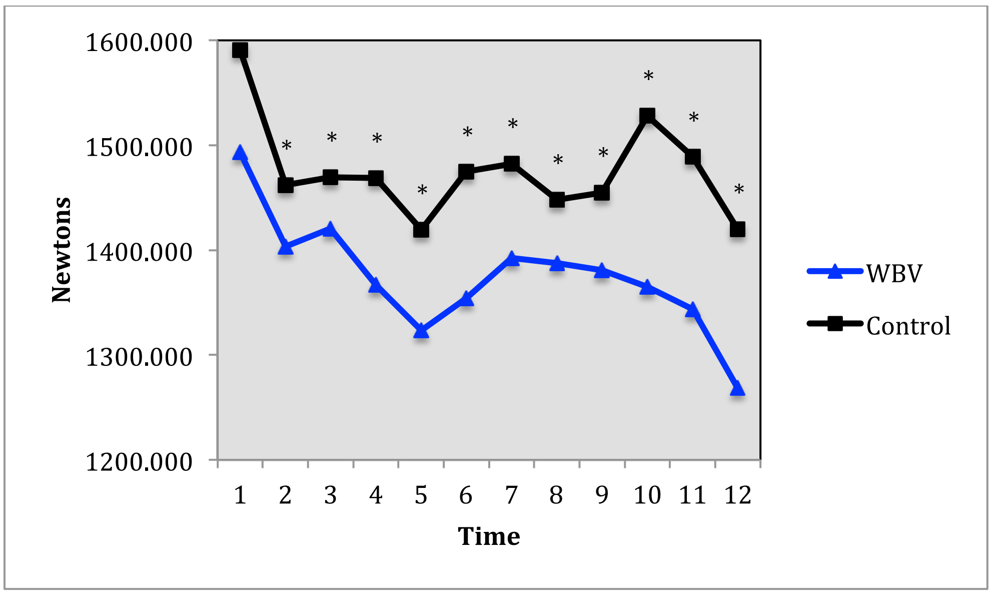

Figure II. Estimated Mean Peak Z Force in RF. Means of estimated peak Z force between groups and across all time points following exercise induced muscle damage. Peak Z force significant main effects were for time was that 0Pre was greater than all other time points. Significant (p<0.05) main effects from 0Pre are indicated with *.

PPT in RF

No significant (p>0.05) interaction of time by group was found for RF PPT. There was a significant main effect for time (p=0.002) but no significant main effect for group (p>0.05).

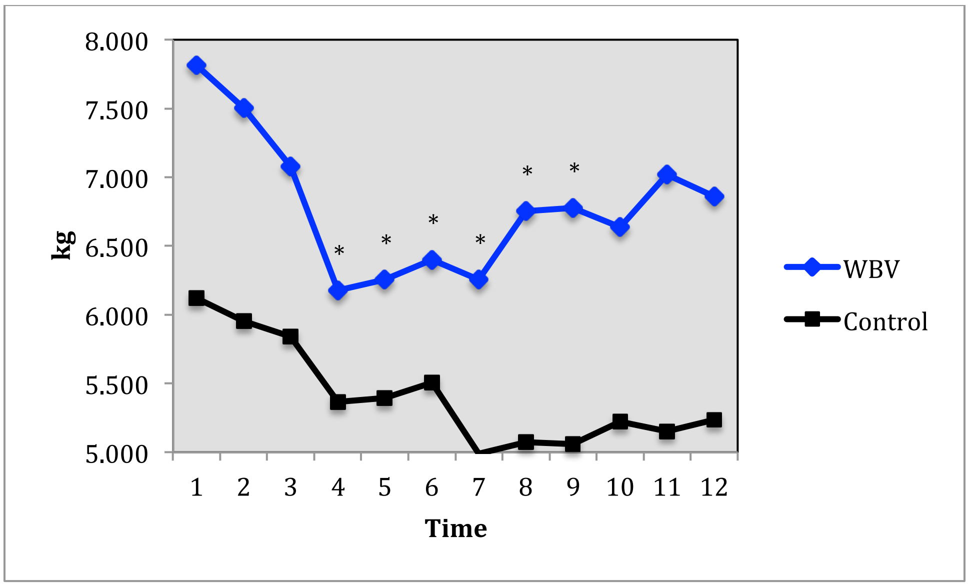

Figure III. Estimated Mean PPT. Means of PPT for RF between groups and across all time points following exercise induced muscle damage. RF PPT's significant main effects were for time was that 0Pre was greater than all time points at 24 and 48 hours Significant (p<0.05) main effects from 0Pre are indicated with *.

