Spinal and Supraspinal Motor Control Predictors of Rate of Torque Development

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Abstract

During explosive movements and potentially injurious situations the ability to rapidly generate torque is critical. Previous research has suggested different phases of rate of torque development (RTD) are differentiately controlled. However, the extent to which supraspinal and spinal mechanisms predict RTD at different time intervals is unknown. RTD of the plantarflexors across various phases of contraction (i.e., 0-25ms, 0-50ms, 0-100ms, 0-150ms, 0-200ms, and 0-250ms) was measured in 37 participants. The following predictor variables were also measured: (a) gain of the resting soleus H-reflex recruitment curve, (b) gain of the resting homonymous post-activation depression recruitment curve, (c) gain of the GABAergic pre-synaptic inhibition recruitment curve, (d) the level of post-synaptic recurrent inhibition at rest, (e) level of supraspinal drive assessed by measuring V waves, and (f) the gain of the resting soleus M Wave. Stepwise regression analyses were used to determine which variables significantly predicted allometrically scaled RTD. The analyses indicated that supraspinal drive was the dominant predictor of RTD across all phases. Additionally, recurrent inhibition predicted RTD in all of the time intervals except 0-150 ms. These results demonstrate the importance of supraspinal drive and recurrent inhibition to RTD.

Keywords:

V wave, Rate of force development, Neuromuscular control

Introduction

The ability to rapidly activate the neuromuscular system to produce torque is important during both explosive movements and potentially injurious situations when the time to stabilize a joint in response to a perturbation is limited (Aagaard, 2003). Rate of torque development (RTD), defined as the rate of the rise of the torque-time curve, is frequently used to characterize rapid torque production. Despite its functional importance, the underlying physiological mechanisms contributing to RTD are not fully known.

Previous researchers have reported RTD is influenced by a variety of factors including intrinsic muscle contractile properties (Andersen and Aagaard, 2006; Andersen et al., 2010), muscle-tendon stiffness (Bojsen-Møller et al., 2005), maximal muscle strength (Andersen and Aagaard, 2006; Andersen et al., 2010; Tillin et al., 2012), and neural drive (Van Cutsem et al., 1998; Aagaard et al., 2002a; Tillin et al., 2012). Interestingly, it appears these mechanisms differentially contribute depending on the time interval of RTD that is examined (Bojsen-Møller et al., 2005; Andersen and Aagaard, 2006; Andersen et al., 2010; Tillin et al., 2012). In fact, those studies overwhelmingly suggest that later phase RTD, is primarily a factor of maximal muscle strength (Andersen and Aagaard, 2006; Andersen et al., 2010; Tillin et al., 2012) and to a lesser extent muscle-tendon stiffness (Bojsen-Møller et al., 2005). Whereas, earlier phase RTD appears to be controlled by different mechanisms than RTD at later time periods. For instance, it has been reported that early-phase RTD was primarily a factor of twitch torques (Andersen and Aagaard, 2006) and muscle fiber type (Andersen et al., 2010). More recently it was reported that the changes in early-phase RTD following a 4-week training for explosive torque production was primarily due to enhanced agonist drive, defined as the amount of voluntary torque produced as a proportion of evoked torque produced (Tillin et al., 2012). While those are informative findings, it does not explain where in the central nervous system those changes are occurring. An examination of the

spinal and supraspinal circuitry may provide novel insight into the production of RTD due to the modulatory effects this circuitry has on the activation of the alpha motor neurons (Wolpaw, 2001).

Several lines of evidence suggest contribution of spinal and supraspinal mechanisms to RTD. One study reported changes in H-reflex amplitude [tested at 20% of maximal voluntary contraction (MVC)] were correlated with greater RTD, but not maximal torque following a 3-week resistance training program (Holtermann et al., 2007). However, they examined RTD at only the 0-300 ms time interval. Additionally, they tested H-reflexes in isolation which only provides a net estimate of motor neuron pool output and cannot fully account for other spinal motor control mechanisms that modulate the motor neuron pool output, such as pre- and post-synaptic inhibition (Zehr, 2002). Another study investigated the gain of the H-reflex recruitment curve (H_{slope}/M_{slope}) at both rest and 10% of MVC before and after a 4-week training that increased RTD (Del Balso and Cafarelli, 2007). They found no change in the H-reflex recruitment curve, but they did report an increase in supraspinal neural drive as measured with V waves (Del Balso and Cafarelli, 2007).

