THE EFFECTS OF WHOLE BODY VIBRATION (WBV) AND LOCAL MUSCLE VIBRATION (LMV) ON PEAK TORQUE (PT) AND RATE OF TORQUE DEVELOPMENT (RTD)

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ABSTRACT

Dustin Lee: The Effects of Whole Body Vibration (WBV) and Local Muscle Vibration (LMV) on Peak Torque (PT) and Rate of Torque Development (RTD) (Under the direction of J. Troy Blackburn)

Purpose: Patients with anterior cruciate ligament (ACL) reconstruction have quadriceps dysfunction from arthrogenic muscle inhibition (AMI). AMI increases the risk of post-traumatic osteoarthritis, and current rehabilitation methods do not address AMI. Direct (local muscle vibration-LMV) and indirect (whole body vibration-WBV) vibratory stimuli improve muscle function and may be used for rehabilitation and performance enhancement. The purpose of this study was to compare the effects of WBV and LMV on quadriceps function.

Methods: Fifty-six healthy volunteers were randomized to one of three groups: WBV (n=19), LMV (n=19), or Control (n=18). All groups performed isometric squats while receiving their assigned intervention. Voluntary knee extensor peak torque (PT) and rate of torque development (RTD) were measured at baseline and again immediately, 10, and 20 minutes following intervention (WBV, LMV, control) during maximal isometric knee extension on an isokinetic dynamometer. Dependent variables were evaluated using 3 (group) by 4 (time) repeated measures ANOVA.

Results: The group by time interaction effect was significant for PT (p = 0.002), and post hoc analyses indicated that PT improved in the WBV group (p < 0.001) from baseline to immediately following intervention, but not in the LMV or control groups. No effects were observed in RTD (p = 0.123).
Conclusions: These findings suggest that WBV increases quadriceps PT in healthy individuals immediately following vibration. Therefore, WBV may improve traditional exercise and rehabilitation programs through neuromuscular enhancements. Future research is necessary to investigate the physiological mechanism associated with vibration therapy (VT) and determine the most effective delivery methods and stimulation parameters.
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CHAPTER 1: INTRODUCTION

The leading cause of physical disability in America is tibiofemoral osteoarthritis (OA), which affects approximately 29 million people and carries an annual cost of $165 billion (Dillon et al., 2006; Maetzel et al., 2004). Tibiofemoral OA is defined as a gradual reduction of articular cartilage within the knee joint (Lohmander et al., 2004). Interestingly, new evidence suggests that people who experience anterior cruciate ligament (ACL) and other knee joint injuries possess a greater risk for developing post-traumatic OA (Genuario et al., 2012; Lohmander et al., 2004; Mather et al., 2013; Neuman et al., 2008; Oiestad et al., 2011; E. M. Roos, 2005; von Porat, Roos, & Roos, 2004), which can occur as early as 5 years after the injury (Neuman et al., 2009; Suomalainen et al., 2012). Specifically, one out of three Americans 65 and older are affected by OA (Lawrence et al., 2008), which carries an annual cost of $5,700 per patient (Maetzel et al., 2004). Moreover, nearly 250,000 ACL injuries occur each year in the United States (Griffin et al., 2006), and these injuries are primarily suffered by younger adults (H. Roos et al., 1995). Above all, these individuals are 3-5 times more likely to develop knee OA than those with non-injured knees, and OA is seen in nearly 13% of people who have experienced an ACL injury, within ten years or less of follow-up (Oiestad et al., 2011). The high costs associated with OA and the increased probability of a younger population acquiring OA could lead to an exasperated healthcare burden, as these younger individuals live with OA for a substantial portion of their lives. Lastly, post-traumatic OA places many people at risk for
experiencing its long term effects, which include sedentary behavior and comorbidities such as cardiovascular disease (Philbin et al., 1996; Singh et al., 2002).

After individuals undergo an ACL reconstruction surgery, quadriceps weakness is commonly an accompanying disorder, which is caused by a complex neuromuscular phenomenon known as arthrogenic muscle inhibition (AMI). AMI refers to a form of neural inhibition which restricts the quadriceps’ ability to be voluntarily activated (Palmieri-Smith & Thomas, 2009a; Slemenda et al., 1997; Slemenda et al., 1998; Urbach et al., 2001). Once an individual suffers an ACL injury, mechanoreceptors from the injured joint send irregular afferent information to the central nervous system, which reduces excitability of the alpha motorneuron pool that controls the quadriceps (Palmieri-Smith & Thomas, 2009b). Pain, joint effusion, damage to joint mechanoreceptors, and abnormal joint translations may all be contributing factors to the changes in afferent input (Palmieri et al., 2004; Palmieri et al., 2005). People suffering from AMI have reported difficulties in completing many activities of daily living such as balance, sitting, stair climbing, and walking (Perry et al., 2007; Winters & Rudolph, 2014). Additionally, abnormal quadriceps activation patterns can lead to functional deficits at the knee joint that contribute to kinematic and kinetic changes during gait, which may lead to OA (M. D. Lewek, Rudolph, & Snyder-Mackler, 2004; Mündermann, Dyrby, & Andriacchi, 2005; Shelburne, Torry, & Pandy, 2006). During the early stance phase of gait, healthy quadriceps act as a shock absorber by dispersing the energy from the ground reaction force by eccentrically controlling knee flexion (Knoll, Kocsis, & Kiss, 2004). Conversely, quadriceps that are weakened by AMI are unable to adequately absorb the forces experienced during gait. Due to inefficient energy absorption, the unhindered forces are transferred to the knee’s articular cartilage, which could damage the joint through articular cartilage degeneration and lead to OA.
Interestingly, patients with ACL reconstruction have lesser internal knee extensor moments and knee flexion angles during gait, which increases the impulsive load on the knee by decreasing the time interval over which force is absorbed (M. Lewek et al., 2002).