Legend for Figures I-III

1: 0 Pre

2: 0 Post 1

3: 0 Post 2

4: 24 Pre

5: 24 Post 1

6: 24 Post 2

7: 48 Pre

8: 48 Post 1

9: 48 Post 2

10: 72 Pre

11: 72 Post 1

12: 72 Post 2

CHAPTER V: DISCUSSION

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

Another aim of the investigation was to determine the effect of WBV following EIMD on VJ performance. The EIMD protocol resulted in an immediate and prolonged detrimental effect on VJ performance. However, no differences were found between WBV and control groups. DOMS peaked at 48 hours post injury, while the main performance findings were that VJH, and peak Z force both decreased over time, indicative of decreased performance. To our knowledge, no previous research has investigated the effects of WBV on VJ performance following EIMD. Current research has either examined the effects of EIMD on VJ performance without WBV (20), the effects of WBV on VJ performance without muscle damage (2, 11, 15, 18, 24, 26, 27, 56), or the effects of WBV on muscle recovery (4, 10, 55, 68, 83), which characterizes this investigation as novel in the performance and muscle recovery literature.

In the current investigation, incorporating WBV as a recovery modality aimed at attenuating any reduction in performance was not successful as measured by VJH and peak Z. Previous literature has shown mixed results when examining the effects of WBV on VJ performance. Some research has shown increases in VJH and peak Z force following WBV exposure (2, 12, 26, 27) indicating neuromuscular facilitation or a potentiation effect. It appears that when muscle is damaged, it alters the effectiveness of WBV during VJ performance. As previously mentioned, WBV has been researched as a recovery modality in the upper (55) and lower extremities (68) when measuring pain, force production and clinical variables with different damage and vibration protocols but has not been investigated for VJ performance effects. These mixed results are most likely due to the use of varying damage protocols, vibration exposures, and extremities tested.

It is necessary to discuss how the potentiating mechanism of the stretch shortening cycle during a VJ attenuates the detrimental performance effects of EIMD. It has been suggested that excitation-contraction coupling is impaired following muscle damage (35), decreasing the release of calcium per action potential (6), leading to an inability to activate force-generating structures. It is proposed that after EIMD, a reduction in stretch reflex sensitivity and muscle stiffness occurs (48), which leads to decreased force potentiating mechanisms during the stretch shortening cycle. Since the stretch shortening cycle is a key component in CMVJ, this may help explain our findings of decreased VJH, and peak Z force following EIMD.

The changes in muscle pain ratings during movement and PPT's observed in the present study are consistent with previous literature following EIMD (4,55,68). Some research shows group difference from WBV and control groups in muscle pain (4, 55, 68) indicating that WBV aids in reducing muscle pain after EIMD. Muscle damage protocols varied in these studies, some used 6 sets of 10 repetitions of eccentric only exercises on an isokinetic dynometer (4, 55) in the elbow flexors (55) and knee flexors (4). Another study used a combination of resistance training, running and sprints to induce muscle damage (68). These studies also used different forms of vibration, such as direct vibration via a handheld device (55) while others used WBV platforms (4, 68). In the current investigation, hip extensors and 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. Since upper and lower body musculature may respond differently and different exposures of vibration may elicit different responses as well.

In clinical pain populations, some potential mechanisms have been suggested that inhibits pain receptors, allowing for individuals to be more tolerant to pain (68). It is proposed that vibration receptors in the skin stimulate inhibitory interneurons in the spinal cord, which reduce the amount of pain signals transmitted to the brain (64). In the 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, increased pain threshold (64). It has been shown that vibration applied to an unexercised muscle reduces the perceived level of pain from local pressure (83, 84) while also showing reduced pain during muscle vibration in individuals suffering from chronic muscle pain (60,83), supporting the gate control hypothesis (64). However, it has been shown that when DOMS is present, at 24 hours, perceived pain from local pressure increased with vibration (53). The authors suggest this was due to sensitization of nociceptors to the point where they became vibration responsive (64, 83). In the current investigation, no differences in muscle pain when WBV was applied were found.