Based on the lack of understanding of how spinal control mechanisms contribute to RTD, especially during different time intervals, we measured a unique collection of variables known to modulate motor neuron pool output to determine which of those predict RTD. Specifically, the variables were (a) gain of the resting soleus H-reflex recruitment curve, (b) gain of the resting homonymous post-activation depression (HPAD) recruitment curve, (c) gamma-aminobutyric-acid (GABA)ergicpre-synaptic inhibition recruitment curve, (d) the level of post-synaptic recurrent inhibition at rest, (e) level of supraspinal drive (V waves), and (f) the gain of the resting soleus M Wave.

Methods

Participants

Forty-one participants (20 females and 21 males) were recruited to participate in this study. Participants ranged between the ages of 18 and 35 and were required to be physically active a minimum of 30 minutes three times a week. Participants were also free from: a) current injury to the back, upper extremity, or lower extremity, b) lower extremity injury in the past six months, and c) history of lower extremity ligament surgery. To control for potential hormonal influences across the menstrual cycle, females were tested between days 1-3 of their menstrual cycle. Data from four participants were unable to be used due to an inability to complete the testing protocol resulting in total sample of 37 participants (18 females and 19 males) in the study (23.8 ± 3.8 yrs, 70.18 ± 13.65 kg, 1.72 ± 0.08 m). Additionally, recurrent inhibition could not be elicited on two participants but those participants remaining data were included. Further, one participant had an HPAD value that was considered an outlier (>3 box lengths from the middle 50% of the data) and one participant had a GABAergic pre-synaptic value that was considered an outlier (>3 box lengths from the middle 50% of the data). In each case, the specific values that were considered outliers were eliminated from the analyses, but all of their remaining data were included in the final model.

Procedures

Participants read and signed an informed consent approved by the University's Institutional Review Board and completed a health history and training history questionnaire to determine eligibility to participate in the study. Height was obtained using a wall mounted stadiometer and weight was determined by a standard scale. The dominant leg was used for all testing and was determined by which leg the participant used for the majority of the following tests: a) kicking a ball, b) recovering from a balance perturbation, and c) stepping up on a 10 inch box (Hoffman et al., 1998).

Dynamometer Positioning

Participants were seated on the chair of the Biodex System 3 dynamometer (Biodex Medical Systems Inc, Shirley, NY) in a semi-recumbent position. The knee was flexed to 60 degrees and ankle positioned in anatomical position (90 degrees of plantar-dorsiflexion and 0 degrees of inversion-eversion). The foot was secured to the ankle attachment foot plate to prevent any movement of the foot from the plate. The non-test leg was in a comfortable, relaxed position with the foot supported. This positioning was used for all subsequent testing.

Electromyography Preparation

The soleus, tibialis anterior, and lateral mallelous were prepared for application of lubricated surface electromyography (EMG) electrodes (Ag/AgCl). The EMG electrodes over the muscle were placed longitudinally with an interelectrode distance of 3 cm for each respective muscle and a single reference electrode was placed on the lateral malleolus. The EMG data were sampled at 2000 Hz and stored on a personal computer equipped with a Biopac MP100 data collection system (Biopac Systems Inc, Goletta, CA).

Stimulating Electrode Placement

To elicit the soleus H-reflex, a stimulating electrode (2 cm^2) was placed over the tibial nerve in the popliteal fossa for current delivery. A dispersal pad (3 cm^2) was placed superior to the patella on the distal thigh. To elicit GABAergic pre-synaptic inhibition of the soleus, a stimulating electrode (1 cm^2) was placed over the common peroneal nerve distal the fibular head for current delivery and a dispersal pad (3 cm^2) was placed just anterior to the fibular head. Care was taken in the placement of the electrode over the peroneal nerve to limit stimulation of the peroneal group.