Strength is defined as the maximum force generating capacity of a muscle, which is frequently calculated using measures of PT. Accordingly, PT has been shown to influence several movement characteristics in the elderly, such as their walking speed and ability to maintain balance during gait (Perry et al., 2007). Moreover, PT is commonly used as a tool to quantify athletic performance in elite athletes (Thompson et al., 2013). The importance of enhancing quadriceps strength is essential to better knee joint function and reduced joint destruction following knee injuries, but it needs to be delivered quickly to efficiently attenuate the ground reaction force at the knee (Winters & Rudolph, 2014). RTD provides an indication of how quickly an individual can develop maximal force. Increasing RTD after knee injuries may protect the joint and mitigate articular cartilage degeneration by actively absorbing ground reaction forces through eccentric control of the knee joint. Specifically, ground reaction forces are reduced at passive structures such as articular cartilage when quadriceps have the ability to absorb the associated energy by controlling knee flexion through rapid force production during the early stance phase of gait. Above all, quadriceps strength and RTD each contribute to absorbing potentially damaging forces experienced during gait, but they need to participate symbiotically at sufficient intensities to efficiently mitigate ground reaction energy before it influences degeneration of the knee’s articular cartilage. Therefore, increasing both quadriceps strength and RTD, in people suffering from neuromuscular dysfunctions following an ACL injury, could be an effective approach at delaying the initiation and progression of OA.
Current ACL rehabilitation programs that aim to increase quadriceps strength through exercise are usually ineffective at restoring knee extensor strength because they do not address the primary muscle activation issues associated with AMI (Hopkins & Ingersoll, 2000). For example, strengthening a muscle suffering from AMI is difficult since it cannot be effectively overloaded to stimulate hypertrophy without near maximal voluntary activation. Therefore, increasing the excitability of a muscle before exercise may be an effective mechanism for increasing muscle function (Hurley, Jones, & Newham, 1994). Consequently, new rehabilitation methods need to be implemented that treat the deficits associated with AMI. Vibration therapy (VT) is a progressing rehabilitation modality that increases muscle strength, power, PT and physical function (Abercromby et al., 2007b; Bosco et al., 1999; Bosco, Cardinale, & Tsarpela, 1999; Luo et al., 2009; Moran, McNamara, & Luo, 2007; Ronnestad, 2009; Samuelson, Jorfeldt, & Ahlborg, 1989). When VT is applied, the muscle experiences a series of rapid lengthening and shortening contractions which triggers a tonic vibratory reflex (TVR). This reflexive contraction increases the excitatory input to the alpha motorneuron pool which may override AMI (D. J. Cochrane et al., 2010; Eklund & Hagbarth, 1966; Rittweger, 2010). This neuromuscular enhancement caused by vibratory stimuli supports the possibility of improving the efficacy of traditional rehabilitation methods, designed to improve muscle function, by increasing the excitability of a muscle with VT before the initiation of strength training.

Currently, whole body vibration (WBV) is the main platform for administering VT, and has been demonstrated to enhance muscle function (Abercromby et al., 2007a; Abercromby et al., 2007b; D. J. Cochrane et al., 2010; De Ruiter et al., 2003; Pollock et al., 2010; Ronnestad, 2009; Salmon, Roper, & Tillman, 2012; Segal et al., 2013; Tihanyi et al., 2007). However, WBV presents many barriers to clinical use such as limited portability and a high cost (up to $12,000).
Conversely, LMV also improves muscle function (Bosco et al., 1999; Moran et al., 2007), and it may counter the limitations of WBV. Specifically, LMV is easily portable and costs substantially less than WBV (~$250), which suggests that it could be a cost-effective alternative to WBV. LMV provides a similar stimulus as WBV, but its effects may differ, as they are administered differently. LMV may deliver a more efficient vibration stimulus as it is applied directly over the muscle being stimulated. Conversely, the stimulus generated by WBV is damped as it passes through the lower extremity to the knee. For example, the transmissibility of WBV is dependent on the stance of the individual receiving the stimulus (i.e. standing erect vs. knees bent), as differences in muscle activation attenuate the stimulus differently (Rubin et al., 2003). Conversely, WBV may provide additional benefits by stimulating multiple sensory receptors in other areas of the lower extremity, such as cutaneous receptors in the feet and mechanoreceptors in other joints. WBV and LMV produce comparable increases in muscle activation and strength in individuals following experimental knee joint effusion (Blackburn et al., 2014), but it remains unclear how long these effects last, which would be necessary information if VT were to be used as a rehabilitation modality. Furthermore, no studies have compared the effects of WBV and LMV on quadriceps strength and RTD in healthy individuals without knee pathologies or determined how long the effects last.

Patients with ACL injuries are hindered by quadriceps dysfunction from AMI, and current rehabilitation methods are inadequate for addressing these functional deficits. WBV improves muscle function, but has multiple limitations which may restrict its clinical application. LMV alleviates many of these limitations and may be a cost-effective, equivalent treatment. However, there are few studies comparing the effectiveness of WBV and LMV. If LMV produces increases in quadriceps muscle function that are equal to or greater than those produced
by WBV, more people suffering from quadriceps weakness associated with AMI could receive
treatment that may delay the initiation and progression of OA due to LMV being a portable, low-
cost alternative to WBV. Therefore, the purpose of this study was to compare the effects of
WBV and LMV on measures of quadriceps function and determine their duration. The specific
aims were as follows:

1) To determine the effects of WBV and LMV on quadriceps strength and RTD during a
maximal isometric contraction in healthy adults. *I hypothesized that WBV and LMV
would increase quadriceps strength and RTD, and that these changes would be
greater than those observed in a control group receiving no treatment.*

2) To compare the effects of WBV and LMV on quadriceps strength and RTD during a
maximal isometric contraction in healthy adults. *I hypothesized that WBV and LMV
would increase quadriceps strength and RTD with similar magnitudes.*

3) To determine the duration of the effect of WBV and LMV on quadriceps strength and
RTD during a maximal isometric contraction in healthy adults. *I hypothesized that
the effect of WBV and LMV on quadriceps strength and RTD would last at least 10
minutes subsequent to the treatment.*
CHAPTER 2: LITERATURE REVIEW

INTRODUCTION

The purpose of this literature review was to analyze relevant studies and identify their areas of limited understanding. Primarily, the muscular function deficits that people experience after suffering an ACL injury were addressed (i.e. quadriceps weakness). Additionally, the muscular shortfalls associated with post traumatic knee injury were highlighted to explain a potential pathway from ACL injury to OA through characteristic alterations in gait kinematics and kinetics. Moreover, the relationship between neuromuscular deficiencies and the failure to complete activities of daily living were identified. This review also explained the insufficiencies regarding current rehabilitation methods and recognized VT as a potential approach to increase muscular function after ACL injury. Lastly, the efficacies of LMV and WBV were compared to determine if LMV could be implemented as a possible cost-effective and portable alternative to WBV.

ACL INJURY EPIDEMIOLOGY

Nearly 250,000 ACL injuries occur each year in the United States, and their annual associated costs are $7.6 billion when treated with ACL reconstruction and $17.7 billion when treated with rehabilitation (Griffin et al., 2006; Mather et al., 2013). Changes in kinematics during gait and altered quadriceps loading patterns are often present after an ACL injury, which
can increase the odds of acquiring tibiofemoral OA (Lohmander et al., 2004; Neuman et al., 2009; E. M. Roos, 2005; von Porat et al., 2004). Tibiofemoral OA is defined as a gradual breakdown of articular cartilage within a joint (Oiestad et al., 2011). Interestingly, patients with an ACL injury are 3-5 more times likely to develop OA than people with non-injured knees (Lohmander et al., 2004; Neuman et al., 2009), and OA development can occur as early as 5 years after an ACL injury (Ajuied et al., 2014). In fact, within ten years or less, OA is present in nearly 13% of all knees with no associated meniscal injury, and up to 48% of knees with an associated meniscal injury (Oiestad et al., 2011). Moreover, ACL injuries are most commonly suffered by younger adults, and the annual costs associated with OA are approximately $5,700 per patient (Maetzel et al., 2004). The high expenses associated with OA and the increased probability of a younger population acquiring OA contributes to an increased healthcare burden, as these younger individuals live with the costly effects of OA for a substantial portion of their lives.