Previous research has studied several ways to control or prevent EIMD symptoms (21). Decreasing these symptoms in individuals is critical in many populations. In exercising, physically active individuals, decreased swelling, pain and stiffness for any period of time is helpful in pain management and enabling activities of daily living. It may be plausible that WBV may be more effective for generally healthy recreational individuals and direct vibration may be more effective in pain management for injured individuals or those clinically diagnosed with pain, 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 (21). Recently, WBV has been explored as a potential modality in treating symptoms associated with EIMD. It is important to note that the timing of when vibration is utilized may contribute to different findings in the literature, as several studies have reported that maximal effects of WBV on VJ performance are within 10 minutes post-treatment (2, 27). Whether WBV is more effective prior to muscle damage or after needs to be investigated further. The literature is sparse and conflicting on findings involving vibrations and alleviation of muscle pain during movement.

The present investigation provides a novel exercise in producing EIMD in the quadriceps, that to our knowledge has not been previously established. As well, investigating muscle pain during movement, PPT in recreationally trained individuals on the lower body effects of alleviating pain with WBV has not previously been done. The research is consistent with other investigations indicating that our participants did experience EIMD in quadriceps. This allows us to be confident that WBV exposure does not effectively aid in muscle pain management in health recreationally trained females, as found in our study. Future research should investigate a variety of populations (i.e. chronic and acute pain patients, recreationally trained males, and athletically trained individuals) for treatment in alleviating muscle pain.

Previous research supports our findings that following EIMD VJ performance decreases immediately and up to 3 days after (20), irrespective of WBV treatment. In one investigation of VJ performance following EIMD, they found decreases in squat jump height, depth jump height and counter-movement jump height following damage and for 3 days after (20). The present study extends findings by measuring peak Z force. Since the current finding of peak Z force has similar trends as VJH, it may be expected that these trends would be similar in other jump performance studies.

There are three studies that support the possibility of a reduction in DOMS when a vibration treatment is applied (4, 10, 55). Each study differed in methods of the procedure from this study. One study applied vibration prior to damage (10), another applied vibration for 30 consecutive minutes (55) and the last utilized direct vibration for non-athletic subjects (4). The differences in procedures could contribute to the discrepancies in results. There is the possibility that the use of split-squats to failure created much more damage when compared to other exercises, such as walking on a treadmill at a decline. Application of direct vibration may also be important, as it may be more effective in treating DOMS, as more of the vibrations are applied to the damaged muscle.

In conclusion, it appears that WBV has no effect on VJH, PPT or peak Z force following EIMD. 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 EIMD. Different levels of soreness caused by EIMD 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 be examined with similar protocols to determine effects of different participant populations.

REFERENCES

1. Abercromby, AF, Amonette, WE, Layne, CS, McFarlin, BK, Hinman, MR and Paloski, WH. Variation in neuromuscular responses during acute whole-body vibration exercise. Med Sci Sports Exerc 39: 1642-1650, 2007.

2. Adams, JB, Edwards, D, Serviette, D, Bedient, AM, Huntsman, E, Jacobs, KA, Del Rossi, G, Roos, BA and Signorile, JF. Optimal frequency, displacement, duration, and recovery patterns to maximize power output following acute wholebody vibration. J Strength Cond Res 23: 237-245, 2009.

3. Alptekin, Ahmet, Özlem Kılıç, and Mehmet Maviş. The effect of an 8-week plyometric training program on sprint and jumping performance. *Serbian Journal of Sports Sciences* 7.2 2013.

4. Aminian-Far, A, Hadian, MR, Olyaei, G, Talebian, S and Bakhtiary, AH. Wholebody vibration and the prevention and treatment of delayed-onset muscle soreness. J Athl Train 46: 43-49, 2011.

5. Armstrong, RB, Warren, GL and Warren, JA. Mechanisms of exercise-induced muscle fibre injury. Sports Med 12: 184-207, 1991.

6. Armstrong, RB. Initial events in exercise-induced muscular injury. Med Sci Sports Exerc 22: 429-435, 1990.

7. Avela, J and Komi, PV. Reduced stretch reflex sensitivity and muscle stiffness after long-lasting stretch-shortening cycle exercise in humans. Eur J Appl Physiol Occup Physiol 78: 403-410, 1998.