H-Reflexes

H-reflex and M wave recruitment curves for the soleus were measured at rest by stimulating the tibial nerve in the popliteal fossa. Stimulation was produced by a Grass S88 stimulator (Grass Technologies, West Warwick, RI). A series of increasing intensity electrical stimuli (1 ms pulse duration) beginning near the threshold of the H-reflex and continuing to M max were applied. There was an approximate 10 second inter-stimulus latency period. To create H and M recruitment curves the peak-to-peak H-reflex and M wave amplitudes were measured and were normalized to M_{max}. Stimulus intensity was normalized to the maximal stimulus. In order to measure the gain of the unconditioned H-reflex and the M wave, the recruitment curves were imported into a custom LabVIEW (National Instruments Corporation, Austin, TX) program. A 4^{th} order polynomial curve was fit to each curve, the curve was then linearly interpolated to 100 data points, and the peak of the first derivative was calculated (Christie et al., 2004) (Fig 1). The peak of the first derivative was utilized because it may provide a better approximation of the gain of the sigmoid shaped H-reflex curve than the more traditional least squares regression line (Christie et al., 2004).

Pre-Synaptic Inhibition

HPAD is a measure of the relative influence of reflex activation history of the synapse on reflex excitability – functionally acting as a modulator of muscle spindle inflow (Trimble et al., 2000; Earles et al., 2002; Kipp et al., 2011). To measure resting HPAD the paired pulse technique was utilized (Trimble et al., 2000; Earles et al., 2002; Kipp et al., 2011). Two stimuli of the same intensity with a 100 ms inter-stimulus interval were given to the tibial nerve in the popliteal fossa (Kipp et al., 2011). There was an approximate 10 second interval between each pair of stimulations and the intensity of the stimulations increased from near threshold to M_{max}. The paired stimulation produced two H-reflexes, with the second stimulation typically being depressed relative to the first stimulation. The peak-to-peak amplitudes of the second (i.e., depressed) reflex were determined and normalized to M_{max}; and the stimulus intensity

was normalized to the maximum stimulus. A full recruitment curve of the second reflex was obtained and the gain of the curve was determined using the same procedures previously described for determining the gain of the unconditioned H-reflex and M wave.

GABAergic pre-synaptic inhibition reduces the amount of neurotransmitter released from the Ia afferent and is mediated from a variety of sources, including, but not limited to peripheral receptors such as antagonistic muscle spindles, cutaneous receptors, and descending supraspinal commands (Pierrot-Deseilligny and Burke, 2005). To measure resting GABAergic pre-synaptic inhibition the H-reflex was conditioned by stimulating the common peroneal nerve (tibialis anterior) 100 ms prior to stimulating the tibial nerve (soleus). The intensity of the conditioning stimulus was 50% of the maximal tibialis anterior M wave. The intensity of the stimulus used to elicit the test reflex followed the same procedure as the H-reflex and paired pulse protocols (i.e., increased from near threshold to M_{max}). The peak-to-peak amplitudes of the conditioned reflex were normalized to M_{max} and stimulus intensity was normalized to the maximum stimulus. The gain of the GABAergic pre-synaptic recruitment curve was determined as previously described for the unconditioned H-reflex, M waves, and HPAD.