NEUROMUSCULAR ALTERATIONS FOLLOWING INJURY

After an ACL injury, quadriceps weakness is often an accompanying disorder that is commonly considered a result of disuse atrophy (Palmieri-Smith & Thomas, 2009a). This may be one factor involved in the development of quadriceps weakness, but this hypothesis remains questionable because near instantaneous reductions in strength have been demonstrated post-injury and quadriceps weakness often persists after arduous rehabilitation programs (Hopkins & Ingersoll, 2000; Hurley et al., 1994). AMI, the failure to completely voluntarily activate a muscle, is often an alternative explanation for reductions in strength that patients experience after an ACL injury (Palmieri-Smith & Thomas, 2009a). AMI is a form of a neural inhibition that reduces efferent motor input to a muscle, which limits the activation of a portion of the motor
units in muscles surrounding an injured joint (Palmieri-Smith, Thomas, & Wojtys, 2008). This neural response is thought to be a protective mechanism initiated to prevent pain and unnecessary motion in the injured joint (Palmieri-Smith & Thomas, 2009a). AMI is nearly a ubiquitous disorder, and it is commonly measured using central activation ratio (CAR), which estimates the percentage of motor units that can be contracted voluntarily. Various studies using CAR to measure quadriceps activation after an ACL injury have reported deficits in nearly all patients with magnitudes ranging from 8% to 45% (Hart et al., 2010). Additionally, these insufficiencies often persist several years after the injury and extensive rehabilitation, potentially leading to joint degeneration. (Hopkins & Ingersoll, 2000). Interestingly, a current meta-analysis conducted by Pietrosimone et al. (2011) emphasized that quadriceps muscle activation deficits exist in persons with tibiofemoral OA at comparable rates with magnitudes of approximately 20%. Similar quadriceps muscle activation patterns between people with ACL injuries and individuals with OA supports the hypothesis that quadriceps weakness could be a precursor to OA (Palmieri-Smith & Thomas, 2009a).

The ACL houses mechanoreceptors which are responsible for sending afferent sensory signals to the central nervous system (CNS) to provide proprioception information regarding joint translations, position, and loading (Andriacchi & Mündermann, 2006). Mechanoreceptors also initiate defensive reflex mechanisms that help protect and stabilize an injured joint (Palmieri-Smith et al., 2008). Once an individual suffers an ACL injury, mechanoreceptors from the damaged joint send irregular afferent information to the central nervous system, which reduces excitability of the alpha motorneuron pool that controls the quadriceps. Pain, joint effusion, damage to joint mechanoreceptors, and abnormal joint translations may all be
contributing factors to the changes in afferent input (Palmieri-Smith & Thomas, 2009a; Rice & McNair, 2010).

**POST-TRAUMATIC OA**

OA refers to the gradual breakdown of articular cartilage within a joint, and is traditionally diagnosed via its symptoms and physical examination. However, its severity and progression are commonly assessed with the Kelgren-Lawrence scale, which uses radiographic data to determine the quantity of osteophytes and the magnitude of joint space narrowing within a patient’s knee (grade 1 – doubtful narrowing of joint space and possible osteophytic lipping, grade 2 – definite osteophytes and definite narrowing of joint space, grade 3 – moderate multiple osteophytes and definite narrowing of joint space, grade 4 – large osteophytes and marked narrowing of joint space) (Ajuied et al., 2014; Oiestad et al., 2011). OA is the primary cause of physical disability in America, affecting nearly 33% of adults older than 65 (Lawrence et al., 2008), and knee OA is the most prevalent form of OA, which affects nearly 29 million people (Dillon et al., 2006), and carries an annual cost of $165 billion (Maetzel et al., 2004). Knee OA is classified as idiopathic when it’s a result of a non-specific cause, but when it occurs after a knee injury, such as a ruptured ACL, it’s categorized as post-traumatic. After patients suffer an ACL injury, their probably of developing knee OA increases by 3-5 times (Oiestad et al., 2011; E. M. Roos, 2005). The long term effects of knee OA are compounded by its tendency to encourage sedentary behavior and its influence in the development of comorbidities such as cardiovascular disease (Singh et al., 2002). Therefore, the progressive nature of OA and the expenses associated with its long term effects demonstrate the need for cost saving rehabilitation methods that delay its initiation and progression.
After an individual suffers an ACL injury, quadriceps weakness is usually an accompanying complication which can be partly associated with AMI, and is present in nearly all patients who are ACL deficient and approximately 80% of patients who have undergone ACL reconstruction (Hart et al., 2010). Healthy quadriceps help maintain normal knee ambulation and protect the knee joint by attenuating and evenly distributing the ground reaction forces imposed during gait (Andriacchi & Mündermann, 2006; Palmieri-Smith et al., 2008; Urbach et al., 2001). Deficits in quadriceps strength can lead to joint laxity and biomechanical alterations, which can influence articular cartilage breakdown by increasing the magnitude of force delivered to the knee and increasing the rate at which it is delivered (Andriacchi & Mündermann, 2006; Palmieri-Smith & Thomas, 2009a).

Articular cartilage is a smooth, lubricating, connective tissue that, when under normal conditions, can efficiently attenuate the cyclic loading patterns experienced in joint articulations (Radin et al., 1984). Additionally, articular cartilage is viscoelastic, which means it can momentarily deform when subjected to a stress and return to its natural state once the stress is removed (Andriacchi & Dyrby, 2005; Chaudhari et al., 2008; Radin et al., 1984). For example, articular cartilage’s viscoelastic properties allow it to efficiently attenuate forces as long as it is afforded adequate time to recover and regain its original state between the applications of forces. Therefore, articular cartilage’s force absorbing abilities are time dependent, and it is capable of absorbing high cyclic loading without damage. However, if forces are delivered too rapidly the associated cartilage can stiffen and begin to degrade, which decreases its ability to efficiently attenuate forces. Interestingly, articular cartilage has poor healing capabilities because it does not receive blood supply or innervation (Andriacchi & Dyrby, 2005). Therefore, biomechanical
alterations caused by changes in knee joint loading patterns that people experience after an ACL injury induce an irreversible form of articular cartilage breakdown and the initiation of OA.