8. Avela, J, Kyrolainen, H, Komi, PV and Rama, D. Reduced reflex sensitivity persists several days after long-lasting stretch-shortening cycle exercise. J Appl Physiol 86: 1292-1300, 1999.

9. Ayles, S, Graven-Nielsen, T and Gibson, W. Vibration-induced afferent activity augments delayed onset muscle allodynia. J Pain 12: 884-891. 2011.

10. Bakhtiary, AH, Safavi-Farokhi, Z and Aminian-Far, A. Influence of vibration on delayed onset of muscle soreness following eccentric exercise. Br J Sports Med 41: 145-148, 2007.

11. Bazett-Jones, D, Finch, H and Dugan, E. Comparing the effects of various wholebody vibration accelerations on countermovement jump perfomance. J Sci Med Sport 7: 144-150, 2008.

12. Bedient, AM, Adams, JB, Edwards, D, Serviette, D, Huntsman, E, Mow, SE, Roos, BA and Signorile, JF. Displacement and Frequency for Maximizing Power Output Resulting From a Bout of Whole-Body Vibration. J Strength Cond Res 23: 1683-1687, 2009.

13. Bosco, C, Cardinale, M and Tsarpela, O. Influence of vibration on mechanical power and electromyogram activity in human arm flexor muscles. Eur J Appl Physiol Occup Physiol 79: 306-311, 1999.

14. Bosco, C, Colli, R, Introini, E, Cardinale, M, Tsarpela, O, Madella, A, Tihanyi, J and Viru, A. Adaptive responses of human skeletal muscle to vibration exposure. Clin Physiol 19: 183-187, 1999.

15. Bosco, C, Cardinale, M, Tsarpela, O, Colli, R, Tihanyi, J, VonDuvillard, S and Viru, A. The influence of whole body vibration on jumping performance. Bio Sports 15: 157-164, 1998.

16. Bosco, Carmelo, M. Iacovelli, O. Tsarpela, M. Cardinale, M. Bonifazi, J. Tihanyi, M. Viru, A. De Lorenzo, and A. Viru. Hormonal Responses to Whole-body Vibration in Men. *European Journal of Applied Physiology* 81.6: 449-54. 2000.

17. Broadbent, S, Rousseau, JJ, Thorp, RM, Choate, SL, Jackson, FS and Rowlands, DS. Vibration therapy reduces plasma IL6 and muscle soreness after downhill running. Br J Sports Med 44: 888-894, 2008.

18. Bullock, N, Martin, DT, Ross, A, Rosemond, CD, Jordan, MJ and Marino, FE. Acute effect of whole-body vibration on sprint and jumping performance in elite skeleton athletes. J Strength Cond Res 22: 1371-1374, 2008.

19. Byrne, C and Eston, R. The effect of exercise-induced muscle damage on isometric and dynamic knee extensor strength and vertical jump performance. J Sports Sci 20: 417-425, 2002.

20. Byrne, C and Eston, R. Maximal-intensity isometric and dynamic exercise performance after eccentric muscle actions. J Sports Sci 20: 951-959, 2002.

21. Cheung, K, Hume, P and Maxwell, L. Delayed onset muscle soreness : treatment strategies and performance factors. Sports Med 33: 145-164, 2003.

22. Clarkson, PM, Nosaka, K and Braun, B. Muscle function after exercise-induced muscle damage and rapid adaptation. Med Sci Sports Exerc 24: 512-520, 1992.

23. Clarkson, PM and Ebbeling, C. Investigation of serum creatine kinase variability after muscle-damaging exercise. Clin Sci (Lond) 75: 257-261, 1988.

24. Cochrane, DJ, Legg, SJ and Hooker, MJ. The short-term effect of whole-body vibration training on vertical jump, sprint, and agility performance. J Strength Cond Res 18: 828-832, 2004.

25. Cochrane, DJ and Stannard, SR. Acute whole body vibration training increases vertical jump and flexibility performance in elite female field hockey players. Br J Sports Med 39: 860-865, 2005.

26. Cormie, P, Deane, RS, Triplett, NT and McBride, JM. Acute effects of whole body vibration on muscle activity, strength, and power. J Strength Cond Res 20: 257-261, 2006.