Post-Synaptic Inhibition

Recurrent inhibition, a post-synaptic modulator of motor neuron pool output, was measured at rest to assess post-synaptic inhibition (Earles et al., 2002; Knikou, 2008). Two stimulations to the tibial nerve were provided as previously detailed (Pierrot-Deseilligny et al., 1976; Knikou, 2008). The first stimulation, S_1, was set at 25% of the soleus M_{max}. The second stimulus, S_2, was set at M_{max}. Ten trials of S_1 alone (i.e., test reflex) and 10 trials of S_1 followed 10 ms later by S_2 (i.e., conditioned reflex) were given. A total of 20 trials were given counterbalanced in pairs. The peak-to-peak amplitudes of the test reflex and the conditioned reflex were measured. The percent difference between the amplitudes of the two different reflexes was considered the amount of recurrent inhibition, i.e.,

$$(1-\frac{\text{Test Reflex}}{\text{Conditioned Reflex}})x100\%$$

Rate of Torque Development

To determine RTD, participants were instructed to isometrically plantarflex his or her ankle against the foot plate of the dynamometer as fast and hard as possible in response to a light stimulus. The light was attached to the wall (3 meters) in front of the participant. Three trials with 60 seconds rest between each trial were performed. The dynamometer was interfaced with the Biopac MP100 data acquisition system and data were sampled at 2,000 Hz. The torque-time curves were analyzed using a custom LabVIEW program. The data were first low-pass filtered at 10 Hz (4^{th} order, zero phase lag, Butterworth). Rate of torque development was calculated by determining the slope of the torque-time curve from the onset of torque production, defined as 2.5% of peak torque, over the following time intervals: 0-50 ms, 0-100 ms, 0-150 ms, 0-200 ms, and 0-250 ms. Torque was normalized to $mass^{0.67}$ (Jaric et al., 2005). The average of the three trials for each of the time intervals was used for analysis.

Suprapinal Drive

V waves, a variant of H-reflexes, were measured to determine the level of supraspinal drive (Gabriel et al., 2006). Participants were instructed to plantarflex as fast and hard as possible following a light stimulus the same as they were during the RTD trials, but once they reached 90% of their maximum torque development a maximal electrical stimulus (i.e., M_{max}) was applied to the tibial nerve (Aagaard et al., 2002b). The threshold for M_{max} stimulation (i.e., 90% of peak torque) was calculated from the RTD trials. Five trials of V waves were collected with 60 seconds rest between trials. The peak to peak amplitude of the M wave and the V wave were measured and averaged. The ratio of V wave to M_{max} was considered the amount of supraspinal efferent neural drive.

Statistical Analysis

Stepwise multiple regression analyses were performed for each of the RTD time intervals ($RTD_{0\text{-}25ms}$, $RTD_{0\text{-}50ms}$, $RTD_{0\text{-}100ms}$, $RTD_{0\text{-}150ms}$, $RTD_{0\text{-}200ms}$, $RTD_{0\text{-}250ms}$). The predictor variables were (1) gain of the resting soleus H-reflex recruitment curve, (2) gain of the HPAD recruitment curve, (3) gain of the GABAergic pre-synaptic inhibition recruitment curve, (4) the level of post-synaptic recurrent inhibition at rest, (5) level of supraspinal drive (V waves), and (6) the gain of the resting soleus M Wave. The probability to enter was set at ≤ 0.05 and ≥ 0.10 to remove. All statistical analyses were performed using SPSS 19 (IBM, Armonk, NY).

Results

Table 1 presents the group means and standard deviations. The results of the regression analysis revealed supraspinal drive as measured by V waves (V_{wave}:M_{max}) and resting recurrent inhibition significantly predicted RTD at time intervals less than 100 ms. Specifically, V waves and recurrent inhibition together explained 34.2% ($p=0.001$) of $RTD_{0\text{-}25ms}$, 35.8% ($p=0.002$) of $RTD_{0\text{-}50ms}$, and 36.8% ($p=0.001$) of $RTD_{0\text{-}100ms}$. V waves alone were significant predictors of $RTD_{0\text{-}150ms}$ explaining 30.8% ($p<0.001$). At the later time intervals, $RTD_{0\text{-}200ms}$ and $RTD_{0\text{-}250ms}$, V waves and recurrent inhibition were significant predictors with an explained variance of 41.0% ($p<0.001$) and 43.0% ($p<0.001$) respectively. See Table 2 for regression coefficients.