When the quadriceps loses its ability to be fully activated, its force generating capability declines, which can alter its response to the external demands experienced during gait (Palmieri-Smith & Thomas, 2009a; Slemenda et al., 1997). Changes in force production and altered joint loads at the knee can induce kinematic and kinetic transformations, which can lead to articular cartilage breakdown and the initiation and progression of OA. For example, during the early stance phase of gait, the quadriceps act as a shock absorber by absorbing and dispersing the energy from the ground reaction force by eccentrically controlling knee flexion (Knoll et al., 2004; M. Lewek et al., 2002; M. D. Lewek et al., 2004; M. D. Lewek, Rudolph, & Snyder-Mackler, 2004). Concerning this, Jefferson et al. (1990) studied the role of quadriceps in controlling impulsive forces during gait, which compared knee flexion angles and ground reaction forces before and after individuals experienced artificially induced quadriceps paralysis. After quadriceps paralysis, decreased knee flexion angles and impulsive loading characteristics were reported. Prior to the heel strike phase of gait, healthy quadriceps smoothly decelerate the lower limb and reduce the ground reaction force by softening the impact of the foot with the ground. Conversely, weak quadriceps interrupts this mechanism, which can interfere with the lower limb’s energy absorption capabilities. Interestingly, people with ACL injuries have reduced internal knee extensor moments and knee flexion angles during gait, which increases the impulsive load characteristics on the knee by decreasing the time interval through which force is absorbed (Glass et al., 2013; Winters & Rudolph, 2014).

Loading characteristics and articular cartilage thickness at the knee joint are regionally dependent, and the thickest areas of articular cartilage are found in regions that are chronically
exposed to high stress (Andriacchi & Mündermann, 2006). This suggests that joints have adaption capabilities that mitigate stress on areas that experience chronic loading by increasing the amount of cartilage at specific locations (Andriacchi & Mündermann, 2006). However, ACL injury can cause knee joint laxity and changes in gait kinematics that alter tibiofemoral contact areas, potentially introducing areas of articular cartilage to forces to which they are unaccustomed (Hopkins & Ingersoll, 2000; Palmieri-Smith et al., 2008). For example, after an ACL injury, joint surfaces that are composed of relatively thin areas of articular cartilage, not conditioned to frequent stress, and less efficient at absorbing forces may be loaded to a greater extent. Articular cartilage breakdown is initiated when thinner regions of cartilage that are less accustomed to frequent stress experience more frequent loading of greater magnitude before they have time to adapt. Therefore, changes in knee joint contact areas and increases in pressure on unaccustomed areas of articular cartilage may initiate OA by narrowing joint space and damaging articular cartilage.

**DEFICITS IN PT AND RTD FOLLOWING INJURY**

Decreased quadriceps strength is a common and persistent deficiency associated with ACL injury, and the inability to activate a muscle with sufficient force at an adequate rate in response to external demands carries many biomechanical risks and decreases an individual’s capacity to complete many activities of daily living (Hurley et al., 1994; Palmieri-Smith & Thomas, 2009a; Winters & Rudolph, 2014). PT is defined as the maximum force generating capacity of a muscle, and it is commonly used as a tool to quantify athletic performance in elite athletes (Thompson et al., 2013). PT has also been shown to influence several movement characteristics in the elderly, such as their walking speed and ability to maintain balance during gait (Perry et al., 2007). Additionally, unimpaired quadriceps have been connected to later onset
and slowed progression of OA (Palmieri-Smith et al., 2008). Therefore, quadriceps that are capable of producing forces needed to complete functional tasks and protect the knee from external forces are more proficient at maintaining a healthy joint than quadriceps that are hindered by decreased strength. However, Sharma et al. (2000) reported that greater quadriceps PT was associated with OA progression in patients who have been diagnosed with OA and have lax knees. This finding suggests that other indices of muscle function may be involved in controlling biomechanical alterations and the progression of OA. Nearly all individuals with knee OA have altered quadriceps activation patterns associated with AMI (Hart et al., 2010), and the failure to adapt to knee joint laxity may be an indication of a decrease in the rate of force delivery by the quadriceps, caused by a deficiency in motor neuron recruitment (i.e. AMI). RTD provides an indication of how quickly an individual can develop maximal force (Winters & Rudolph, 2014), and rapid RTD has been shown to compensate for decreased quadriceps strength by delivering counter forces quickly in response to external demands. Interestingly, Staehli et al. (2010) demonstrated that quadriceps RTD is more closely related to activities of daily living that quadriceps PT. Therefore, the functional and biomechanical associations to RTD illustrate its importance to knee joint function. Lastly, the kinetic and kinematic alterations caused by quadriceps weakness that have been demonstrated to initiate joint degeneration could be mitigated by rehabilitation programs aimed at increasing both PT and RTD.

VT

Traditional rehabilitation programs that are designed to increase quadriceps strength in individuals with pathologic knees through exercise are commonly ineffective at restoring muscle function because they do not address the primary muscle activation issues associated with neural inhibition caused by AMI (Hopkins & Ingersoll, 2000; Hurley et al., 1994). Specifically,
rehabilitation professionals, focused on improving strength, commonly prescribe exercises with the fundamental purpose of inducing muscular hypertrophy (Hellebrandt, 1958). Muscular hypertrophy is a multidimensional process that is defined as an increase in muscle mass and cross-sectional area, which can be accomplished via the overload principle (Robergs & Roberts, 1997; Russell, Motlagh, & Ashley, 2000). In brief, the overload principle is a muscular adaptation that occurs in response to exercise stimuli that force muscle fibers to experience tensile forces near their maximum capacity (Hellebrandt, 1958). Subsequently, the accompanying musculature is forced adapt through hypertrophy, leading to an increase in the size of the individual muscle fibers and their consequent force production capabilities (Jones & Lees, 2003; Pearson et al., 2000; Russell et al., 2000). However, individuals suffering from neuromuscular deficits may not have the capacity to effectively overload all their muscle fibers and experience muscular hypertrophy and its associated strength gains, as activation scarcities limit muscle’s ability to efficiently respond to training stimuli (Hopkins & Ingersoll, 2000).

Specifically, if a motor unit is not activated, its associated muscle fibers cannot be overloaded, and no adaptation can occur without overload (McNicol et al., 2009; Russell et al., 2000). Furthermore, neuromuscular deficiencies not only restrict individuals’ ability to acquire strength gains, it places them at risk to experience muscle atrophy and its debilitating effects (Hurley et al., 1994).

Consequently, innovative rehabilitation methods are needed that treat the strength deficits associated with AMI. VT is an emerging rehabilitation modality that has been demonstrated to enhance muscle function as measured by strength, power, and electromyography (D. J. Cochrane et al., 2010; D. Cochrane, 2011). Aside from the multiple reports of enhanced muscle function, VT has also displayed equivocal and detrimental effects (De Ruiter et al., 2003). However, VT’s
potential neural enhancement capabilities may validate it as a possible rehabilitation modality that could be used in combination with traditional strength training programs aimed at increasing muscle function.