27. Dabbs, NC, Munoz, CX, Tran, TT, Brown, LE and Bottaro, M. Effect of different rest intervals after whole-body vibration on vertical jump performance. J Strength Cond Res 25: 662-667, 2011.

28. Davis, WJ, Wood, DT, Andrews, RG, Elkind, LM and Davis, WB. Elimination of delayed-onset muscle soreness by pre-resistance cardioacceleration before each set. J Strength Cond Res 22: 212-225, 2008.

29. da Silva, MA, Nunez, V and Vaamonde, D. Effect of different frequencies of whole body vibration on muscular performance. . Biology of Sport 23: 267-281, 2006.

30. de Ruiter, CJ, van der Linden, RM, van der Zijden, MJ, Hollander, AP and de Haan, A. Short-term effects of whole-body vibration on maximal voluntary isometric knee extensor force and rate of force rise. Eur J Appl Physiol 88: 472-475, 2003.

31. Desmedt, J. E., & Godaux, E. Recruitment patterns of single motor units in the human masseter muscle during brisk jaw clenching. Archives of oral biology, 24(2), 171-178. 1979.

32. Ebben, WP. A brief review of concurrent activation potentiation: theoretical and practical constructs. J Strength Cond Res 20: 985-991, 2006.

33. Ebben, W. P., Flanagan, E. P., & Jensen, R. L. Jaw clenching results in concurrent activation potentiation during the countermovement jump. *The Journal of Strength & Conditioning Research*, *22*(6), 1850-1854. 2008.

34. Ebben, W., Petushek, E., Fauth, M., & Garceau, L. EMG analysis of concurrent activation potentiation. *Medicine and science in sports and exercise*. 2010.

35. Edwards, R, Hill, D, Jones, D and Merton, P. Fatigue of Long Duration in Human Skeletal Muscle After Exercise. . Journal of Physiology 272: 769-778, 1977.

36. Edge, J, Mundel, T, Weir, K and Cochrane, DJ. The effects of acute whole body vibration as a recovery modality following high-intensity interval training in welltrained, middle-aged runners. Eur J Appl Physiol 105: 421-428, 2009.

37. Enoka, RM. Eccentric contractions require unique activation strategies by the nervous system. J Appl Physiol 81: 2339-2346, 1996.

38. Fagnani, F, Giombini, A, Di Cesare, A, Pigozzi, F and Di Salvo, V. The effects of a whole-body vibration program on muscle performance and flexibility in female athletes. Am J Phys Med Rehabil 85: 956-962, 2006.

39. Fitts, RH. Cellular mechanisms of muscle fatigue. Physiol Rev 74: 49-94, 1994.

40. Friden, J and Lieber, RL. Structural and mechanical basis of exercise-induced muscle injury. Med Sci Sports Exerc 24: 521-530, 1992.

41. Friden, J, Sjostrom, M and Ekblom, B. Myofibrillar damage following intense eccentric exercise in man. Int J Sports Med 4: 170-176, 1983.

42. Gajdosik, RL and Bohannon, RW. Clinical measurement of range of motion. Review of goniometry emphasizing reliability and validity. Phys Ther 67: 1867-1872, 1987.

43. Gibson, W, Arendt-Nielsen, L and Graven-Nielsen, T. Delayed onset muscle soreness at tendon-bone junction and muscle tissue is associated with facilitated referred pain. Exp Brain Res 174: 351-360, 2006.

44. Hagbarth, K. E., Burke, D. A. V. I. D., Löfstedt, L., & Wallin, B. G. The responses of human muscle spindle endings to vibration of non-contracting muscles. The Journal of physiology, 261(3), 673-693. 1976.

45. Hamada, T., Sale, D. G., MacDougall, J. D., & Tarnopolsky, M. A. Postactivation potentiation, fiber type, and twitch contraction time in human knee extensor muscles. Journal of Applied Physiology, 88(6), 2131-2137. 2000.

46. Hikida, RS, Staron, RS, Hagerman, FC, Leonardi, M, Gilders, R, Falkel, J,

Murray, T and Appell, K. Serum creatine kinase activity and its changes after a muscle biopsy. Clin Physiol 11: 51-59, 1991.