Discussion

RTD is important for both injury prevention and explosive movements. However an understanding of the mechanisms, particularly at the supraspinal and spinal level, contributing to RTD is not complete. Our results suggest that neural drive from supraspinal centers predict RTD regardless of the time period analyzed. Additionally, resting recurrent inhibition – a post-synaptic modulator of motor neuron pool

output – was a significant predictor of RTD up to 100 ms from onset of contraction and during the later time intervals of 0-200 ms and 0-250 ms.

Supraspinal Neural Drive and RTD

Supraspinal neural drive, measured by V waves, was the only variable that significantly predicted RTD during all of the different time periods. Individuals with elevated V waves also tended to have greater RTD regardless of the time interval examined (see Figures 2 and 3). The fact that suprapinal drive predicted RTD across all time intervals makes intuitive sense. During activation of the plantarflexors, the soleus motor neurons are facilitated by motor commands from supraspinal centers via the corticospinal tract. Since participants were asked to voluntarily plantarflex as hard and fast as possible against the footplate this would invoke a motor command from the supraspinal centers.

These results tie in nicely with previous reports of differential contributions to RTD at different time periods. A recent study reported that early-phase RTD was primarily a factor of agonist neural drive; however, they did not examine the specific mechanism responsible for the changes but it most likely was due to central factors (Tillin et al., 2012). Additionally, the studies that reported that early-phase RTD was primarily a factor of intrinsic muscle properties had explained variances of less than 40% (Andersen and Aagaard, 2006; Andersen et al., 2010). This corresponds with the explained variances we found in that same time window (see Table 2). Additionally, it has been reported that maximal muscle strength is the primary contributor to RTD after 100 ms from onset of contraction; with the explained variances much higher than that reported for earlier-phase RTD (Andersen and Aagaard, 2006). Again our results parallel nicely with those findings because maximal torque production is a byproduct of neural drive from the central nervous system and intrinsic muscle characteristics.

Based on our findings it appears that no matter the time interval examined elevated supraspinal spinal drive as measured by V waves are a predictor of greater RTD. These results fit with previous

reports of contributors to RTD and are important when developing programs designed to enhance rapid torque production.

Recurrent Inhibition and RTD

Recurrent inhibition measured at rest, along with V waves, significantly predicted RTD at time intervals up to 100 ms from onset of contraction and at the later phase time intervals of 0-200 ms and 0-250 ms. While recurrent inhibition was a significant predictor at those intervals, the variance recurrent inhibition explained was less than the amount explained by supraspinal drive at each time interval. Despite having a smaller amount of explained variance than V waves, recurrent inhibition explained over a third of the variance at the 0-25 and 0-50 ms time intervals, approximately a quarter of the variance at the 0-100 ms interval, and less than 15% of the variance at the 0-200 and 0-250 ms time intervals. In short, recurrent inhibition predicted more at the earlier time intervals than the later, particularly at the early phase time intervals identified in previous studies (Andersen and Aagaard, 2006; Andersen et al., 2010; Tillin et al., 2012). Why recurrent inhibition was not a significant predictor to RTD from 0-150 ms is unclear.

Traditionally it has been thought that greater levels of recurrent inhibition reduces the sensitivity of neurons to changes in excitatory drive and decreases the discharge frequency of the alpha motor neuron (Knikou, 2008). However, recurrent inhibition is not simply a negative feedback loop in that it also synchronizes motor neuron discharges during voluntary contractions (Mattei et al., 2003). Greater motor unit synchronization at the onset of contraction has been proposed to result in a greater rate of torque development (Semmler, 2002). While our results cannot directly support this, recurrent inhibition does appear to play an important role as a gain regulator motor neuron pool output (Hultborn and Pierrot-Deseilligny, 1979).

