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Samuel T. Johnson

The nervous system has an integral role in rapid torque production of the lower extremity, which is often necessary for proper motor response to a dynamic environment. The nervous system is complex with numerous pathways, including spinal motor control networks, which influence the ability to move. However, few studies have investigated the influence of spinal motor control mechanisms on this ability to produce torque rapidly, often measured by the rate of torque development (RTD), after an explosive strength training program. Therefore, the purpose of this study was to investigate spinal motor control mechanisms following a three week explosive strength training program of the plantarflexors. Specifically, RTD, H-reflexes, supraspinal neural drive as measured by V-waves, and postsynaptic inhibition as measured by recurrent inhibition, were investigated during explosive contractions. The dependent variables used to measure these physiological

characteristics were RTD from 0-100 ms, H_{max} to M_{max} ratio (H_{max}:M_{max}), V-wave to M_{max} ratio (V:M_{max}), and percent recurrent inhibition at both 10% and 30% of M_{max}. None of these dependent variables showed a significant interaction or main effects across either group (control and training) or time (pre-test and post-test). Although expected that changes would occur in spinal motor control mechanisms, it is unknown if lack of changes are related to the training program's failure in producing significant adaptations related to RTD. More research is necessary to more fully understand the role of spinal motor control mechanisms on adaptations following explosive strength training.

Spinal Motor Control Adaptations to Explosive Strength Training

by

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APPROVED:

Major Professor, representing Exercise and Sport Science

Co-Director of the School of Biological and Population Health Sciences

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Dean of the Graduate School

I understand that my thesis will become part of the permanent collection of Oregon State University libraries. My signature below authorizes release of my thesis to any reader upon request.

Clara A. Stone, Author

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Chapter 1

Introduction

The ability to rapidly generate muscular force, especially in early phases of muscle contraction, allows individuals to more effectively respond to potentially injurious situations and perform functional motor tasks. For example, more than 300 ms is often needed to reach peak muscle force (Thorstensson, Karlsson, Viitasalo, Luhtanen, & Komi, 1976). However, anterior cruciate ligament injury may occur within 40 ms of landing (Koga et al., 2010) which is well before maximal force can be used to respond to the situation. Additionally, many performance tasks also occur in less than 250 ms, such as sprinting (Kuitunen, Komi, & Kyröläinen, 2002) and high jumping (Dapena & Chung, 1988). Therefore, measures of rapid force production rather than peak force may be more functional. Despite its functional importance the underlying mechanisms of rapid force production are not fully known.

Previous researchers have identified both muscular and neural mechanisms that contribute to this rapid force generation. An area of particular interest is spinal motor control mechanisms due to their role in modulation of motor output (Palmieri, Ingersoll, & Hoffman, 2004). Cross-sectional research on the Hoffmann (H)-reflex, a measure of net motor neuron pool excitability, found that explosively trained athletes had lower H_{max} to M_{max} ratios than endurance trained and untrained individuals (Casabona, Polizzi, & Perciavalle, 1990; Maffiuletti et al., 2001). While an important finding, the H-reflex alone does not fully illustrate the mechanistic complexity in neural modulation of motor output. Several underlying mechanisms such as presynaptic and postsynaptic inhibition as well as supraspinal neural drive contribute to modulation of motor output in the spinal cord (Zehr, 2002).

Recurrent inhibition, a specific type of postsynaptic inhibition resulting from activation of Renshaw cells, is thought to be a variable gain regulator of motor neuron pool output (Hultborn, Brownstone, Toth, & Gossard, 2004; Hultborn, Lindström, & Wigström, 1979), and may have a role in synchronization of motor unit firing frequency (Knikou, 2008; Maltenfort, Heckman, & Rymer, 1998; Mattei et al., 2003). Through recurrent inhibition's possible role in firing frequency (Aagaard, 2003; Knikou, 2008), which is known to increase explosive strength (Aagaard, 2003), changes in recurrent inhibition could influence explosive strength. In an attempt to understand these mechanisms, one study found recurrent inhibition, a postsynaptic inhibitory mechanism, was greater in explosively trained athletes compared to endurance trained athletes (Earles, Dierking, Robertson, & Koceja, 2002). Additionally, it was reported that recurrent inhibition was associated with early phases of explosive strength production when tested at rest (Johnson, Kipp, Norcross, & Hoffman, 2014). However, it is unknown if recurrent inhibition differences in variously trained individuals can be attributed to genetic predisposition or neural plasticity after explosive strength training.

While little information exists on changes in motor neuron pool excitability following explosive strength training programs, some studies have used the H-reflex to investigate spinal motor control measures before and after strength training interventions. Consistently, no change in H_{max}:M_{max} or H_{slope}:M_{slope} was found when the H-reflex was tested at rest (Aagaard, Simonsen, Andersen, Magnusson, & Dyhre-Poulsen, 2002; Del Balso & Cafarelli, 2007; Gruber et al., 2007; Holtermann, Roeleveld, Engstrøm, & Sand, 2007). However, testing H-reflexes at rest is problematic because the nervous system is highly variable and dependent on the functional task (Zehr, 2002). Therefore, resting H-reflex measures may not fully capture adaptations that occur during movement. In contrast to the lack of changes seen at rest, an increased H-reflex

amplitude was seen after training when tested during maximal isometric ramp contractions (Aagaard et al., 2002). Vila-Cha et al. noted a decreased H-threshold during 10% background contraction after both strength and endurance training (2012). While there is no information on changes in recurrent inhibition after a strength training program, there have been reported changes in V-waves after strength training (Aagaard et al., 2002; Del Balso & Cafarelli, 2007; Vila-Chã, Falla, Correia, & Farina, 2012). V-waves, which are measured by applying a supramaximal stimulus during a maximal voluntary contraction, provide a measurement of supraspinal neural drive. The changes observed in V-waves with training support the essential involvement of the central nervous system in influencing strength measures.

While these studies indicate changes in H-reflexes tested during a contraction and V-waves after strength training, little is known about how these variables, as well as recurrent inhibition, change following an explosive strength program. Coupled with the results of cross-sectional studies, this research indicates differences in recurrent inhibition between explosive and endurance trained individuals. However, there is a gap in knowledge concerning whether recurrent inhibition can change following a short-term explosive training program. A greater understanding of spinal motor control adaptations, specifically recurrent inhibition, after training, could help answer *why* changes in explosive strength occur and provide a link between spinal control mechanisms and explosive strength production. This information could help pinpoint specific adaptations to explosive strength training to produce more specific functional training outcomes. Therefore, the primary purpose of this study was to investigate changes in H-reflexes, V-waves, and recurrent inhibition tested during an explosive contraction following a three week explosive strength training program of the plantarflexors.

Chapter 2

Literature Review

2.1 Introduction

While it has long been accepted that neural mechanisms contribute to initial strength gain following training (Aagaard, 2003), the underlying mechanisms are less understood. Specifically, there is a knowledge gap concerning the adaptations to motor neuron pool excitability with explosive strength training. A greater understanding of these mechanisms could have valuable implications for developing explosive strength in relation to avoiding injurious situations, rehabilitating after injury, and enhancing athletic performance.

2.2 The Nervous System and Explosive Strength

Increases in explosive strength allow individuals to more quickly respond to their environment during unexpected events such as catching a fumbled object or adjusting to uneven ground. Athletes, especially those in sports involving sprinting, jumping, or avoiding collision, require explosive muscle contraction for performance. It is well established that the nervous system is integral in increasing strength and rate of torque development (RTD), which is defined as the slope of the torque-time curve (Gabriel, Kamen, & Frost, 2006; Van Cutsem, Duchateau, & Hainaut, 1998). Therefore, neurological adaptations to resistance training have many implications for enhancing motor skills both in force production and control of movement.

Evidence for this neural contribution to strength is given by the short time with which adaptations occur. For example, increases in maximal voluntary contraction (MVC) and RTD have been observed within the first eight weeks of resistance training before measureable muscle hypertrophy (Moritani & deVries, 1979). Therefore, the initial increase in force is attributed to

nervous system adaptations, and further research explains the mechanisms behind this strength gain. More specifically, muscle contraction is effected by the nervous system's ability to activate muscle fibers through motor unit recruitment and firing frequency, also known as rate coding (Aagaard, 2003). Both motor unit recruitment and rate coding act together to produce muscular force. The nervous system is thought to be able to synchronize the activation of motor units which can also result in increased force production (Aagaard, 2003). Doublet discharges, which are characterized by particularly short intervals between firing action potentials, are another specific pattern of rate coding (Gabriel et al., 2006). Supporting evidence for the contribution of doublet discharges in producing muscular force is presented by Van Cutsem et al. who investigated motor unit doublet discharges after 12 weeks of dynamic training (1998). In addition, motor unit firing frequency has a large impact on explosive strength, which is related to muscle contraction speed (Aagaard, 2003; Van Cutsem et al., 1998).

Less clear is the underlying mechanisms behind these adaptations, including motor neuron pool excitability. With a vast number of neurons that constantly communicate with each other via action potentials, the nervous system is complex. The signals received by alpha motor neurons are modulated at the spinal cord level through presynaptic and postsynaptic inhibition. There is a current lack of understanding concerning these spinal pathways and possible adaptations to explosive strength training. It is unknown whether these mechanisms could underlie the neural plasticity of alpha motor neurons.

2.3 Measure of Net Motor Neuron Pool Excitability: H-reflex

Through constant excitatory and inhibitory signals from surrounding neurons, the motor unit's sensitivity to action potentials is adjusted. As explained by Wolpaw, "All movements

reflect the interaction of supraspinal commands, sensory inputs, and spinal cord interneurons" (2001). The net motor neuron pool excitability of these interactions can be measured by the H-reflex.

The H-reflex is evoked by stimulation of a mixed peripheral nerve. After stimulation of the peripheral nerve, action potentials travel along Ia sensory (afferent) nerves to the spinal cord and synapse with alpha motor neurons. Next, action potentials travel down the alpha motor neurons to the muscle and cause a contraction, which is called an H-reflex. The stimulation also causes a shorter, direct activation of the alpha motor neurons, called an M-wave, as well as antidromic action potentials that travel towards the spinal cord. These antidromic action potentials collide with orthodromic signals that started with the initial stimulation of the afferent nerves. As the stimulus intensity increases, the antidromic action potentials will eventually cancel out the orthodromic action potentials in the alpha motor neuron. This means that the M-wave will continue to increase until all motor units are recruited while the H-reflex eventually disappears (Palmieri et al., 2004).