Vibration provides a mechanical oscillation of force, acceleration, and displacement over time. VT is defined as a forced oscillation during which energy is transferred from an actuator (vibration device) to a resonator (human body) (Bazett-Jones, Finch, & Dugan, 2008). The transfer of energy causes reactive forces within the body, which can cause beneficial neuromuscular responses, but it also carries the potential to harm tissues within the body. VT inflicts a force on the body that is proportional to its mass, and it causes the body to accelerate through sinusoidal oscillations in which the affected muscles and tendons act like springs by storing and releasing mechanical energy (Rittweger, 2010). An accumulation of mechanical energy within the body can damage muscles through increases in internal forces when the frequency of the actuator matches the natural frequency of the resonator, which can be controlled by the body’s stiffness and mass (D. J. Cochrane et al., 2010; D. J. Cochrane, 2011). Therefore, this damaging resonance effect can be overcome by muscles damping the signal through modifications in body position and changes in muscle stiffness. For example, the transmissibility of ground based vibrations is dependent on posture and the associated contractions of accompanying muscles. Rubin et al. (2003) compared the transmissibility of low level frequencies using ground-based vibrations between three positions: (standing erect, relaxed, and knees bent) and found the signal transmissibility varied greatly between different postures. Specifically, the knees bent position revealed that more than 50% of the stimuli were absorbed by the lower extremity, and that the transmissibility of a signal decreases as the ankle, knee and hip joint angles decrease.
When VT is applied, the energy transfer causes the muscle to go through a series of rapid lengthening and shortening contractions which triggers a tonic vibratory reflex (TVR) (Burke et al., 1976; Eklund & Hagbarth, 1966). The vibration causes muscles and their associated muscle spindles to lengthen and they become more easily excitable. The reflexive contraction experienced by the muscles increases the excitatory input to the alpha motorneuron pool which can increase a muscle’s force generating capability by increasing the activation of its motor units (Rittweger, 2010). There are also additional mechanisms that can explain the neuromuscular increases associated with VT such as muscle tuning, motor unit synchronization, central motor command, and intramuscular coordination (D. J. Cochrane et al., 2010; D. Cochrane, 2011). Additionally, muscle enhancements can be caused by escalations in muscle temperature and blood flow, similar to a warm-up. Lastly, VT also inflicts a training-effect, which is caused by a muscle’s contractual response to increases in gravitational forces caused by vibration (D. J. Cochrane, 2011; D. Cochrane, 2011).

Effects on Muscle Function

The studies showing increases in power and muscle strength following vibration suggest that VT could be used in rehabilitation programs aimed at treating muscular deficits. For instance, Bosco et al. (1999) revealed enhanced leg press power of nearly 10% in young elite volleyball players succeeding VT. Similar improvements have been reported for elbow flexion power in boxers (Bosco et al., 1999; Roelants, Delecluse, & Verschueren, 2004) and vertical jump height in elite hockey players (Issurin & Tenenbaum, 1999; Rittweger, 2010). Moreover, VT has also been shown to increase one repetition maximum in healthy populations (K. Mileva et al., 2006) and muscle strength in patients following stroke (Tihanyi et al., 2007). Additional studies have also reported improvements in balance and proprioception, which implies the
various enhancements in muscle function associated with VT may be a result of neuromuscular improvements (D. Cochrane, 2011). Regardless of these positive findings, there are also studies that show ambivalent results in one repetition maximum (Lau, Yip, & Pang, 2012; Pamukoff, Ryan, & Blackburn, 2014; Segal et al., 2013), and RTD (Pamukoff et al., 2014). Additionally, others have demonstrated negative effects on muscle strength (De Ruiter et al., 2003; Erskine et al., 2007a; Herda et al., 2009). For example, Erskine et al. (2007a) found a 9% decrease in knee extensor strength following 10 by 1 minute isometric half squat exercises while being exposed to WBV. Interestingly, the aforementioned VT studies were administered via different application methods and with dissimilar treatment parameters (i.e. duration, amplitude and frequency). Therefore, the lack of treatment guidelines existing for VT may be an explanation for the inconsistencies between these findings and the basis for the heterogeneous stimulation parameters employed (D. J. Cochrane et al., 2010). The method by which VT is received may also change its potential benefits because changes in posture and muscle stiffness alter the transmissibility of vibration (Rubin et al., 2003). For example, Pamukoff et al. (2014) compared the effects of LMV on PT at 30 Hz and at 60 Hz and showed increases in PT at 30 Hz but no effect at 60 Hz, which enforces the idea that a muscle’s mechanical response varies with frequency.

**WBV vs. LMV**

The majority of VT studies have focused on the muscular improvements associated with WBV. However, WBV platforms have limited portability and they carry cost restrictions (~$12,000) (Blackburn et al., 2014). LMV also improves muscle function (Blackburn et al., 2014; Erskine et al., 2007a; Luo et al., 2009; Pamukoff et al., 2014) and may be a more accessible alternative to WBV due to its portability and cost effectiveness (~$250). Concerning
the efficacy of LMV, which is applied directly to the muscle-tendon unit, Iodice et al. (2011) revealed that leg extensor muscle strength increased following acute exposure to LMV. However, other investigators have reported equivocal outcomes after LMV (Moran et al., 2007).

Although WBV and LMV produce similar stimuli, the efficacy of the neuromuscular enhancements that they provide may be altered by their modes of application due to different muscle damping characteristics.

By changing the delivery method of the VT (i.e. standing on platform or local application), the body’s musculature absorbs the associated energy differently, which may change the neuromuscular response (Abercromby et al., 2007a). Additionally, the characteristics of vibration stimuli are dependent on the physical properties of the material through which they travel. Specifically, changes in posture alter the frequency received by muscles through inherent alterations in muscle stiffness associated with variations in stance (Abercromby et al., 2007b; Rubin et al., 2003). Consequently, deviations in vibration frequency can affect the magnitude of a muscle’s TVR, which is directly associated with the extent of excitation increases in the alpha motorneuron pool that control the quadriceps (Rittweger, 2010).

During WBV, the stimulus that is received at the quadriceps is first dampened by the calf muscles and the musculature adjacent to the knee and ankle joints, which may decrease the efficacy of its neurological response through decreases in energy absorption. Conversely, the quadriceps may receive a more effective stimulus if it is applied directly to the muscle via LMV rather than WBV due to less signal absorption by the lower extremity. However, only one study (Blackburn et al., 2014) has compared the effects of WBV and LMV on muscle function, which showed comparable effects on PT and voluntary quadriceps activation in a group of healthy subjects with artificial knee effusion. Nevertheless, the study had limitations, such that the
increases in muscle function may have been a result of the vibration treatment influencing the diffusion of saline from the artificially effused knees. However, no studies have compared the effects of WBV and LMV on quadriceps strength and RTD in healthy individuals without knee pathologies or determined how long the effects last.
CHAPTER 3: EXPERIMENTAL DESIGN AND METHODS

SUBJECTS

Sixty healthy individuals (30 males and 30 females) were recruited from the student and employee populations at the University of North Carolina at Chapel Hill and from the surrounding area. Subjects were included if they were between the ages of 18 and 30 years and recreationally active, defined as participation in physical activity for at least 30 minutes 3 times per week. Subjects were excluded for any history of musculoskeletal injury within 6 months prior to testing, lower extremity surgery, neurological disorder, cardiovascular disease, hypertension, diabetes mellitus, concussion or head injury, stroke, epilepsy, peripheral neuropathy, migraine headaches, cranial neural surgery, cancer in the brain or thigh musculature, cardiac pacemaker, implanted foreign metal object, or diagnosed psychiatric disorder. Inclusion and exclusion criteria were confirmed via self-report. Each subject was required to read and sign an informed consent form prior to data collection.