While little is known about the relationship between recurrent inhibition and rapid muscle activation there is evidence that recurrent inhibition is greater in power-trained versus endurance-trained individuals (Earles et al., 2002). Those authors suggested that differences in recurrent inhibition occur because power-trained athletes habitually try to fully activate the motor neuron pool during performance (Earles et al., 2002). Interestingly, a study comparing RTD between explosive trained athletes and untrained controls found greater RTD at 50 ms in the athletes, but no differences between the groups during RTD at 100 and 150 ms (Tillin et al., 2010). The fact that the initial 50 ms of explosive contraction appears to be differentially controlled deserves more research. This early-phase RTD is critical in injury situations due to that fact there is limited time to generate torque to stabilize a perturbed joint. It would be interesting to examine changes in recurrent inhibition and early-phase RTD following an intervention designed to increase explosive torque production.

Limitations and Future Directions

The current results extend previous reports on the mechanisms underlying the functionally important measure of RTD. While our study only examined spinal and supraspinal measures, future studies on contributors to RTD should collectively examine factors intrinsic to the muscle, factors that modulate motor neuron pool output, and descending drive from supraspinal centers. By examining these factors in combination a better understanding how these factors together contribute to rapid torque production can be gained. Additionally, research into the relationship of how both RTD and the spinal and supraspinal variables change in response to different types of resistance training may help guide development of better training regimens for both injury prevention and explosive movements.

A limitation of our study is that we measured spinal motor control of the soleus - a muscle not known for its explosive characteristics. We chose the soleus due to well-established protocols for assessing spinal level modulation of the soleus motor neuron pool. Our objective was to assess the

contribution of a unique collection of spinal and supraspinal variables to RTD. Unfortunately, measures of GABAergic pre-synaptic inhibition, recurrent inhibition, and V waves of other muscles of the lower extremity considered more functional to explosive movements such as the gastrocnemii and the quadriceps have not been adequately developed.

Another limitation of our study is that we collected all of the spinal level variables (i.e., H-reflex, HPAD, GABAergic pre-synaptic inhibition, and recurrent inhibition) at rest. It is well known that these measures are context dependent (Zehr, 2002) and the results may not extend to when the muscle is active. Again, our goal was to measure a large collection of variables known to modulate motor neuron output and protocols for measuring many of the variables we were interested in during movement are not well established. Although caution is urged in extending these findings to movement, the novel collection of variables collected provide a first step in understanding the role of supraspinal and spinal mechanisms on rapid torque production. As additional techniques are developed it may be possible to examine other muscles and movements that are more functional in nature.

Perspectives

Rapid torque production, particularly during time periods when injuries may occur or explosive movements need to be performed, is critical. The results of this study suggest neural drive from supraspinal centers predicts RTD across all time intervals. Additionally, recurrent inhibition, a post-synaptic modulator of motor neuron pool output, when measured at rest predicts RTD across different time intervals, but more so at earlier time frames. Greater recurrent inhibition has been previously suggested to be different in explosive athletes compared to endurance athletes (Earles et al., 2002). These results suggest that supraspinal and spinal mechanisms when measured at rest predict RTD, but is not the same across all time intervals. Combined with the results of others (Andersen and Aagaard, 2006; Andersen et al., 2010; Tillin et al., 2012) it appears influences on RTD are multifactorial

encompassing neural and muscular elements. Injury prevention programs and training regimens should take this into account because many of these factors may show adaptive plasticity.

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Table 1: Means and standard deviations for dependent and independent variables

Variable	Mean values ± SD
H-reflex (mV/V)	10.08 ± 4.15
Intrinsic pre-synaptic inhibition (mV/V)	1.97 ± 1.83
Extrinsic pre-synaptic inhibition (mV/V)	8.46 ± 3.73
Recurrent inhibition (%)	0.77 ± 0.28
V wave (V/M_{max})	0.24 ± 0.19
M wave (mV/V)	6.57 ± 2.05
$RTD_{0\text{-}25ms}$ ($Nm \cdot s^{-1} \cdot [kg^{0.67}]^{-1}$)	11.80 ± 4.83
$RTD_{0\text{-}50ms}$ ($Nm \cdot s^{-1} \cdot [kg^{0.67}]^{-1}$)	15.92 ± 6.90
$RTD_{0\text{-}100ms}$ ($Nm \cdot s^{-1} \cdot [kg^{0.67}]^{-1}$)	19.70 ± 8.62
$RTD_{0\text{-}150ms}$ ($Nm \cdot s^{-1} \cdot [kg^{0.67}]^{-1}$)	18.16 ± 7.89
$RTD_{0\text{-}200ms}$ ($Nm \cdot s^{-1} \cdot [kg^{0.67}]^{-1}$)	15.15 ± 6.23
$RTD_{0\text{-}250ms}$ ($Nm \cdot s^{-1} \cdot [kg^{0.67}]^{-1}$)	12.42 ± 4.98