This method provides researchers with the ability to investigate the human nervous system noninvasively. Therefore, the H-reflex is a valuable tool for gaining insight into how the nervous system relates to motor control. Variations of the H-reflex can also be used to measure more specific neural mechanisms and components of net motor neuron pool excitability, including postsynaptic inhibition, presynaptic inhibition, and supraspinal neural drive.

2.4 Recurrent Inhibition

Recurrent inhibition is a postsynaptic inhibitory mechanism. As action potentials travel down an alpha motor neuron axon toward the muscle, several collateral axons branch off and

synapse with interneurons. The interneurons, called Renshaw cells, release inhibitory signals to the alpha motor neurons, thereby decreasing alpha motor neuron excitability (Knikou, 2008).

Recurrent inhibition is measured in the soleus by comparing an unconditioned H-reflex, called H1, to a conditioned H-reflex, called H'. H1 is obtained by stimulating the common tibial nerve with a single stimulus at a consistent percentage of M_{max}. This unconditioned stimulus causes activation of motor units and a normal H-reflex as described previously. H' is obtained by two stimuli given 10 ms apart. The intensity of the first stimulus is equal to that used to obtain H1, and the second stimulus is supramaximal. Because the first stimulus activated alpha motor neuron collaterals that resulted in Renshaw cell inhibitory signals, the H' is inhibited as compared to H1. By comparing H' to H1, researchers can obtain an estimate or measure of recurrent inhibition (Katz & Pierrot-Deseilligny, 1999).

Interestingly, this method can only evaluate recurrent inhibition of the motor units first activated by the submaximal stimulus given to determine H'. This method relies on the orthodromic action potentials of the conditioned stimulus to collide and cancel out the antidromic action potentials of the H' test stimulus. Only then are the afferent, orthodromic signals of the test stimulus allowed to reach the muscle (Bussel & Pierrot-Deseilligny, 1977).

As a regulator and contributor of postsynaptic inhibition, recurrent inhibition is an important factor in firing frequency of motor units. For example, recurrent inhibition has a role in stabilization of firing frequency and inhibition of motor units near threshold (Granit, Haase, & Rutledge, 1960; Maltenfort et al., 1998). Recurrent inhibition also affects the timing and possibly the synchronization of motor units (Knikou, 2008; Maltenfort et al., 1998). As mentioned previously, control of firing frequency effects RTD (Aagaard, 2003), which has a clear role in functional tasks. In addition, recurrent inhibition at rest (along with supraspinal drive) was found

to be a significant predictor of RTD, especially during the early phase of contraction (less than 100 ms) (Johnson et al., 2014). Knowledge concerning recurrent inhibition adaptations with explosive strength training could have implications for increasing RTD through training both in rehabilitation and in athletic populations.

2.5 Supraspinal Neural Drive

During voluntary contraction, a signal is sent from supraspinal centers through descending tracts to the muscle, thereby causing muscular contraction. Collectively, these signals are termed supraspinal or volitional neural drive and can be studied by evaluating V-waves.

V-waves are elicited by a supramaximal stimulus while the participant is maximally contracting (Upton, McComas, & Sica, 1971). During the MVC, electrical signals to the skeletal muscle originate in the motor cortex, travel down the corticospinal tract, and cause motor unit activation, which results in muscle contraction. These voluntary signals traveling to the muscle collide with antidromic signals from the supramaximal stimulus. This collision then opens up the pathway for the orthodromic reflex signal to reach the muscle and cause a contraction, called a V-wave (Aagaard et al., 2002; Upton et al., 1971).

By measuring V-wave amplitude, researchers may infer possible amounts of neural drive from descending neural pathways. By understanding V-wave measures, researchers can more accurately determine where neural changes do and do not occur. This gives more specific mechanistic knowledge beyond the net motor neuron pool excitability determined by H-reflex amplitude alone.

2.6 Cross-Sectional Studies Investigating Motor Neuron Pool Excitability

Interestingly, individuals with long-term training have different motor neuron pool excitability profiles as indicated by H-reflex differences. A cross-sectional study of explosively trained sprinters and volleyball players compared to a non-trained population showed significantly lower H-reflexes of the explosively trained (Casabona et al., 1990). This study measured H-reflexes as a proportion of maximum H-reflex amplitude (H_{max}) to maximum M-wave amplitude (M_{max}). This ratio, H_{max}:M_{max}, describes the motor neuron pool activation at H_{max} normalized to activation of the entire motor neuron pool and is a common variable in assessing H-reflexes (Palmieri et al., 2004). In agreement with these results, Maffiuletti, Martin, Babault, Pensini, Lucas, & Schieppati found endurance trained athletes had increased H_{max}:M_{max} while power-trained athletes had decreased H_{max}:M_{max} compared to a control group (2001).

In addition to H_{max}:M_{max}, researchers also investigated twitch strength and rate of twitch tension of power and endurance trained athletes (Maffiuletti et al., 2001). They concluded power trained athletes had a greater overall force output, but endurance trained athletes had a greater motor neuron pool excitability (Maffiuletti et al., 2001). This could provide evidence that explosive training may change the input-output gain of motor units. In other words, explosively trained athletes may require less neural input to produce a given amount of motor output. These observed differences in H-reflex profiles suggest specificity of training adaptations on motor unit excitability. More specifically, the decrease in motor neuron pool excitability seen in explosively trained athletes could result from an increased ability to regulate force production via spinal pathways.

The authors speculate that the differences in motor neuron pool excitability profiles among endurance and power trained individuals could be attributed to fiber type and motor unit

size (Casabona et al., 1990; Maffiuletti et al., 2001). The authors argue, that, due to a higher percentage of type II fibers, explosively trained athletes may need to recruit fewer motor units to produce the same amount of force as endurance trained athletes with a higher proportion of type I fibers. This might explain the decrease in motor neuron pool excitability of power trained individuals. However, because the soleus is primarily used for postural control and is 70-90% slow twitch fibers (Edgerton, Smith, & Simpson, 1975), it seems unlikely that these significant differences observed in the soleus by Maffiuletti et al. can fully be attributed to differences in fiber type.

Through a cross-sectional study, a group of researchers investigated postsynaptic and presynaptic inhibition (Earles, Dierking, et al., 2002). Specifically, recurrent inhibition and intrinsic presynaptic inhibition were measured in power athletes, endurance athletes, and untrained individuals. Intrinsic presynaptic inhibition, which is modulation of incoming Ia afferent action potentials by previously activated nerves (Rudomin & Schmidt, 1999), was assessed using a paired reflex depression protocol (Earles, Dierking, et al., 2002; Earles, Morris, Peng, & Koceja, 2002). Participants were tested at 10% and 30% of M_{max} while standing. Recurrent inhibition and intrinsic presynaptic inhibition were greater at 30% M_{max} compared to 10% M_{max}. This implies a greater amount of inhibition occurs when a greater proportion of the motor neuron pool is activated (Earles, Dierking, et al., 2002).

When recurrent inhibition was analyzed between groups (explosive, endurance, and untrained), the researchers found explosive athletes and untrained individuals demonstrated greater recurrent inhibition compared to endurance athletes. However, intrinsic presynaptic inhibition showed the opposite result with endurance athletes having greater intrinsic presynaptic inhibition. The authors suggest that the increased recurrent inhibition could be related to the

greater number of larger, type II motor units typically recruited by explosively trained athletes (Earles, Dierking, et al., 2002). Activation of larger, type II fibers recruited in ballistic movements may result in more activation of alpha motor neuron collaterals. These collaterals, which are thought to be more numerous in larger motor units, may activate more Renshaw cells, resulting in greater recurrent inhibition. (Earles, Dierking, et al., 2002; Earles, Morris, et al., 2002).

However, the differences observed in both recurrent inhibition and intrinsic presynaptic inhibition could also result from spinal motor control adaptations which are known to occur after various activities, such as endurance training, hopping training, and balance training (Pérot, Goubel, & Mora, 1991; Trimble & Koceja, 1994; Voigt, Chelli, & Frigo, 1998). As a known contributor to reflex gain (Hultborn et al., 1979), recurrent inhibition in explosively trained athletes could thereby be an influencing factor to the decreased H_{max}:M_{max} as compared to endurance trained and untrained individuals (Casabona et al., 1990; Maffiuletti et al., 2001). Further, the model of recurrent inhibition as a negative feedback loop with the number of activated collaterals corresponding to a specific amount of inhibition may be too simplified. In addition to alpha motor neuron collaterals, Renshaw cells receive input from synergistic and neighboring motor neurons, descending tracts, and other Renshaw cells (Katz & Pierrot-Deseilligny, 1999). This creates a complex, intricate network that is effected by the entire motor unit and its synapses, not just the muscle fiber type.

While fiber type may be a contributing factor, neural changes in presynaptic inhibition, postsynaptic inhibition, and supraspinal drive may also play a critical role. Filling the gap in knowledge concerning neural mechanisms relating to differences in athletes' H-reflex profiles requires investigation using longitudinal rather than cross-sectional studies. Longitudinal studies

could provide additional evidence towards the role of neural adaptation rather than genetic predisposition concerning motor neuron pool excitability characteristics.

2.7 Training Studies

A few studies have examined spinal motor control variables before and after training protocols. These studies provide a more comprehensive look into spinal motor control adaptations. However, while differences in testing condition, training protocols, and dependent variables analyzed provide unique and interesting information, they also make synthesizing results difficult. These training intervention studies raise further questions regarding explosive strength training and specific spinal motor control mechanisms.