EXPERIMENTAL DESIGN

This project was part of an ongoing study that evaluated the effects of vibratory stimuli on several measures of neuromuscular function (corticospinal excitability, Hoffman’s reflex, and voluntary quadriceps activation). Each respective measurement was tested on a separate day determined in a random order, and PT and RTD was measured during the assessment of voluntary quadriceps activation.
This investigation utilized a randomized controlled trial experimental design. Subjects were randomized to 1 of 3 groups following pre-test assessments and received either WBV, LMV, or Control (no vibration) treatment (n = 20 per group). Although subjects completed 3 testing visits as part of the larger ongoing study, data for this study was collected from a single visit to the Neuromuscular Research Laboratory at the University of North Carolina at Chapel Hill which lasted approximately 1 hour. Each subject completed a baseline evaluation of knee extensor PT and RTD during a 5-second maximal knee extension, received one of the aforementioned interventions, and completed follow-up testing immediately, 10 minutes, and 20 minutes following the intervention.

ASSESSMENTS

PT and RTD

Subjects first underwent a brief 5-minute aerobic warm-up on a stationary cycle ergometer followed by baseline tests of isometric knee extensor PT and RTD using a dynamometer (Humac Norm, Stoughton MA). This test was performed on the dominant limb, defined as the limb that would be used to kick a ball. The thighs, hips, and upper body were firmly stabilized with straps. The lever arm of the dynamometer was adjusted so that the ankle strap was placed 2 finger widths above the medial malleolus. The knee was positioned so that the lateral femoral epicondyle was aligned with the rotational axis of the dynamometer. The knee was flexed 60° (Figure 1, Left), and torque data was sampled at 2kHz for 5 seconds using the Biopac data acquisition system (MP150WSW, Biopac Systems Inc., Santa Barbara, CA). Three trials were completed at each respective time point (pre-intervention, post intervention, 10-min post intervention, and 20-min post intervention). After the subjects received a visual stimulus to
initiate knee extension, they were instructed to “kick as hard and as fast as possible” and received verbal encouragement for each trial to ensure maximal effort. Sixty seconds of rest was given between assessments.

**Intervention Procedures**

Following baseline testing, subjects were randomized to LMV, WBV, or Control (no vibration) groups. The LMV group received 6 exposures of 60 seconds of vibration with 2 minutes of rest between each exposure while standing in approximately 40° of knee flexion. A custom-made LMV device was positioned on the quadriceps tendon (Figure 1, Center). Subjects randomized to the WBV group stood on a vibrating platform that delivered a similar stimulus (Figure 1, Right). The LMV and WBV stimuli were delivered at 2g of acceleration at a frequency of 30Hz. The control group did not receive vibration, but assumed the same knee flexion position in 6x60 second intervals with 2 minutes of rest between exposures. All subjects stood on the WBV device during their respected interventions, but it was only activated for the subjects assigned to the WBV group. These parameters were similar to a previous study in our laboratory that represented comparable effects between LMV and WBV on voluntary muscle activation (Blackburn et al., 2014). Directly after the intervention, subjects repeated the aforementioned assessments of PT and RTD during a maximal isometric contraction, and again 10 minutes and 20 minutes following the cessation of the intervention.

**Data Reduction**

Torque data was low-pass filtered at 50Hz (4th order Butterworth), and PT and RTD was calculated from the resulting torque vs. time curve. PT was defined as the maximal voluntary torque value normalized to body mass. Change in torque (final-initial) was calculated over
successive 20ms intervals and divided by time to represent RTD. The peak RTD value was identified and normalized to body mass for statistical analysis. The mean PT and RTD across trials was calculated at each time point and used for all subsequent analyses.

Data Analysis

Data was inspected for normality using the Shapiro-Wilk test and homogeneity of variance using Levene’s test to confirm the assumptions for analysis of variance (ANOVA). All data was screened for outliers using boxplots. Three (group) by four (time) repeated measures ANOVA was used to examine the difference between groups (WBV, LMV, control) from pretest to immediately post test, 10 minutes post test, and 20 minutes post test for PT and RTD. All analyses were conducted with an a priori alpha level of 0.05. Bonferroni post hoc procedures were used to evaluate pairwise comparisons within groups (baseline vs. each respective time point) and between groups (at each testing time point) where a significant F-statistic was found ($\alpha = 0.05/18 = 0.003$).
Figure 1: Left - Testing Position, Middle - LMV, Right - WBV
CHAPTER 4: RESULTS

Box plots were utilized to identify outliers, defined as values more than 1.5 times the interquartile range. Four subjects’ data were identified as outliers and excluded from statistical analysis for subsequent evaluation. Demographic data for subjects retained in the final analyses are detailed in Table 1.

PT

PT did not differ between groups at baseline (p = 0.675; Table 2). Additionally, the PT data violated the assumption of sphericity. As such, the Greenhouse-Geisser correction was used to assess the group by time interaction. The group by time interaction was significant (F2,53 = 4.26, p = 0.002). Post hoc testing (Table 3) revealed a significant increase for the WBV group (+0.29 Nm/kg, 95%CI: 0.18 – 0.42, p < 0.001) from baseline to immediately following treatment. However, due to overly conservative post hoc analyses, there was not a significant change in the WBV group at 10 or 20 minutes following intervention. However, there was a statistical trend that approached significance 10 minutes (+0.13 Nm/kg, 95%CI: 0.05 – 0.21, p = 0.007) and 20 minutes (+0.08 Nm/kg, 95%CI: 0.01 - 0.15, p = 0.028) after WBV. There were no significant differences in either the LMV or control groups anytime following treatment. Similarly, though significant improvements were observed with WBV, PT did not differ between groups at any time point. The effects of the vibratory interventions on PT are detailed in Figure 2 and Table 4.
RTD

RTD did not differ between groups at baseline (p = 0.631; Table 2). The group by time
interaction was not significant for RTD (F_{2,53} = 1.71, p = 0.123). The effects of the vibratory
interventions on RTD are detailed in Figure 3 and Table 4.
<table>
<thead>
<tr>
<th>Demographic Data</th>
<th>Control (n=18; 8 men, 10 women)</th>
<th>WBV (n=19; 10 men, 9 women)</th>
<th>LMV (n=19; 9 men, 10 women)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>20.5 ± 1.2</td>
<td>20.2 ± 0.9</td>
<td>19.5 ± 1.4</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>172.0 ± 10.7</td>
<td>167.3 ± 8.9</td>
<td>171.1 ± 8.8</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>69.4 ± 12.9</td>
<td>66.4 ± 10.5</td>
<td>65.5 ± 10.7</td>
</tr>
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</table>
Table 2. One way ANOVA of baseline values (mean ± SD)