Table 2: Regression models with standardized regression coefficients across different time-intervals for RTD normalized to body-mass$^{0.67}$ ($Nm \cdot s^{-1} \cdot [kg^{0.67}]^{-1}$).

	Regression Model	R^2_{Adj}
$RTD_{0\text{-}25ms}$	= 0.609·V wave‡ + 0.388·RI*	0.342†
$RTD_{0\text{-}50ms}$	= 0.619· V wave ‡ + 0.397·RI*	0.358‡
$RTD_{0\text{-}100ms}$	= 0.647· V wave ‡ + 0.343·RI*	0.368‡
$RTD_{0\text{-}150ms}$	= 0.573· V wave ‡	0.308‡
$RTD_{0\text{-}200ms}$	= 0.693· V wave ‡ + 0.291·RI*	0.410‡
$RTD_{0\text{-}250ms}$	= 0.708· V wave ‡+ 0.290·RI*	0.430‡

Note: V wave = V:M_{max} ratio, RI = Recurrent inhibition.

* $p < .05$

† $p \leq .01$

‡ $p \leq .001$

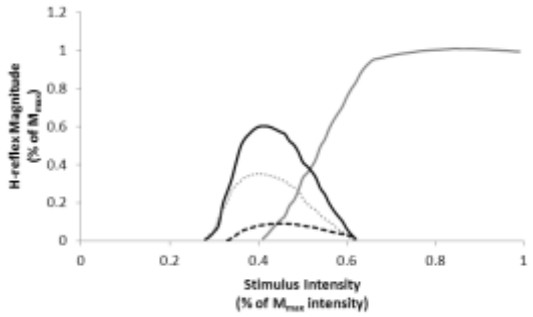
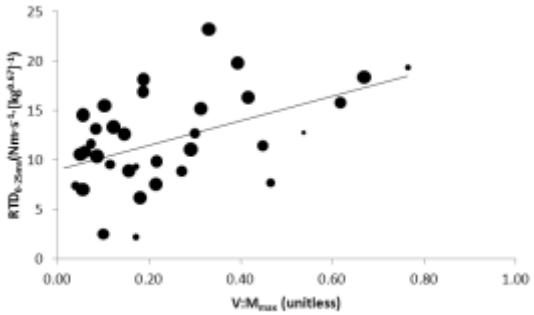


Figure 1: Representative recruitment curve. *Solid black line* - H-reflex, *solid gray line* - M wave, *dashed (diamonds) gray line* - extrinsic pre-synaptic inhibition, *dashed (hash marks) black line* - intrinsic pre-synaptic inhibition.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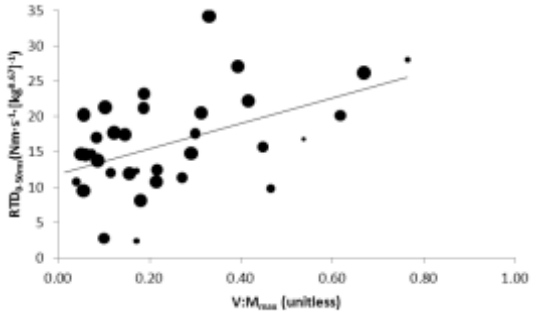
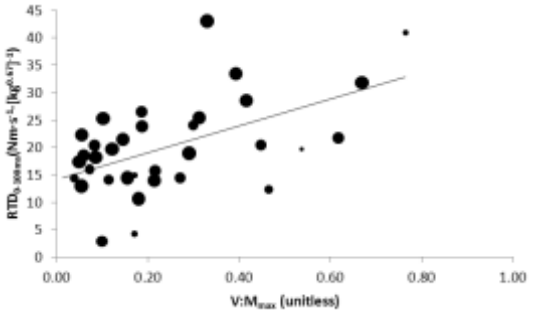
10
5
0
0.00
0.20
0.40
0.60
0.80
1.00
V:M$_{max}$ (unitless)