Gruber et al. (2007) investigated neural adaptations following four weeks of either a balance or ballistic strength training program. Balance exercises consisted of various postural tasks using wobble board, spinning top, soft mat, and cushion. Ballistic strength exercises consisted of plantarflexion and dorsiflexion movements against 30-40% of participants' one repetition maximum. During contractions, participants were instructed to contract with maximal force and speed. Before and after training, the researchers measured stretch reflex peak-to-peak amplitude, H_{max}:M_{max}, dynamic postural control, maximal voluntary contraction (MVC), maximal RTD, and time to achieve maximal RTD. (Please note that the authors used slightly different terminology in their article. Specifically, they used "rate of force development" which has been replaced with "rate of torque development" in this document for consistent terminology. Based on the described methods, these terms were determined to be interchangeable). (Gruber et al., 2007)

After training, both balance and strength groups significantly increased maximal RTD while MVC remained unchanged for all groups. The ballistic strength training group also displayed a decreased time to reach maximal RTD while the balance and control groups did not. The balance group displayed a significant decrease in both stretch reflex amplitude and H_{max}:M_{max} while the strength and control groups did not significantly change after training. The authors attribute the changes seen in the balance group to an increase in presynaptic inhibition modulated from supraspinal centers. Due to the lack of changes in neural measures observed after ballistic strength training, the authors concluded that the decreased H-reflexes seen in cross-sectional studies of power trained individuals are due to genetic factors rather than neural adaptations. (Gruber et al., 2007)

However, this study only measured motor neuron pool excitability at rest. During voluntary contraction, motor neuron pool excitability is particularly subject to neural drive, presynaptic inhibition, and postsynaptic inhibition. Therefore, it is recommended that H-reflexes be obtained during a consistent voluntary background contraction (Zehr, 2002). It is also critical that this voluntary contraction be set to an ascending portion of the H-reflex recruitment curve in order to avoid interference from antidromic collisions (Grosprêtre & Martin, 2012). Because Gruber et al. did not test motor neuron pool excitability during contraction, it may be premature to conclude lower H-reflex profiles seen in power athletes result only from genetic predisposition and muscle architecture.

Another longitudinal study investigated neuron pool excitability after a lengthier intervention consisting of 38 sessions over 14 weeks (Aagaard et al., 2002). The training group performed heavy resistance exercises including seated calf raise, hack squat, incline leg press, isolated knee extension and hamstring curl. Before and after training, the researchers compared

V-waves normalized to M_{max} ($V:M_{max}$), $H_{max}:M_{max}$ at rest, $H_{max}:M_{max}$ at 20% M_{max} during an isometric ramp contraction, and maximal eccentric and concentric plantarflexion strength. Stimuli for both V-waves and H-reflexes assessed during contraction were triggered at 90% MVC. (Aagaard et al., 2002)

After training, the researchers found a significant increase of both motor neuron pool excitability during contraction and supraspinal drive. Specifically, $H_{max}:M_{max}$ during contraction increased approximately 20% whereas $V:M_{max}$ increased approximately 50%. During rest, there was no significant change in $H_{max}:M_{max}$ after training, which is consistent with the previously mentioned findings of Gruber et al. (2007). Overall, these results indicate neural adaptations with strength training, even though changes were only seen during contraction. (Aagaard et al., 2002)

Changes in neural adaptations both during contraction and rest were investigated by Holtermann, Roeleveld, Engstrom, & Sand (2007) before and after a three week strength training protocol. In the experimental group, twelve participants performed isometric plantarflexion exercises three times a week for three weeks. Each training session consisted of five sets of ten repetitions with subjects holding each maximal, isometric contraction for four seconds. Subjects rested for ten seconds between reps and three minutes between sets. The researchers measured RTD and H-reflex amplitude both at rest and at 20 and 60% of MVC. In addition to a 28.4% increase in RTD, Holtermann et al. found a significant increase of H-reflex amplitude at both 20 and 60% MVC but not at rest (2007). H-reflex amplitude at 20% MVC was positively correlated with increases in RTD after training. However, when analyzed across the training and non-training groups, H-reflex amplitudes were not significantly different from pre to post tests. This could be due to the control group having higher baseline amplitudes than the experimental group, or, as reasoned by the authors, to small sample sizes. The authors found no significant change of

H_{max}:M_{max} after the training program (Holtermann et al., 2007). However, as in the study by Gruber et al. (2007), H_{max}:M_{max} was tested during rest, and the authors attribute this lack of change to H-reflex testing without a background voluntary muscle contraction. (Holtermann et al., 2007)

Avoiding the weakness in assessing motor neuron excitability at rest, another group of researchers tested individuals during a background contraction of 10% MVC after three weeks of either endurance or strength training (Vila-Chã et al., 2012). After endurance training, which consisted of cycling on an ergometer, the researchers found a significant increase in time-to-task failure and an increase in motor neuron pool excitability as indicated by increased H_{max}:M_{max} and H_{slope}:M_{slope}. In contrast to the endurance group, the strength group displayed a significant increase in MVC after training but did not display any significant changes in H_{max}:M_{max} or H_{slope}:M_{slope}. The strength group also significantly increased V:M_{max} while the endurance group did not. Interestingly, both groups developed a significantly decreased H-threshold after training. (Vila-Chã et al., 2012)

These results indicate differing neural adaptations following either endurance or strength training. The lack of H_{max}:M_{max} and H_{slope}:M_{slope} changes after strength training as tested during a background contraction (Vila-Chã et al., 2012) are also in agreement with findings at rest (Aagaard et al., 2002; Del Balso & Cafarelli, 2007; Gruber et al., 2007; Holtermann et al., 2007). However, Vila-Cha et al. did not investigate changes in RTD. Therefore, it is unknown whether the observed changes in motor neuron pool excitability, or lack of changes, can be linked to RTD. Difficulty also arises when comparing these results with Holtermann et al. who positively correlated H-reflex amplitude at 20% MVC with increases in RTD after training (2007).

Del Balso and Carfarelli addressed this limitation by measuring MVC, RTD, H-reflexes, and V-waves before and after a four week isometric training program of the plantarflexors (2007). The authors concluded that increased MVC after isometric resistance training was likely due to increased supraspinal neural drive rather than changes in motor unit excitability. Although there was a significant increase in MVC and RTD, H-reflexes (as compared by H_{slope}:M_{slope}) did not significantly change when measured both at rest and during a 10% background contraction. Therefore, in agreement with Vila-Cha et al., the authors attribute the change in MVC to the significant increase in V-wave amplitude (2007). The authors state they have "…attempted to link changes in descending volitional drive, the spinal cord, the alpha-motor neuron pool, and the rates at which muscle is activated and develops torque to an early increase in MVC" (Del Balso & Cafarelli, 2007). This research provides valuable knowledge concerning neural adaptations to resistance training. However, while volitional neural drive seems to be a key component of increases in RTD, the authors do not address the possible roles of presynaptic inhibition, reciprocal inhibition, or recurrent inhibition (Del Balso & Cafarelli, 2007).

When evaluated at rest, the discussed training studies consistently demonstrate no change in motor neuron pool excitability after strength training (Aagaard et al., 2002; Del Balso & Cafarelli, 2007; Gruber et al., 2007; Holtermann et al., 2007). During a 10% voluntary background contraction, Vila-Cha et al. found a decrease in H-threshold but no change in H_{max}:M_{max} or H_{slope}:M_{slope} (2012). Aagaard et al. found a change in motor neuron pool excitability when tested during maximal isometric ramp contractions (2002). Although these studies provide valuable knowledge towards understanding motor neuron excitability adaptations with strength training, the inconsistencies in background contraction during testing make

interpretation difficult. Several of the studies found increases in supraspinal drive after training (Aagaard et al., 2002; Del Balso & Cafarelli, 2007; Vila-Chã et al., 2012). Few of the studies investigated presynaptic and postsynaptic contributors to net motor neuron excitability. Instead, the previous research indicates changes in supraspinal neural drive as contributors to RTD after short term explosive strength training (Del Balso & Cafarelli, 2007).

As a net measure of motor neuron pool excitability, the H-reflex by itself paints an incomplete picture of neural adaptations. Information regarding V-wave adaptations adds to current scientific knowledge, but is not comprehensive. While these studies present valuable insight into motor neuron pool excitability, there are still inconsistencies relating to specific variables and conditions analyzed. Further, possible adaptations concerning recurrent inhibition are still unknown.

2.8 Clinical Implications

A greater knowledge of motor neuron pool excitability and recurrent inhibition adaptations to explosive strength training is essential for a comprehensive understanding of neural adaptation. By determining the contribution of specific spinal mechanisms behind explosive strength training, researchers can better answer *why* changes in strength, RTD, and functional ability occur. When researchers know what normal looks like, they are more easily able to identify abnormalities that could contribute to disease or dysfunction. This broader basis of neural physiology allows researchers to better grasp the nervous system's complexities and comes with the potential for application. Pinpointing specific adaptations to explosive strength training could produce more purposeful manipulation of the nervous system for more specific functional and training outcomes.

A more comprehensive knowledge of motor neuron pool excitability may have implications for injury rehabilitation. It is commonly accepted that neural plasticity occurs in the spinal cord after disease or injury, such as spinal cord damage (Wolpaw & Tennissen, 2001). An examination of individuals with chronic ankle instability (CAI) found that individuals with CAI had significantly greater recurrent inhibition then a control group. The results also showed healthy individuals could adjust presynaptic inhibition when changing from a double to single leg stance whereas individuals with CAI could not (Sefton et al., 2008). In agreement, decreased H_{max}:M_{max} in the soleus and peroneals with functional ankle instability have also been reported (McVey, Palmieri, Docherty, Zinder, & Ingersoll, 2005). In an investigation of Achilles tendinopathy, elite athletes showed increased V-waves and decreased RTD of involved Achilles without significant difference in H-reflex measures (Wang et al., 2011). These studies investigating spasticity, CAI, and Achilles tendinopathy indicate neuromuscular manifestations of injury. If explosive strength training increases motor neuron pool excitability and decreases recurrent inhibition, then clinicians may better design and implement more effective rehabilitation protocols.