<table>
<thead>
<tr>
<th>Variable</th>
<th>WBV (n=19)</th>
<th>LMV (n=19)</th>
<th>Control (n=18)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTnorm (N m/kg)</td>
<td>2.55 ± 0.55</td>
<td>2.58 ± 0.58</td>
<td>2.62 ± 0.62</td>
<td>0.675</td>
</tr>
<tr>
<td>RTDnorm (Nm/s/kg)</td>
<td>24.0 ± 9.2</td>
<td>21.9 ± 10.7</td>
<td>21.1 ± 7.6</td>
<td>0.631</td>
</tr>
<tr>
<td>Comparison</td>
<td>Mean Difference (%)</td>
<td>95% confidence interval</td>
<td>Sig. (1-tailed)</td>
<td></td>
</tr>
<tr>
<td>-----------------------------</td>
<td>---------------------</td>
<td>-------------------------</td>
<td>-----------------</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lower</td>
<td>Upper</td>
<td></td>
</tr>
<tr>
<td>Baseline vs. Post *</td>
<td>0.299</td>
<td>0.18</td>
<td>0.42</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Baseline vs. 10 min Post †</td>
<td>0.128</td>
<td>0.05</td>
<td>0.21</td>
<td>.0065</td>
</tr>
<tr>
<td>Baseline vs. 20 min Post †</td>
<td>0.081</td>
<td>0.01</td>
<td>0.15</td>
<td>.028</td>
</tr>
</tbody>
</table>

* Indicates Significant Differences
† Indicates Statistical Trend
<table>
<thead>
<tr>
<th>Intervention</th>
<th>Baseline</th>
<th>Intervention</th>
<th>Intervention</th>
<th>Intervention</th>
<th>group x time interaction, p:</th>
</tr>
</thead>
<tbody>
<tr>
<td>PT (Nm·kg⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.002</td>
</tr>
<tr>
<td>WBV</td>
<td>2.55 ± 0.56</td>
<td>2.85* ± 0.47</td>
<td>2.68‡ ± 0.51</td>
<td>2.63‡ ± 0.53</td>
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</tr>
<tr>
<td>LMV</td>
<td>2.58 ± 0.58</td>
<td>2.46 ± 0.60</td>
<td>2.45 ± 0.57</td>
<td>2.42 ± 0.52</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>2.62 ± 0.62</td>
<td>2.62 ± 0.66</td>
<td>2.59 ± 0.54</td>
<td>2.56 ± 0.51</td>
<td></td>
</tr>
<tr>
<td>RTD (Nm·sec⁻¹·kg⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.123</td>
</tr>
<tr>
<td>WBV</td>
<td>24.02 ± 9.17</td>
<td>25.95 ± 10.85</td>
<td>23.75 ± 8.60</td>
<td>24.67 ± 7.35</td>
<td></td>
</tr>
<tr>
<td>LMV</td>
<td>21.90 ± 10.67</td>
<td>21.45 ± 11.46</td>
<td>22.31 ± 12.38</td>
<td>23.03 ± 12.98</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>21.08 ± 7.58</td>
<td>22.16 ± 7.92</td>
<td>24.07 ± 8.44</td>
<td>22.49 ± 7.77</td>
<td></td>
</tr>
</tbody>
</table>

* Significantly different versus baseline.

‡ Statistical trend versus baseline (P < .03).
Figure 2. Normalized PT.
Figure 3. Normalized RTD.
CHAPTER 5: DISCUSSION

The primary findings of this study were that WBV significantly increased quadriceps PT in healthy individuals immediately following treatment. Additionally, our results suggested that quadriceps PT remained elevated 10 and 20 minutes after WBV relative to baseline values. Though not statistically significant due to overly conservative post hoc analyses these outcomes displayed a statistical trend that approached significance. Moreover, there were no substantial improvements in PT following LMV at any time point. With respect to RTD, there were no significant differences in WBV or LMV anytime following intervention.

These findings are consistent with previous studies with regards to improvements in quadriceps strength in healthy individuals following WBV (Jacobs & Burns, 2009). For example, Mileva et al. (2006) reported a 10% increase in quadriceps PT during maximum voluntary isometric contraction exercises in healthy adults following WBV. Vibratory stimuli enhance muscle function through the excitation of alpha motorneurons by repeatedly stimulating primary muscle spindle endings (type Ia afferent neuron), resulting in reflexive contributions to muscle force known as the tonic vibratory reflex (Burke et al., 1976; Eklund & Hagbarth, 1966). However, the improvements in muscle function caused by the tonic vibratory reflex would likely only account for amplified muscle function during exposure to vibratory stimuli. Consequently, this hypothesis does not likely explain the increases we discovered in PT following treatment (i.e. in the absence of concurrent stimulation). Interestingly, prior studies suggest the prolonged muscle function improvements following vibration exposure may be attributable to changes in
corticospinal excitability and intracortical processes (K. N. Mileva, Bowtell, & Kossev, 2009; Siggelkow et al., 1999). Accordingly, prospective research is needed to gain knowledge of the mechanism behind the muscle enhancements associated with WBV.

Another important finding was the presence of a trend approaching significance that quadriceps PT was still enhanced 10 and 20 minutes after WBV. Previous studies have reported increases in muscle force 5 minutes (Bazett-Jones et al., 2008) and 8 minutes (McBride et al., 2010) after vibration treatment. Moreover, many researchers credit the prolonged enhancements in muscle function succeeding vibration to elevated muscle temperature, synchronization, muscular coordination and proprioceptor response (Adams et al., 2009; Cardinale & Bosco, 2003; D. J. Cochrane et al., 2008). However, our findings are in contrast to those of Erskine et al. (2007b) who found no increase in knee extensor strength following WBV. The discrepancy in results could be due to heterogeneous stimulation parameters between the two studies (2g vs. 5g), as skeletal muscle is a specialized tissue that modifies its overall functional capacity in response to different stimuli (Erskine et al., 2007b). In particular, the neurological adaptations elicited from vibration are directly related to the characteristics of the load imposed by the stimulus (D. J. Cochrane, 2011; Rittweger, 2010). Therefore, the 2 g acceleration delivered in this study as opposed to 5 g in the aforementioned investigation may be more appropriate to elicit positive neuromuscular adaptations in the quadriceps. Consequently, vibratory parameters such as amplitude, frequency, duration, and acceleration should be investigated further in efforts to improve future vibratory protocols.

Although significant improvements were observed immediately following WBV, our statistical analysis did not detect differences in PT between groups at any time point. However, visual inspection of the data (Figure 2) indicated differential effects between groups. Moreover,
the ANOVA model had a power of 0.34 (ES = 0.40), suggesting that we may have been underpowered to detect differences in PT. Accordingly, exploratory post hoc power analyses revealed that 78 subjects per group would be necessary to achieve a power of 0.80. The lack of difference between groups may have been a result of the heterogeneous nature of our sample. Specifically, the sample utilized in this study consisted of individuals with diverse physical activity statuses, ranging from minimal physical activity to DI athletes. Consequently, the large variability in PT discovered within groups caused their respective standard deviations to overlap, producing insignificant statistical differences between the groups.