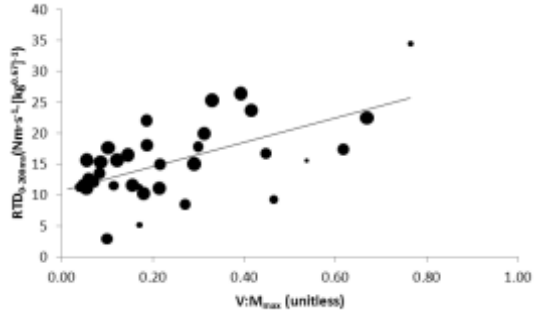
a)


35
30
25
20
15
10
5
0
0.00
0.20
0.40
0.60
0.80
1.00
V:M$_{max}$ (unitless)

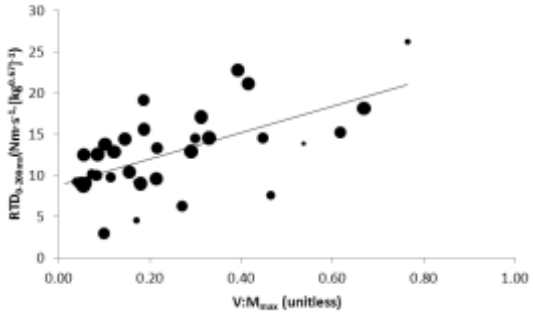
b)


45
40
35
30
25
20
15
10
5
0
0.00
0.20
0.40
0.60
0.80
1.00
V:M$_{max}$ (unitless)

c)


40
35
30
25
20
15
10
5
0
0.00
0.20
0.40
0.60
0.80
1.00
V:M$_{max}$ (unitless)

d)



e)

Figure 2: Relation between normalized V wave magnitude [V:M_{max} (unitless)] and body-mass$^{0.67}$ normalized rate of torque development [RTD ($Nm \cdot s^{-1} \cdot [kg^{0.67}]^{-1}$)] V:$M_{max}$ to **a)** 0-25 ms ($RTD_{0\text{-}25ms}$), **b)** 0-50 ms ($RTD_{0\text{-}50ms}$), **c**) 0-100 ms ($RTD_{0\text{-}100ms}$), **d**) 0-200 ms ($RTD_{0\text{-}200ms}$), and **e**) 0-250 ms ($RTD_{0\text{-}250ms}$). The size of a dot indicates the magnitude of recurrent inhibition (RI) at rest for a given individual (i.e., a larger dot indicates more RI). Note that smaller dots (i.e., individuals with lesser RI) generally fall below the regression line.

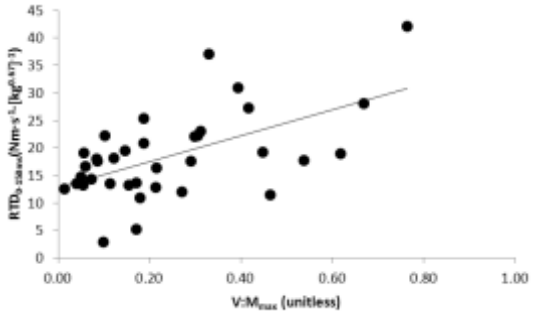


Figure 3: Relation between normalized V wave magnitude [V:M_{max} (unitless)] and body-mass$^{0.67}$ normalized rate of torque development from 0-150 ms [$RTD_{0\text{-}150ms}$ ($Nm \cdot s^{-1} \cdot [kg^{0.67}]^{-1}$)]