Spasticity treatment may also benefit from study of neuromuscular mechanisms. Spasticity, often characterized by tonic muscles and hyperreflexia, is a common result of stroke, spinal cord damage, and other neurological diseases such as cerebral palsy and multiple sclerosis. Several studies indicate a pathological change in spinal mechanisms, including presynaptic, postsynaptic, and recurrent inhibition, in spastic patients (Morita, Crone, Christenhuis, Petersen, & Nielsen, 2001; Nielsen, Crone, & Hultborn, 2007). More specifically, a recent study investigated the effect of botulinum neurototoxin type A (BoNT-A), a common therapeutic treatment for tonic muscles, on recurrent inhibition in patients with lower leg

spasticity developed after stroke. BoNT-A treatment was correlated with depression of recurrent inhibition in spastic patients and a decrease in muscle tone (Marchand-Pauvert et al., 2013). These findings indicate recurrent inhibition changes as a possible causative factor in spasticity. Therefore, training programs resulting in decreased recurrent inhibition could have implications in future spasticity treatment.

Understanding the basic neural adaptations to strength training provides a foundation for further research of motor neuron pool excitability and its underlying mechanisms, including volitional neural drive and recurrent inhibition. While cross-sectional studies demonstrate differences between individuals with varying strength and endurance training backgrounds (Casabona et al., 1990; Maffiuletti et al., 2001; Nielsen, Crone, & Hultborn, 1993), the role of genetic influence is unknown. Investigation of explosive strength training adaptations on motor pool excitability and its underlying mechanisms could have future application in increasing RTD and rehabilitating spasticity, chronic ankle instability, and other neuromuscular pathologies.

Chapter 3

Materials & Methods

3.1 Participants

Twenty-four individuals (12 men and 12 women) volunteered to be in this study. See Table 1 for participant characteristics. All participants were recreationally active as defined by exercising at least 150 minutes per a week. Participants were excluded if they: 1) had any neurological disorder, 2) had any lower extremity or low back injury within the previous 6 months, 3) had any illness or injury that currently interfered with regular physical activity, 4) had a history of back or lower extremity surgery, or 5) ran on average more than 20 miles per a week. Participants agreed to maintain their current activity levels and not begin new hopping or lower leg strength training programs during the study duration.

Group	Sex	Age (yrs) Mean ± SD	Height (cm) Mean ± SD	Pre-Test Body Mass (kg) Mean ± SD	Post-Test Body Mass (kg) Mean ± SD
Control	Men	24.3 ± 4.5	177.8 ± 4.8	75.5 ± 6.3	75.6 ± 5.5
	Women	24.2 ±4.3	168.4 ± 7.4	66.5 ±13.3	66.0 ±13.4
Training	Men	22.3 ±3.5	181.4 ± 5.6	84.2 ± 16.7	83.8 ± 15.8
	Women	22.2 ± 2.0	169.2 ± 11.2	67.5 ±9.6	67.4 ± 9.7

Table 1: Participant characteristics including group, sex, age, height, pre-test body mass, and post-test body mass expressed as mean ± standard deviation.

3.2 Procedures

All participants reported to the Biomechanics Laboratory at Oregon State University for a pre-test. At the pre-test, participants gave written consent, and the study approved by the Oregon State University Institutional Review Board. Following consent, participants were screened to

ensure eligibility to participate in the study (See Appendix A for Screening Questionnaire). Leg dominance was then determined by recording the leg used to complete at least two of the following tasks: 1) kicking a ball, 2) reacting to a small, off-balancing push from behind, and 3) stepping onto a box (Hoffman, Schrader, Applegate, & Koceja, 1998). Height and weight were then measured as the last step before strength testing.

In order to measure plantarflexor torque and spinal motor control, participants were positioned on a Biodex System 3 dynamometer (Biodex Medical Systems Inc., Shirley, New York) that was interfaced with a BIOPAC MP100 Data Collection System (BIOPAC systems Inc., Goleta, California). The dominant foot was strapped against a rigid footplate attachment and the non-dominant leg placed in a comfortable position. Using a goniometer, the ankle joint was positioned in 10 degrees of plantarflexion and the knee was flexed 70 degrees from full extension. The trunk was reclined to 70 degrees. In order to improve participant comfort and limit sciatic nerve compression, participants sat on a high density foam pad. See Figure 1.

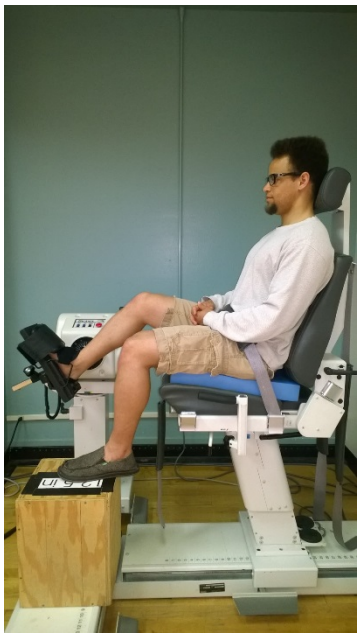

Figure 1: Strength Testing Set-up

Two EMG electrodes (Ag/AgCl) were placed 3 cm apart over the soleus muscle approximately 8 cm below the distal head of the gastrocnemius, just medial to midline, and parallel with suspected muscle fiber alignment. A reference electrode was placed on the medial malleolus. Before placement of the electrodes, the skin was cleaned with alcohol and slightly abraded.

Participants were first given the opportunity to practice isometric plantarflexion against the dynamometer footplate without torque being recorded. They were instructed to plantarflex

against the footplate using their ankle joint "like a gas pedal." Participants were given as much time as necessary for them to feel comfortable with the motion. Following the practice trials, participants were instructed to push against the rigid footplate "as hard and fast as possible" for about a second and then relax after the researcher said "go." Sixty seconds of rest was given between trials. A minimum of three valid trials was completed, with valid trials defined as those without a visible countermovements on the torque-time curve.

Following the plantarflexor torque trials, participants' spinal motor control was measured. A dispersive electrode (3 cm^2) was placed just superior to the ipsilateral patella. A stimulating electrode (2 cm^2) was placed over the tibial nerve in the popliteal fossa to elicit H-reflexes. The optimal placement of the stimulating electrode was determined by moving the electrode to various positions, applying a stimulus at each location, and looking for an H-reflex response at rest. All stimulations were applied using a Grass S88 stimulator (Grass Technologies, West Warwick, RI, USA) and 1 ms square waves. Once the optimal placement was determined, the electrode was held in place using tape, a small wooden block, and a rubber strap. See Figure 2.

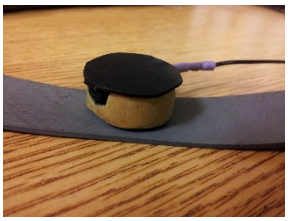

Figure 2: Stimulating electrode with wooden block and rubber strap

Following stimulating electrode placement, participants were instructed to push against the foot plate "as fast as possible" after the researcher said "go" and then relax once a stimulus was felt. Stimulations were automatically triggered once participants reached 20% of MVC. Twenty percent of MVC was determined using the average of the peak torque from the three highest valid RTD trials during the previous strength measures.

To measure spinal motor control, H-reflexes and M-waves were elicited during rapid contractions. Specifically the peak-to-peak amplitude of the H-reflex and M-wave were

measured and a full recruitment curve was mapped. Initially, the participant's M_{max} was determined by noting a plateau of the M-wave after delivering 5-10 supramaximal stimuli with approximately five seconds of rest provided between each stimulation. Following identification of M_{max}, $H_{\text{-threshold}}$ was found.

$H_{\text{-threshold}}$ was determined by providing sub-threshold stimulations of increasing intensity until an H-reflex was elicited. Once $H_{\text{-threshold}}$ was determined, the stimulus was gradually increased to find an H-reflex with a peak-to-peak amplitude of approximately 10% of M_{max}. When 10% of M_{max} was found, a stable H-reflex at 10% of M_{max} was established as defined by three consecutive H-reflexes within a 5-15% window of M_{max}. For some individuals this was difficult to find, possibly due to inherent H-reflex variability. Therefore, the acceptable window was expanded to 2-20% of M_{max} for these participants. At this intensity, recurrent inhibition was obtained using the procedure described by Katz and Peirrot-Deseilligny (1999).

Specifically, the procedure used to elicit recurrent inhibition relied on comparing conditioned and unconditioned H-reflexes. The unconditioned H-reflex was obtained by stimulating the common tibial nerve with a single stimulus at a consistent percentage of M_{max}-in this case 10% of M_{max}. The conditioned H-reflex was obtained by delivering two stimuli 10 ms apart, with the intensity of the first stimulus equal to the unconditioned H-reflex. The second stimulus was a supramaximal stimulus (i.e. M_{max}).

After recurrent inhibition at 10% of M_{max}, stimulus intensity was then gradually increased to 30% of $M_{max.}$ This was done using the same procedure as for 10% of M_{max} except for the greater target intensity. The target window for the H-reflex was 25-35% of M_{max}, and the expanded window was 20-40% of M_{max}. Using the same methods as described, three trials of both unconditioned and conditioned reflexes were obtained at 30% of M_{max}.

Following the recurrent inhibition measurement, the stimulus was gradually increased to determine H_{max} to complete the mapping of the H-reflex recruitment curve. Then, the stimulus intensity was further increased to find M_{max} again in order to fully complete the mapping of the M-wave recruitment curve.

Lastly, V-waves were used to measure the supraspinal neural drive of a voluntary contraction (Upton et al., 1971). Participants were instructed to isometrically plantarflex against the foot plate as "hard and fast as possible" after the researcher said "go." Once 90% of MVC was reached, a supramaximal stimulus was delivered. Three successful trials were obtained with 60 seconds rest between each. A trial was considered unsuccessful if the participant reported not contracting maximally.

At the end of the pre-test, participants were assigned to either the training or control group. This was done by having the participants select a piece of paper out of a hat with either "control" or "training" written on it. Once six men or six women were assigned to either the control or training group, all subsequent men or women were assigned to the other group. This ensured the control and training groups had equal numbers of men and women.