LMV did not influence PT at any point following application. Our hypothesis that PT would increase after LMV was based on several findings that demonstrated improvements in muscular function following LMV (Bongiovanni & Hagbarth, 1990; Couto et al., 2013; Iodice et al., 2011; Mischi & Cardinale, 2009; Ribot-Ciscar, Butler, & Thomas, 2003). For example, Iodice et al. (2011) revealed that leg extensor muscle strength and jump performance increased following acute and prolonged exposure to LMV. Moreover, LMV’s ability to elicit neuromuscular improvements along with its portability capabilities and cost effectiveness made it an appealing modality to investigate in effort to find an alternative to large and expensive WBV platforms. However, other studies have suggested a decline in force output following LMV (Kouzaki, Shihohara, & Fukunaga, 2000; Mottram et al., 2006), which could be associated with the parameters of stimulation. Accordingly, the inconsistencies in our PT results between WBV and LMV could be attributable to differences in the somatosensory receptors that are targeted by the WBV and LMV. For example, WBV stimulates receptors in skin and musculature surrounding the ankle and knee joints as well as the receptors within these joints, all of which potentially influence quadriceps function (Pollock et al., 2010). Conversely, LMV’s
effects are likely isolated to receptors surrounding the muscle-tendon unit to which it’s applied, potentially limiting its effectiveness. However, Blackburn et al. (2014) demonstrated significant improvements in quadriceps function following LMV in individuals with artificially induced quadriceps inhibition similar to individuals with knee pathologies. As such, this form of vibratory stimulus may be effective for enhancing quadriceps function in individuals with pathological knees, and prospective investigations are necessary to elucidate its effects since WBV platforms can be cost prohibitive and provide limited portability.

We did not observe a significant change in RTD subsequent to WBV or LMV at any point following treatment. RTD is influenced by neural and mechanical factors, and improved RTD is derived from increases in neural drive (i.e. firing frequency and motor unit activation) (Aagaard et al., 2002). Hence, our hypothesis that RTD would increase after VT was based on findings of improved alpha motorneuron excitability via the muscle spindle system (Cardinale & Bosco, 2003; Ritzmann et al., 2013) which could cause increases in firing frequency and motor unit activation (Burke & Gandevia, 1995; Eklund & Hagbarth, 1966). In opposition to our findings, Tihanyi et al. (2007) discovered a 19% increase in RTD in patients with stroke during a maximal isometric contraction following WBV. However, our subjects were young, recreationally active and healthy, which is in contrast to the sample of elderly stroke victims investigated by Tihanyi et al. It is plausible that the healthy individuals in our study were functioning at near-maximal motor unit firing frequency, and their improvements in PT resulted from recruitment of additional high threshold motor units and not increased rate coding. In brief, as our sample likely possessed “normal” neural drive during maximal contraction, they may not have had the capacity to increase firing frequency, thus limiting increases in motor unit firing frequency. Also, the lack of an effect of vibratory stimuli on RTD could be attributed to data
processing techniques. In our study, change in torque (final-initial) was calculated over successive 20ms intervals and divided by time to represent RTD. Conversely, Tihanyi et al. (2007) discovered a significant change in RTD by calculating RTD as the tangent \( \frac{dM}{dt} \) fitted to the steepest part of the torque-time curves. Consequently, future studies are needed to determine the most effective methodological approach to calculate RTD.

Quadriceps PT and RTD are important considerations for athletic performance and injury prevention, and key determinants for the elderly’s capacity to complete activities of daily living (Bohannon & Andrews, 1990; Perry et al., 2007; Thompson et al., 2013; Winters & Rudolph, 2014). Moreover, muscle strength plays a significant role in balance and ambulation, and even healthy elderly persons complete activities of daily living such as stepping and walking at levels near their maximal voluntary joint torques (Hortobagyi et al., 2003; Kim & Eng, 2003). Pohl et al. (2002) reported that RTD is a better predictor of gait speed in stroke patients than PT. Moreover, rapid RTD has been shown to compensate for decreased quadriceps strength by delivering counter forces quickly in response to external demands (Nadeau et al., 1999; Winters & Rudolph, 2014). Unfortunately, individuals with knee OA and ACL injury often have deficits in PT and RTD originating from inhibited voluntary quadriceps activation (Hart et al., 2010; Pietrosimone et al., 2011), which have been shown to reduce the effectiveness of rehabilitation (Hopkins & Ingersoll, 2000; Hurley et al., 1994). Consequently, the prolonged effects of WBV on muscle function and its potential for producing local and peripheral neuromuscular enhancements suggest it could represent an effective exercise intervention for improving the performance of both competitive athletes and the general population. More importantly, our findings carry significant clinical applications, as VT could be incorporated in rehabilitation programs aimed at improving muscle strength in patients with neuromuscular deficits.
LIMITATIONS

There are inherent limitations associated with our study that should be considered when interpreting the results. First, our findings may reveal a ceiling effect due to the presence of healthy, young, and recreationally active subjects. Accordingly, a study conducted by Blackburn et al. (2014) that compared the effects of WBV and LMV on individuals with artificially induced quadriceps arthrogenic inhibition discovered greater benefits from vibration in individuals with more severe quadriceps inhibition. Additionally, other studies have reported reductions in pain and inflammation and increases in proprioception following VT (Aaboe et al., 2009; Kitay et al., 2009; Simão et al., 2012). Therefore, the positive effects of vibration stimuli may be greater in pathological and elderly populations who possess neuromuscular deficits. Secondly, our study did not investigate the mechanisms by which WBV enhances maximal voluntary quadriceps strength, which could be a result of enhanced excitation of alpha motorneurons via the muscle spindle system (Burke & Gandevia, 1995; Eklund & Hagbarth, 1966) or may be credited to alterations in corticospinal excitability and intracortical processes (K. N. Mileva et al., 2009; Siggelkow et al., 1999). Lastly, we did not investigate the source of increased quadriceps PT following WBV. Specifically, we did not measure the associated agonist and antagonist muscle co-activation patterns, which means the positive net force discovered from knee extension may have originated from increased agonist activity, reductions in antagonist activity, or a combination of both. Accordingly, Tihanyi et al. (2007) studied EMG\textsubscript{rms} during eccentric contractions in individuals with stroke and discovered a 33% increase in the vastus lateralis and a 23% decrease in the biceps femoris after WBV. Future research should evaluate the effects of vibration on agonist and antagonist musculature concomitantly.
CONCLUSIONS

Our findings suggest that WBV significantly increases quadriceps PT in healthy individuals immediately following vibration, and these enhancements may persist for up to 20 minutes following treatment. Based on these findings, WBV may improve traditional exercise and rehabilitation programs through neuromuscular enhancements. However, LMV had no effect on PT. Additionally, WBV and LMV had no effect on RTD. Future research is necessary to investigate the physiological mechanism associated with VT and determine the most effective delivery methods and stimulation parameters.
REFERENCES