47. Hodgson, M, Docherty, D and Robbins, D. Post-activation potentiation: underlying physiology and implications for motor performance. Sports Med 35: 585-595, 2005.

48. Horita, T, Komi, PV, Nicol, C and Kyrolainen, H. Stretch shortening cycle fatigue: interactions among joint stiffness, reflex, and muscle mechanical performance in the drop jump [corrected]. Eur J Appl Physiol Occup Physiol 73: 393-403, 1996.

49. Howell, JN, Chleboun, G and Conatser, R. Muscle stiffness, strength loss, swelling and soreness following exercise-induced injury in humans. J Physiol 464: 183-196, 1993.

50. Huber, A, Suter, E and Herzog, W. Inhibition of the quadriceps muscles in elite male volleyball players. J Sports Sci 16: 281-289, 1998.

51. Issurin, VB. Vibrations and their applications in sport: a review. J Sports Med Phys Fitness 45: 424-436, 2005.

52. Issurin, VB and Tenenbaum, G. Acute and residual effects of vibratory stimulation on explosive strength in elite and amateur athletes. J Sports Sci 17: 177-182, 1999.

53. Kakigi, R and Shibasaki, H. Mechanisms of pain relief by vibration and movement. J Neurol Neurosurg Psychiatry 55: 282-286, 1992.

54. Kelly, Stephen B., et al. "The effect of warm-up with whole-body vibration vs. cycle ergometry on isokinetic dynamometry." *The Journal of Strength & Conditioning Research* 24.11: 3140-3143. 2010.

55. Lau, WY and Nosaka, K. Effect of vibration treatment on symptoms associated with eccentric exercise-induced muscle damage. Am J Phys Med Rehabil 90: 648-657, 2011.

56. Lamont, HS, Cramer, JT, Bemben, DA, Shehab, RL, Anderson, MA and Bemben, MG. The acute effect of whole-body low-frequency vibration on countermovement vertical jump performance in college-aged men. J Strength Cond Res 24: 3433-3442, 2010.

57. Lin, JG and Yang, SH. Effects of acupuncture on exercise-induced muscle soreness and serum creatine kinase activity. Am J Chin Med 27: 299-305, 1999.

58. Lohman, EB, 3rd, Petrofsky, JS, Maloney-Hinds, C, Betts-Schwab, H and Thorpe, D. The effect of whole body vibration on lower extremity skin blood flow

in normal subjects. Med Sci Monit 13: CR71-76, 2007.

59. Lund, H, Vestergaard-Poulsen, P, Kanstrup, IL and Sejrsen, P. The effect of passive stretching on delayed onset muscle soreness, and other detrimental effects following eccentric exercise. Scand J Med Sci Sports 8: 216-221, 1998.

60. Lundeberg, T, Nordemar, R and Ottoson, D. Pain alleviation by vibratory stimulation. Pain 20: 25-44, 1984.

61. Mackey, A. L., Bojsen-Moller, J., Qvortrup, K., Langberg, H., Suetta, C., Kalliokoski, K. K., ... & Magnusson, S. P. Evidence of skeletal muscle damage following electrically stimulated isometric muscle contractions in humans. Journal of Applied Physiology, 105(5), 1620-1627. 2008.

62. McHugh, MP, Connolly, DA, Eston, RG and Gleim, GW. Exercise-induced muscle damage and potential mechanisms for the repeated bout effect. Sports Med 27: 157-170, 1999.

63. McKune, Andrew, Stuart Semple, and Edith Peters-Futre. "Acute Exercise-Induced Muscle Injury." *Biology of Sport* 29.1: 3-10. 2012.

64. Melzack, R and Wall, PD. Pain mechanisms: a new theory. Science 150: 971-979, 1965.

65. Morgan, DL and Allen, DG. Early events in stretch-induced muscle damage. J Appl Physiol 87: 2007-2015, 1999.

66. Proske, U and Morgan, DL. Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. J Physiol 537: 333-345, 2001.

67. Rehn, B, Lidstrom, J, Skoglund, J and Lindstrom, B. Effects on leg muscular performance from whole-body vibration exercise: a systematic review. Scand J Med Sci Sports 17: 2-11, 2007.