Participants in the training group completed three weeks of isometric explosive strength training of the plantarflexors (Holtermann et al., 2007). They completed three sessions per week with each session consisting of five sets of 10 contractions. Participants rested 10 seconds between each contraction and three minutes between each set. For each contraction, participants were instructed to push as hard and fast as possible for four seconds against a custom-built isometric plantarflexion training apparatus. The apparatus was designed so the participants, while seated, placed their foot on a wedge that positioned the foot in approximately 10 degrees of plantarflexion. A padded wooden board was placed over the thigh and strapped down along

the shank of the lower leg and attached to the foot wedge, placing the knee in approximately 90 degrees of flexion. In this position, participants were unable to move through a range of motion when contracting, thereby ensuring the motion was isometric. Participants in the control group were asked to continue with their regular training and return in three weeks for a post-test.

After three weeks the participants returned to the laboratory for a post-test. The post-test included measuring height, weight, plantarflexor torque, and spinal control identically to the pre-test.

3.3 Data Reduction

Torque and EMG data were both sampled at 2,000 Hz. Torque data were recorded as voltage and analyzed using custom-built LabVIEW software (National Instruments, Austin, TX). The raw torque was processed using a fourth-order 10 Hz low-pass Butterworth filter. The voltage was then converted to Newton meters (Nm) using a scaling equation. Using the same software, rate of torque development was calculated by fitting a line of best fit from torque onset (defined as the point when the torque signal was greater than 2.5% of maximum torque) to 100 ms after onset. RTD was then normalized using the participants' body mass ($Nm/s \bullet [kg^{-1}]$). The trial with the greatest RTD was used for analysis.

The H-reflex, including unconditioned and conditioned H-reflexes used for recurrent inhibition, and M-wave data were also analyzed using custom-built LabVIEW software. All H-reflexes and M-waves were calculated by measuring the peak-to-peak amplitude of each evoked potential. Individual participant latencies from the stimulus pulse were visually identified in Acqknowledge 3.9.1.6 from trials that displayed easily recognizable H-reflexes and M-waves.

The latencies were then entered into the LabVIEW software in order for the software to measure the peak-to-peak of the H-reflex and M-wave during each trial during those latencies. The maximal H-reflex (H_{max}) and the maximal M-wave (M_{max}) were used for analysis. Two participants' H_{max}:M_{max} data was excluded from analysis of the H_{max}:M_{max} dependent variable due to ratios being greater than 1.

The same procedures were used to determine the peak-to-peak amplitudes of the conditioned H-reflex to determine recurrent inhibition. Percent recurrent inhibition was calculated by dividing the peak-to-peak amplitude of the conditioned H-reflex by the peak-to-peak amplitude of the unconditioned H-reflex and then subtracting from one, i.e., [1-(conditioned reflex/unconditioned reflex)]. Conditioned and unconditioned peak-to-peak amplitudes were analyzed using the average of the three respective trials. In situations where stable unconditioned H-reflexes could not be elicited at either 10% or 30% of M_{max}, the participants' data were omitted. Overall, five participants data were omitted from recurrent inhibition analysis at 10% of $M_{max,}$ and nine participants' data were omitted from recurrent inhibition analysis at 30% of M_{max}.

The three V-wave trials were not processed in LabVIEW. Instead, they were identified visually in Acqknowledge 3.9.1.6. In each trial, the peak-to-peak amplitude of the V-wave was divided by the peak-to-peak amplitude of the M-wave in the same trial. Each of these V to Mmax ratios (V:Mmax) were averaged across all three trials to calculate average V: M_{max} used for analysis.

3.4 Statistical Analyses

A 2 (Group) x 2 (Time) mixed model ANOVA was performed for each dependent variable (i.e., RTD 0-100 ms, H_{max}:M_{max}, percent recurrent inhibition at 10% M_{max} and at 30% M_{max}, and V:M_{max}). The independent variables were time (pre-test and post-test) and group (control and training). A priori alpha levels were set at 0.05. All statistical analyses were performed using RStudio (Version 0.98.1062).

Chapter 4

Results

There was not a significant interaction for RTD 0-100 ms ($p = 0.615$) nor were the main effects for group ($p = 0.388$) or time ($p = 0.377$) significant. There was also not a significant interaction for H_{max}:M_{max} ($p = 0.505$) or significant main effects for group ($p = 0.690$) and time ($p = 0.926$). Recurrent inhibition at 10% of M_{max} did not have a significant interaction ($p = 0.498$) or main effects for group ($p = 0.599$) and time ($p = 0.592$). Interaction effects for recurrent inhibition at 30% of M_{max} were also not significant ($p = 0.998$) and neither were the main effects for group ($p = 0.363$) and time ($p = 0.685$). Lastly, V:M_{max} did not have a significant interaction ($p = 0.829$) and main effects for group ($p = 0.527$) and time ($p = 0.232$) were not significant.

Dependent Variable	Group	Pre-Test Mean ± SD	Post-Test Mean ± SD
RTD (Nm/s•[kg^{-1}])	Control	3.591 ± 1.469	4.278 ± 1.882
	Training	4.268 ± 1.614	4.457 ± 1.808
H_{max}:M_{max}	Control	0.758 ± 0.207	0.719 ± 0.142
	Training	0.733 ± 0.224	0.703 ± 0.107
Recurrent Inhibition at 10% M_{max} (Percent Inhibition)	Control	-1.004 ± 1.837	-0.940 ± 0.908
	Training	-0.914 ± 1.669	-1.648 ± 2.476
Recurrent Inhibition at 30% M_{max} (Percent Inhibition)	Control	0.059 ± 0.702	-0.058 ± 0.822
	Training	-0.207 ± 0.886	-0.326 ± 0.715
V:M_{max}	Control	0.211 ± 0.166	0.265 ± 0.211
	Training	0.234 ± 0.146	0.313 ± 0.339

Table 2: Mean and standard deviations of dependent variables for groups (control and training) and time (pre-test and post-test)

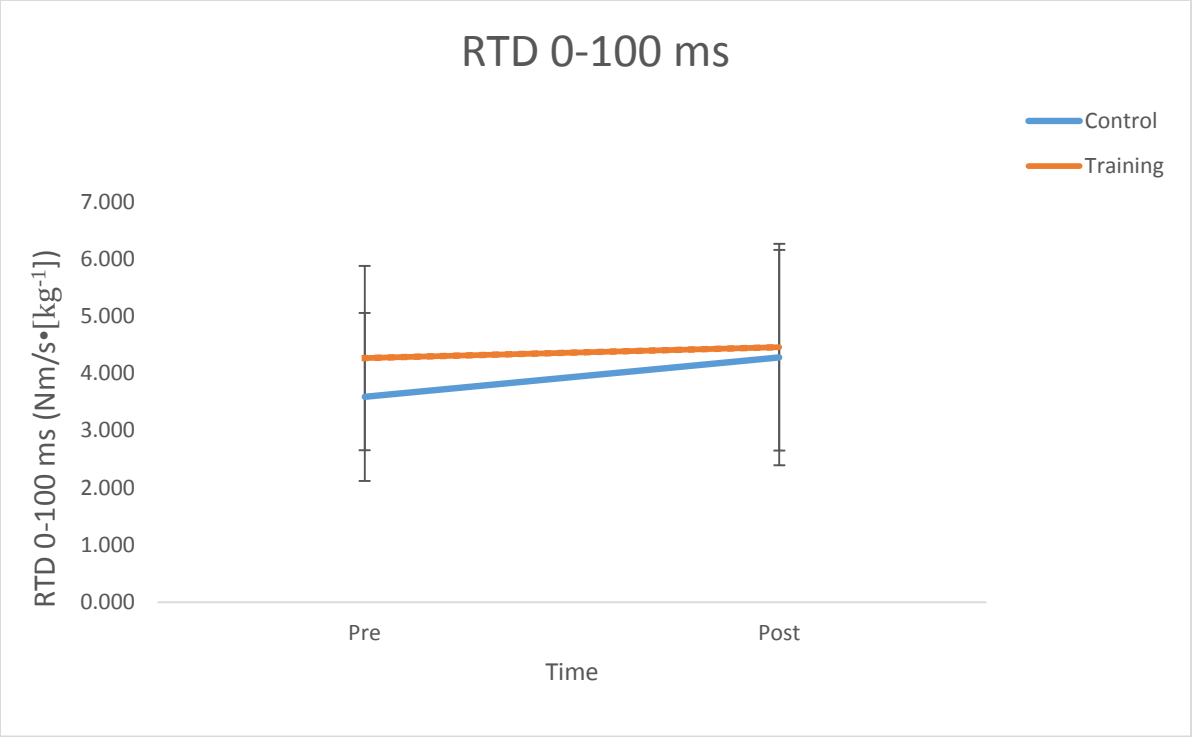

Figure 3: The above graph illustrates the average RTD from 0-100 ms of the control and training group at the pre- and post-test.

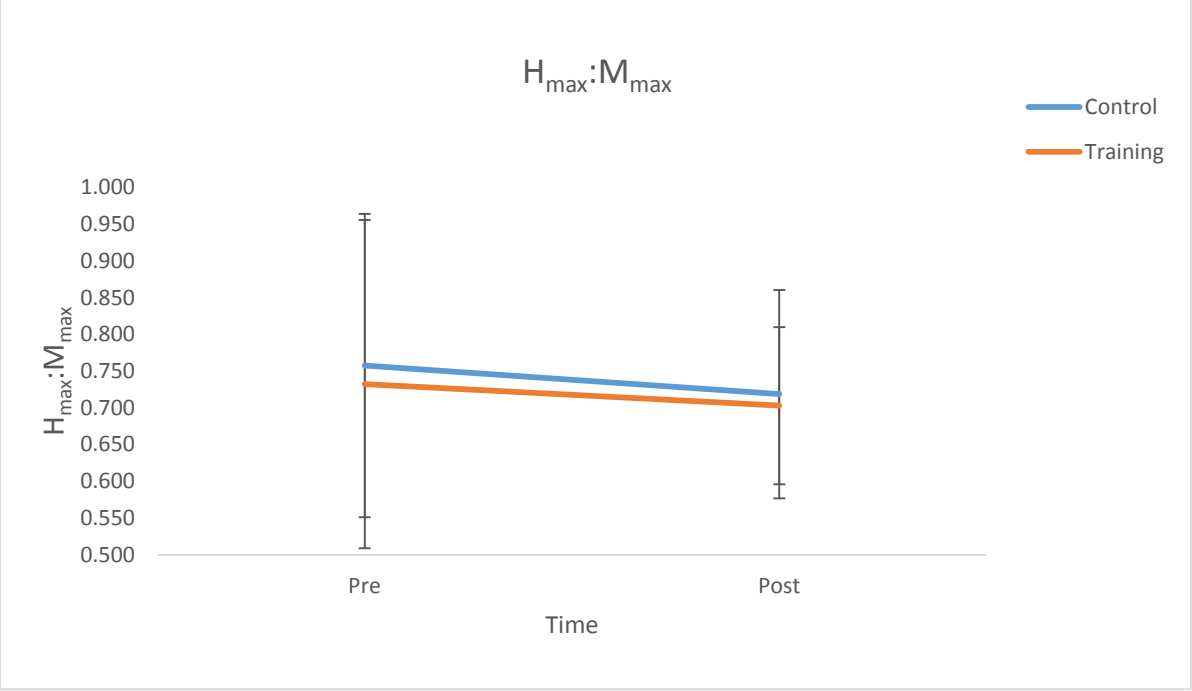

Figure 4: The above graph illustrates the H_{max}: M_{max} values of the control and training group as measured at the pre-and post-tests.

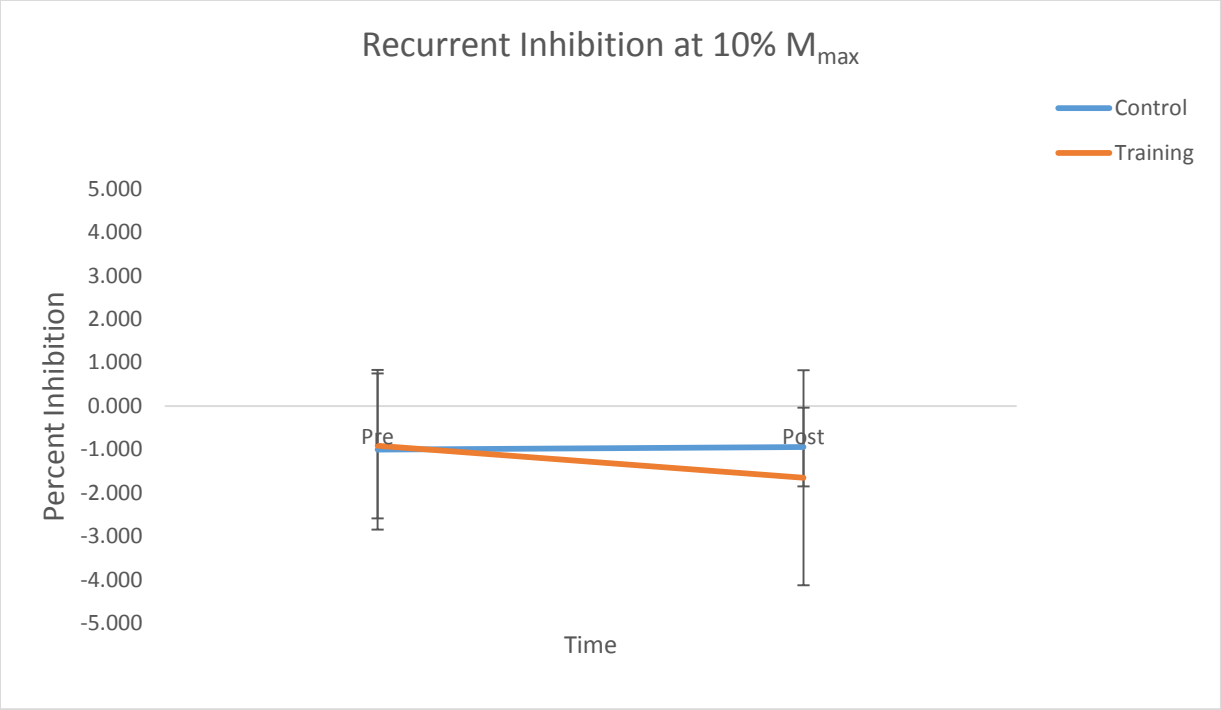

Figure 5: The above graph illustrates recurrent inhibition at 10% of M_{max} in the control and training group as measured during the pre- and post-tests.

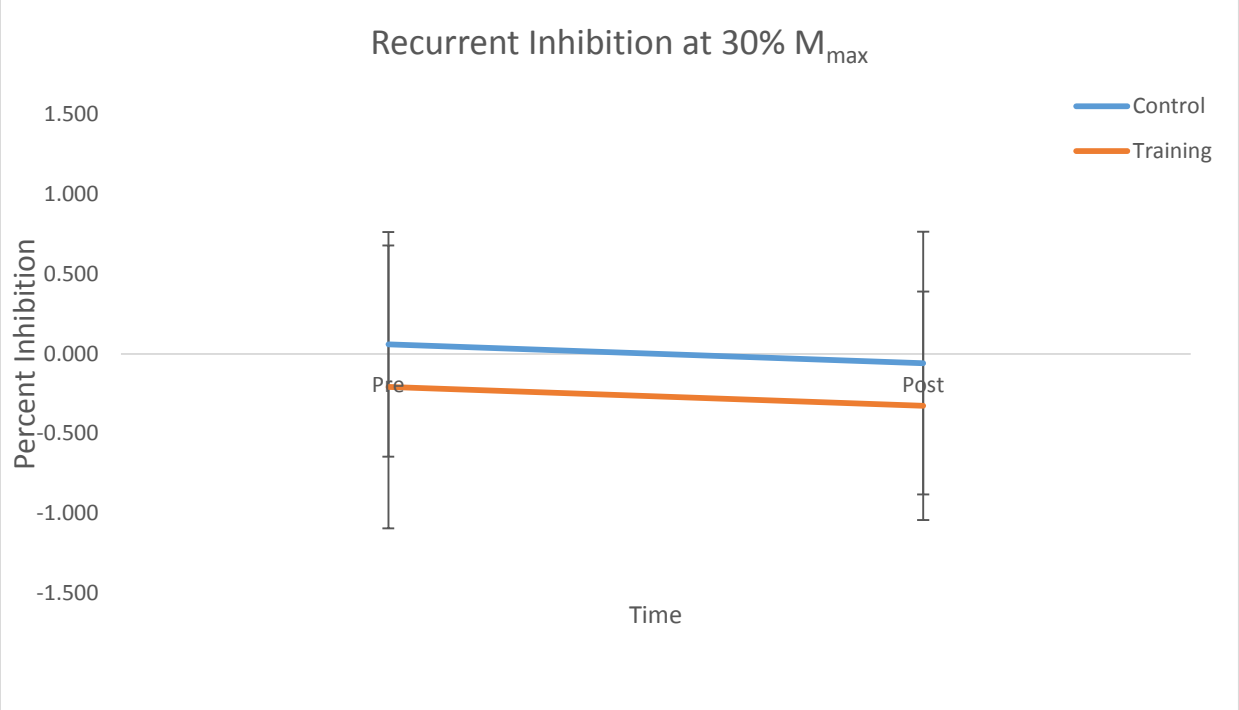

Figure 6: The above graph illustrates recurrent inhibition at 30% of M_{max} in the control and training group as measured during the pre- and post-tests.

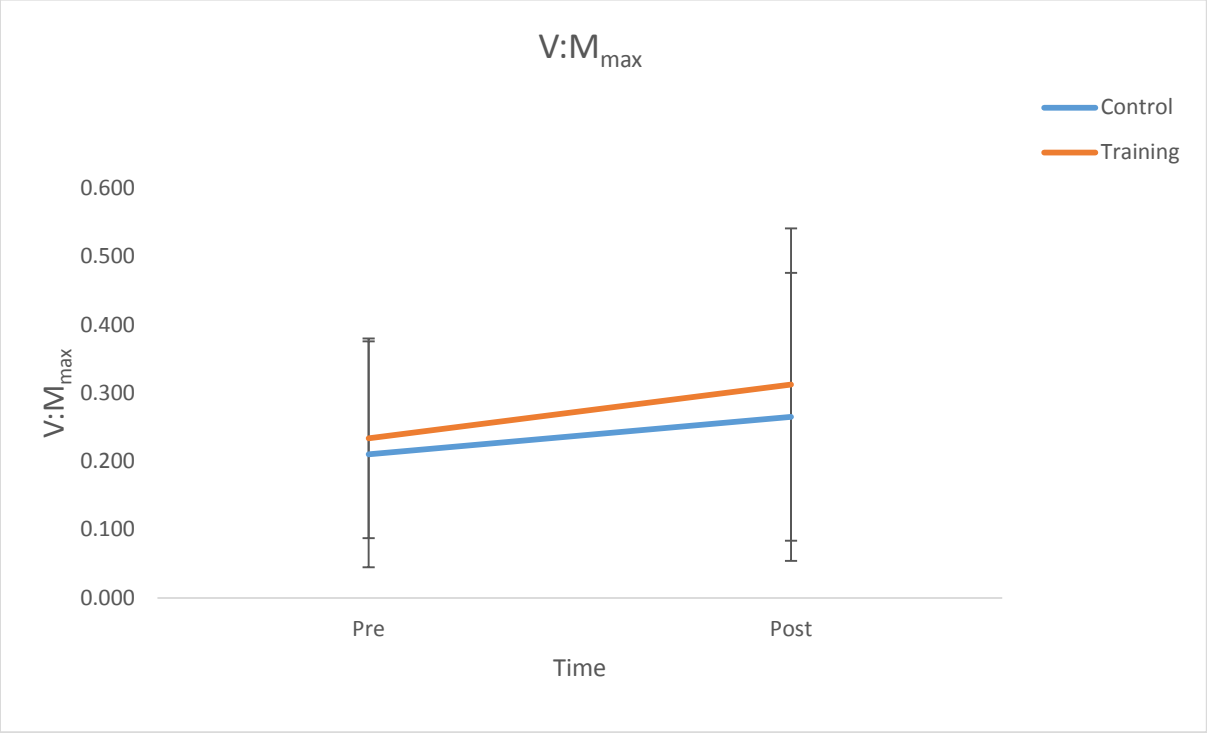

Figure 7: The The above graph illustrates V:M_{max} in the control and training group as measured during the pre- and post-tests.