68. Rhea, MR, Bunker, D, Marin, PJ and Lunt, K. Effect of iTonic whole-body vibration on delayed-onset muscle soreness among untrained individuals. J Strength Cond Res 23: 1677-1682, 2009.

69. Rittweger, J, Beller, G and Felsenberg, D. Acute physiological effects of exhaustive whole-body vibration exercise in man. Clin Physiol 20: 134-142, 2000.

70. Rodenburg, JB, Bar, PR and De Boer, RW. Relations between muscle soreness and biochemical and functional outcomes of eccentric exercise. J Appl Physiol 74: 2976-2983, 1993.

71. Romaiguere, P, Vedel, JP and Pagni, S. Effects of tonic vibration reflex on motor unit recruitment in human wrist extensor muscles. Brain Res 602: 32-40, 1993.

72. Sargeant, AJ and Dolan, P. Human muscle function following prolonged eccentric exercise. Eur J Appl Physiol Occup Physiol 56: 704-711, 1987.

73. Sayers, SP, Knight, CA, Clarkson, PM, Van Wegen, EH and Kamen, G. Effect of ketoprofen on muscle function and sEMG activity after eccentric exercise. Med Sci Sports Exerc 33: 702-710, 2001.

74. Schutzer, KA and Graves, BS. Barriers and motivations to exercise in older adults. Prev Med 39: 1056-1061, 2004.

75. Scaglioni, G, Ferri, A, Minetti, AE, Martin, A, Van Hoecke, J, Capodaglio, P, Sartorio, A and Narici, MV. Plantar flexor activation capacity and H reflex in older adults: adaptations to strength training. J Appl Physiol 92: 2292-2302, 2002.

76. Stauber, WT. Eccentric action of muscles: physiology, injury, and adaptation. Exerc Sport Sci Rev 17: 157-185, 1989.

77. Tesch, PA, Dudley, GA, Duvoisin, MR, Hather, BM and Harris, RT. Force and EMG signal patterns during repeated bouts of concentric or eccentric muscle actions. Acta Physiol Scand 138: 263-271, 1990.

78. Tufano, JJ, Brown, LE, Coburn, JW, Tsang, KK, Cazas, VL and LaPorta, JW. Effect of aerobic recovery intensity on delayed-onset muscle soreness and strength. J Strength Cond Res 26: 2777-2782, 2012.

79. Tiidus, PM. Estrogen and gender effects on muscle damage, inflammation, and oxidative stress. Can J Appl Physiol 25: 274-287, 2000.

80. Twist, C and Eston, R. The effects of exercise-induced muscle damage on maximal intensity intermittent exercise performance. Eur J Appl Physiol 94: 652-658, 2005.

81. Warren, GL, Lowe, DA and Armstrong, RB. Measurement tools used in the study of eccentric contraction-induced injury. Sports Med 27: 43-59, 1999.

82. Warren, GL, Ingalls, CP, Lowe, DA and Armstrong, RB. Excitation-contraction uncoupling: major role in contraction-induced muscle injury. Exerc Sport Sci Rev 29: 82-87, 2001.

83. Weerakkody, NS, Percival, P, Hickey, MW, Morgan, DL, Gregory, JE, Canny, BJ and Proske, U. Effects of local pressure and vibration on muscle pain from eccentric exercise and hypertonic saline. Pain 105: 425-435, 2003.

84. Weerakkody, NS, Whitehead, NP, Canny, BJ, Gregory, JE and Proske, U. Large fiber mechanoreceptors contribute to muscle soreness after eccentric exercise. J Pain 2: 209-219, 2001.

85. Yackzan, L, Adams, C and Francis, KT. The effects of ice massage on delayed muscle soreness. Am J Sports Med 12: 159-165, 1984.

86. Young WB, Jenner A, Griffiths K. Acute enhancement of power performance from heavy load squats. J Strength Cond Res 12:82–84. 1998.

87. Yu, J. G., Carlsson, L., & Thornell, L. E. Evidence for myofibril remodeling as opposed to myofibril damage in human muscles with DOMS: an ultrastructural and immunoelectron microscopic study. Histochemistry and cell biology, 121(3), 219-227. 2004.

88. Zehr, E. P., & Stein, R. B. Interaction of the Jendrassik maneuver with segmental presynaptic inhibition. *Experimental brain research*, *124*(4), 474-480. 1999.