Chapter 5

Discussion

The purpose of this study was to develop greater understanding of the connection between RTD and spinal motor control mechanisms. Specifically, changes in H-reflexes, V-waves, and recurrent inhibition tested during an explosive contraction were investigated following a three week explosive strength training program of the plantarflexors. Training did not significantly change these dependent variables.

The lack of significant changes in RTD following explosive strength training was unexpected and in contrast to those seen by Holtermann et al. (2007). Holtermann et al. observed a 28.4% increase in RTD during 0-300 ms after contraction onset (2007). The current study used a training regimen consisting of the same time, number of sessions, repetitions, sets, and rest periods.

One possible critical difference between Holtermann et al.'s and the current study's training protocol is the participants were able to instantaneously observe the torque they produced during each training contraction. This method of providing biofeedback was not attempted in the current study. This was due to concern participants training on the dynamometer would exhibit strength gains attributable to a learning effect rather than neuromuscular adaptations. In addition, the time needed for dynamometer training use and set-up was impractical. Secondly, differences in data analysis could contribute to the discrepancies between RTD results observed in the current and Holtermann et al.'s study. The current study investigated RTD from 0-100 ms following torque onset rather than the 0-300 ms analyzed by Holtermann et al (2007). Therefore, it is possible the training regimen resulted in changes during later time

periods not analyzed in the current study. Holtermann et al. also normalized RTD data using MVC while the current study used body mass.

Finally, it is possible differences in knee joint angle between training and testing positions influenced the lack of RTD changes. Previous research indicates gains from isometric training is related to the specific joint angle at which training occurs (Kitai & Sale, 1989; Weir, Housh, Weir, & Johnson, 1995). The difference between training and testing knee joint angle was approximately 20 degrees. However, the ankle joint was both trained and tested at approximately 10 degrees of plantarflexion. Because the soleus muscle does not cross the knee joint, it seems unlikely that lack of significant RTD increase can be attributed to knee angle differences.

Although it was hypothesized that changes in spinal motor control mechanisms would occur, it is possible the lack of changes were related to the training program's failure in to produce significant adaptations related to RTD. There were also several methodological difficulties. Firstly, while a unique aspect of the current study was testing spinal motor control measures during explosive contractions, it is unknown if this would increase variability of the H-reflex response or how sensitive the H-reflex would be to even slight changes in the voluntary contraction. Because the H-reflex reflects net motor neuron pool excitability, it is subject to the impulses of extensive neural networks throughout the peripheral nervous system. For example, even slight postural differences can contribute to changes in H-reflex measures (Zehr, 2002). From a functional perspective, the innate versatility of the nervous system is desirable because it also allows individuals to adapt to dynamic tasks and environments. Therefore, the measurement of H-reflexes during rapid contractions is required to adequately investigate a versatile system during a specific task. However, it also creates difficulty in measuring spinal motor control

mechanisms as observed in the current study by the high variability in recurrent inhibition measures.

In some cases, it took several attempts to find three H-reflexes within the specified 10% and 30% M_{max} window needed to test recurrent inhibition. This was particularly true if participants had a relatively small M_{max} value. When the M_{max} value was small, it was more difficult to observe H-reflex values at 10% of M_{max}, possibly because the H-reflex was so close to threshold. In addition, a small M_{max} value decreased the range at which H-reflexes within the desired window could be found and, therefore, it was not always possible to identify a stable H-reflex at 10% and 30% of M_{max}. In those participants, recurrent inhibition was not tested. It is also possible that the relatively lengthy time needed to find the H-reflex values within the desired window could result in compression of the sciatic nerve from prolonged sitting in some participants. Attempts were made to limit this by asking participants if their foot or toes were "numb" and, if so, giving them a break from having their foot strapped against the footplate.

In agreement with several previous studies, there was no significant change in H_{max}:M_{max} following training (Aagaard et al., 2002; Del Balso & Cafarelli, 2007; Gruber et al., 2007; Vila-Chã et al., 2012). However, because increases in MVC or RTD after training were observed in previous research and not in the current study, caution should be taken in comparing the results. It is possible that changes in spinal motor control mechanisms could still occur with neuromuscular adaptations but were not present in the current study due to lack of increase in RTD. Further research is necessary to determine if the lack of H_{max}:M_{max} during explosive contractions can be associated with adaptations that occur with strength training.

Previous studies also observed significant increases in V-wave amplitude following training (Aagaard et al., 2002; Del Balso & Cafarelli, 2007; Vila-Chã et al., 2012) that was not

matched in the current study. The previous researchers attribute the increase in supraspinal neural drive as an indication of adaptations that occur after strength training. However, because there were no significant changes in RTD following the current study, it may be unwise to directly compare.

This study is also distinctive in its investigation of recurrent inhibition during a rapid contraction. Unexpectedly, the majority of recurrent inhibition values were negative, representing an excitatory, rather than inhibitory, mechanism. This was particularly unexpected because the maximal conditioned H-reflex response should only be able to measure recurrent inhibition of the motor units activated by the unconditioned H-reflex. This is due to the orthodromic and antidromic collision of action potentials during the conditioned H-reflex test stimulus (Bussel & Pierrot-Deseilligny, 1977). However, it is theorized that supraspinal neural drive during the contraction could provide additional clearing in the pathway for a greater signal to reach the muscle (Hultborn & Pierrot-Deseilligny, 1979; Knikou, 2008). Therefore, in their investigation of recurrent inhibition during isotonic and phasic contractions, Hultborn & Peirrot-Deseilligny subtracted the V-wave from the conditioned H-reflex (1979). However, because both the unconditioned and conditioned H-reflex response were obtained during 20% MVC of an explosive contraction, it seems both responses would be subject to similar supraspinal influences.

In the future, the impact of V-waves on the conditioned H-reflex response should be investigated in order to ascertain the conditioned H-reflex response is not disproportionately inflated. While a potential factor, the current results of less inhibition and possible facilitation of the H-reflex during recurrent inhibition during explosive contractions is in agreement with Hultborn & Peirrot-Deseilligny's findings during strong tonic contractions. They expected greater recurrent inhibition due to an increased number of activated Renshaw cells through

voluntarily contraction. Instead, they observed decreased recurrent inhibition (Hultborn & Pierrot-Deseilligny, 1979). While recurrent inhibition was not compared to at rest values during this study, there was an observed trend toward facilitation of the H-reflex during explosive contractions as evidenced by a conditioned H-reflex greater than the unconditioned in all groups, times, and intensities except the pre-tested controls during 30% of M_{max}.

Interestingly, this trend toward facilitation of the H-reflex during recurrent inhibition is different than the findings of a previous study. Earles et al. found recurrent inhibition at both 10% and 30% of M_{max} during standing and significantly greater inhibition at 30% of M_{max} as compared to 10% of M_{max} (2002). This supports the possibility that recurrent inhibition may be dependent on the specific demands placed on the motor system. For example, the amount of recurrent inhibition may change depending on whether an individual is quietly standing or explosively contracting.

In conclusion, more research is needed to determine if changes in H-reflexes, V-waves, and recurrent inhibition tested during explosive contractions of the plantarflexors accompany adaptations in RTD after explosive strength training. In this study, there was no significant change in the observed spinal motor control mechanisms, but there were also no significant change in RTD after training. Further research should focus on continued longitudinal studies investigating possible spinal motor control mechanisms following explosive strength training.

Chapter 6

Conclusion

The ability to generate muscular force rapidly is important for responding to dynamic and changing surroundings. Therefore, measuring the rate of torque development represents a more functional measure than maximal strength alone. It is well established that rate of torque development is influenced by neuromuscular mechanisms (Aagaard et al., 2002; Van Cutsem et al., 1998). However, due to the complexity of the nervous system, the mechanisms underlying neural adaptations are not completely understood. Specifically, there are knowledge gaps concerning the role of spinal motor control mechanisms as related to RTD. In an attempt to fill those gaps, the current training study investigated rate of torque development, motor neuron pool excitability, recurrent inhibition, and supraspinal neural drive following explosive strength training.

Following three weeks of explosive strength training of the plantarflexors, there were no significant changes in spinal motor control mechanisms, but there was also no significant change in RTD. Therefore, it is still unknown if spinal motor control mechanisms are related to changes in RTD. The methodologies presented, particularly related to testing spinal motor control mechanisms during explosive contractions, provide a unique contribution to the current literature. This study supports the notion that the nervous system is inherently complex and variable, potentially contributing to individuals' ability to function in dynamic environments.

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APPENDICIES

Appendix A: Institutional Review Board Approval

Institutional Review Board
Office of Research Integrity | Oregon State University
B308 Kerr Administration Building, Corvallis, OR 97331-2140
Telephone (541) 737-8008
irb@oregonstate.edu | http://research.oregonstate.edu/irb

APPROVAL NOTICE

Date of Notification	01/16/2015		
Study ID	6584		
Study Title	Spinal Motor Control Adaptations to Explosive Strength Training		
Principal Investigator	Sam Johnson		
Study Team Members	Abegail Carpenter, Cody Farnes, Mark Hoffman, Marc Norcross, Clara Stone, Maria Torres, Kelsey Uno		
Submission Type	Project Revision	Date Approved	01/16/2015
Level	Expedited	Category(ies)	4
Number of Participants	40 *Do not exceed this number without prior IRB approval*		
Waiver(s)	None		
Risk Level for Children	N/A		
Funding Source	None	PI on Grant or Contract	N/A
Proposal #	N/A	Cayuse #	N/A

The above referenced study was reviewed and approved by the OSU Institutional Review Board (IRB).

EXPIRATION DATE: 01/07/2016

Annual continuing review applications are due at least 30 days prior to expiration date

Documents included in this review:

- ☒ Protocol
- ☒ Consent forms
- ☐ Assent forms
- ☐ Alternative consent
- ☐ Letters of support
- ☐ Recruiting tools
- ☐ Test instruments
- ☐ Attachment A: Radiation
- ☐ Alternative assent
- ☒ Project revision(s)
- ☐ External IRB approvals
- ☐ Translated documents
- ☐ Attachment B: Human materials
- ☐ Grant/contract
- ☐ Other:

Comments: Added study members.

Principal Investigator responsibilities for fulfilling the requirements of approval:

- All study team members should be kept informed of the status of the research.
- Any changes to the research must be submitted to the IRB for review and approval prior to the activation of the changes. **This includes, but is not limited to, increasing the number of subjects to be enrolled.**
- Reports of unanticipated problems involving risks to participants or others must be submitted to the IRB within three calendar days.
- Only consent forms with a valid approval stamp may be presented to participants.
- Submit a continuing review application or final report to the IRB for review at least four weeks prior to the expiration date. Failure to submit a continuing review application prior to the expiration date will result in termination of the research, discontinuation of enrolled participants, and the submission of a new application to the IRB.

Appendix B: Screening Questionnaire

Screening Questionnaire

Participant Code: _____ Date _____

Age: _____ Sex: _____

Do you currently have any injuries or illnesses that limit your ability to perform your regular physical activity?	Yes	No
Do you currently have any leg or low back injuries?	Yes	No
Have you had an injury to your low back, hip, knee or ankle that required surgery?	Yes	No
Do you perform on average 150 minutes of physical activity per week?	Yes	No
Do you run less than 20 miles per a week?	Yes	No
Do you have any neurological disorders?	Yes	No
Will you agree to not begin any new hopping or lower leg strength training programs over the course of this study?	Yes	No

Appendix C: Participant Information

Participant Code_____ Date_____

Height_____ Weight_____

Dominant Leg Determination

Leg used to catch oneself from a push	Right	Left
Leg used to step up onto a box	Right	Left
Leg used to kick a soccer ball	Right	Left

Participant's Dominant Leg is _____

Appendix D: Training Documentation Sheet

Isometric Explosive Strength Training of the Plantarflexors

Participant Code_____ Date _____ Session # _____

Training Leg _____

Remember: Shoe off, strap down tightly

Each Set:

- Instruct to push "as hard and fast as possible"
- 10 contractions holding each for 4 seconds. 10 sec rest between contractions
- 3 minutes rest between sets

Set 1____ Set 2_____ Set 3 _____ Set 4 _____ Set 5 _____

Isometric Explosive Strength Training of the Plantarflexors

Participant Code_____ Date _____ Session # _____

Remember: Shoe off, Strap down tightly

Each Set:

- Instruct to push "as hard and fast as possible"
- 10 contractions holding each for 4 seconds. 10 sec rest between contractions
- 3 minutes rest between sets

Set 1____ Set 2_____ Set 3_____ Set 4_____ Set 5_____

Appendix E: ANOVA Tables

Dependent Variable ANOVA Tables

RTD 0-100 ms	DF	Sum Sq	Mean Sq	F Value	P Value
Time (Pre- & Post-test)	1	2.301	2.30125	0.7955	0.3773
Group (Control &Training)	1	2.197	2.19735	0.7596	0.3882
Time & Group	1	0.743	0.74252	0.2567	0.6149
Residuals	44	127.287	2.89289		

H_{max}:M_{max}	DF	Sum Sq	Mean Sq	F Value	P Value
Time (Pre- & Post-test)	1	0.00027	0.0002708	0.0087	0.926
Group (Control &Training)	1	0.005	0.0050021	0.1611	0.6901
Time & Group	1	0.01401	0.0140083	0.4512	0.5053
Residuals	44	1.36596	0.0310446		

Recurrent Inhibition at 10% of M_{max}	DF	Sum Sq	Mean Sq	F Value	P Value
Time (Pre- & Post-test)	1	0.940	0.9397	0.2928	0.5920
Group (Control &Training)	1	0.905	0.9053	0.2820	0.5988
Time & Group	1	1.507	1.5070	0.4695	0.4979
Residuals	34	109.138	3.2099		

Recurrent Inhibition at 30% of M_{max}	DF	Sum Sq	Mean Sq	F Value	P Value
Time (Pre- & Post-test)	1	0.1043	0.10427	0.1686	0.6847
Group (Control &Training)	1	0.5309	0.53091	0.8584	0.3627
Time & Group	1	0.0000	0.00000	0.0000	0.9978
Residuals	26	16.0799	0.61846		

V:M_{max}	DF	Sum Sq	Mean Sq	F Value	P Value
Time (Pre- & Post-test)	1	0.05360	0.053600	1.4709	0.2317
Group (Control &Training)	1	0.01484	0.014840	0.4073	0.5267
Time & Group	1	0.00173	0.001728	0.0474	0.8286
Residuals	44	1.603370	0.036440		

Appendix F: Dependent Variable Data

Subject	Pre/Post	Group	Sex	Height (cm)	Body Mass (kg)	RTD 0-100 ms (Nm/s•[kg^{-1}])	Peak Torque (Nm/s•[kg^{-1}])	H_{max}:M_{max}	Recurrent Inhibition at 10% M_{max} (% Inhibition)	Recurrent Inhibition at 30% M_{max} (% Inhibition)	V:M_{max}
1	Pre	Control	F	161.9	54.2	135.746	0.9710	0.457	-217.846		0.080
1	Post	Control	F	161.9	52.8	87.357	0.5344	0.597	-77.577		0.106
2	Pre	Control	M	177.8	75.5	233.832	1.0892	0.949		-51.845	0.554
2	Post	Control	M	177.8	75.5	435.99	1.6077	0.866		-138.075	0.491
3	Pre	Training	F	176.5	64.2	319.181	0.9721	0.600	1.842		0.244
3	Post	Training	F	176.5	63.1	222.111	1.0520	0.734	87.170		0.281
6	Pre	Training	M	174.0	53.8	265.834	1.0652	0.924			0.252
6	Post	Training	M	174.0	54.4	182.612	0.8138	0.876			0.238
7	Pre	Control	M	185.4	78.2	306.45	1.1081	1.025		11.040	0.268
7	Post	Control	M	185.4	77.9	416.992	1.5603	0.603		28.075	0.205
8	Pre	Training	M	181.6	100.2	557.03	1.2388	0.574	92.918	89.843	0.482
8	Post	Training	M	181.6	98.9	684.873	1.6571	0.609	-56.741	24.875	0.544
9	Pre	Training	M	181.0	98.7	258.44	0.4128	0.112	85.644		0.026
9	Post	Training	M	181.0	95.1	505.448	1.0008	0.518	95.174		0.149
10	Pre	Control	M	181.6	77.5	189.369	0.6663	0.687	88.908	74.142	0.076
10	Post	Control	M	181.6	76.6	243.994	0.6546	0.683	-16.531	97.448	0.006
11	Pre	Control	M	175.3	63.8	205.262	1.0284	0.915	79.035		0.029
11	Post	Control	M	175.3	65.7	255.289	1.3611	0.720	-73.383		0.112
12	Pre	Control	M	174.0	82.7	514.043	1.1215	0.721	-79.379	-41.451	0.192
12	Post	Control	M	174.0	82.5	541.126	1.5080	0.636	-226.676	-57.854	0.517
13	Pre	Training	F	179.1	74.3	221.814	0.6609	0.694		-100.503	0.331
13	Post	Training	F	179.1	74.5	471.288	1.2457	0.693		-19.095	0.216

14	Pre	Control	F	168.3	57.7	256.954	0.9853	0.722	-220.137	-91.849	0.398
14	Post	Control	F	168.3	58.7	420.94	1.6785	0.737	-151.118	-21.771	0.408
15	Pre	Control	M	172.7	75.5	169.542	0.5600	0.547	61.233	97.212	0.034
15	Post	Control	M	172.7	75.9	222.939	0.6697	0.619	33.350	81.642	0.009
16	Pre	Training	F	157.5	55.6	216.642	0.7455	0.845	-396.648	-27.414	0.187
16	Post	Training	F	157.5	55.6	149.821	0.6367	0.864	-216.948	-30.969	0.245
17	Pre	Control	F	168.9	87.7	205.15	0.3773	1.106	-297.046		0.325
17	Post	Control	F	168.9	87.3	269.246	0.6097	1.090	-200.836		0.399
18	Pre	Training	M	191.1	86.2	320.87	0.6767	0.778	-175.271	-37.051	0.269
18	Post	Training	M	191.1	88.4	236.06	0.5747	0.654	-589.079	34.308	0.103
19	Pre	Training	M	179.1	81.6	339.2	0.9991	0.735	-146.468	-103.669	0.333
19	Post	Training	M	179.1	81.2	504.792	1.4007	0.671	-421.355	-101.871	0.491
21	Pre	Control	F	170.2	66.5	185.699	1.1245	0.505	-33.019	43.879	0.299
21	Post	Control	F	170.2	66.4	407.199	1.7927	0.725	-169.586	-30.264	0.584
22	Pre	Training	M	181.6	84.5	361.18	0.7568	0.679	-33.180	87.702	0.099
22	Post	Training	M	181.6	84.8	447.761	1.1989	0.786	19.777	62.482	0.156
23	Pre	Control	F	181.0	76.7	251.118	0.7374	0.790	-438.593		0.054
23	Post	Control	F	181.0	76	124.249	0.7465	0.750	-78.171		0.044
24	Pre	Control	F	161.3	56.3	370.842	1.7669	0.670	53.058		0.218
24	Post	Control	F	161.3	55	218.573	0.9091	0.601	20.074		0.304
25	Pre	Training	F	154.9	57.8	454.162	2.1858	0.986	-266.884	-129.065	0.428
25	Post	Training	F	154.9	58.2	365.353	1.9069	0.784	-377.618	-127.791	0.869
26	Pre	Training	F	180.3	78.9	112.942	0.2760	0.680	15.387		0.039
26	Post	Training	F	180.3	79	113.203	0.3303	0.798	-23.953		0.069
27	Pre	Training	F	166.4	73.9	353.857	0.7495	0.832		54.490	0.115
27	Post	Training	F	166.4	74.1	257.208	0.5598	0.805		-102.546	0.390