Neuromuscular fatigue of the knee extensors does not differ following repeated maximal intensity leg cycling sprints interspersed with 30s and 180s of rest.

By

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Abstract

The objective of this thesis was to examine the development of neuromuscular fatigue during a leg-cycling repeated sprint exercise interspersed with short and long duration recovery times. Ten recreationally active males completed two sessions of ten 10s sprints, interspersed with either 30s or 180s of recovery. Participants completed a knee extensor maximal voluntary contraction (MVC) with interpolated twitch technique (ITT) protocol pre-, mid-, and post-repeated sprint exercise. Participant's peak power was significantly higher during sprint 5 and 10, but not sprint 1, when sprints were interspersed with 180s of rest compared to 30s of rest. Post-sprint 5 there was a significant decreased in MVC force and potentiated twitch force. Post-sprint 10 participant's voluntary activation was significantly decreased. There was no difference between groups for knee extensor MVC force, potentiated twitch force or voluntary activation. Results indicate that a longer recovery time may improve repeated sprint performance but not improve neuromuscular function.

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Bpm	Beats Per Minute
CMEP	Cervicomedullary Evoked Potential
EMG	Electromyography
HR	Heart Rate
ITT	Interpolated Twitch Technique
KgB.W.	Kilogram of Body Weight
MEP	Motor Evoked Potential
M_{max}	Maximal Compound Muscle Action Potential
MVC	Maximal Voluntary Contraction
M-wave	Compound Muscle Action Potential
OSI	Optimal Stimulation Intensity
RMS	Root Mean Square
TMS	Transcranial Magnetic Stimulation
VA	Voluntary Activation
VAS	Visual Analogue Scale
VL	Vastus Lateralis
WT	Wingate Test

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Chapter 1: Review of Literature

1.1. Introduction

1.1.1. Sprinting Exercise:

The ability to repeatedly produce maximal or near-maximal efforts interspersed with brief recovery periods is a vital determinant to successful performance in many sporting activities (e.g. basketball, badminton, soccer, swimming, and cycling). During maximal intensity repeated sprint exercise (sprints of duration of ≤10 seconds) with short recovery periods (i.e. ≤60 seconds), an individual's capacity to produce maximal speed (e.g. running) or high levels of power (e.g. cycling) decreases as the total number of sprints increases (Girard, Mendez-Villanueva, & Bishop, 2011). These decrements are thought to be the result mechanical, metabolic, and neural factors that lead to neuromuscular fatigue (Girard et al., 2011). However, studies involving maximal intensity sprint exercise with longer recovery periods (i.e. >60 seconds) have demonstrated that the longer recovery periods between sprints may allow for a sufficient amount of time to have near-complete recovery from neuromuscular fatigue (Girard et al., 2011). Increased recovery time may help reduce fatigue by, for example, assisting with the removal of intracellular hydrogen ions (Bishop & Claudius, 2005), which are associated with increased neuromuscular fatigue (Amann et al., 2011). As well, the additive time may allow for a greater recovery of phosphocreatine stores (Bishop, 2012), which have been shown to be the most immediate reserve for the rephosphorylation of

adenosine triphosphate and thus utilized during high-intensity short duration activities (Girard et al., 2011).

1.1.2. Neuromuscular Fatigue:

Neuromuscular fatigue is the exercise-induced reduction of the muscle's ability to produce force (Amann, 2011; Gandevia, Allen, Butler, & Taylor, 1996). It is comprised of both peripheral and central mechanisms. Most of the decrements in maximal voluntary force production can be attributed to changes within the muscle that may occur at or distal to the neuromuscular junction (i.e. peripheral fatigue) (Gandevia et al., 1996). Such changes may include alterations in the electrical transmission across the muscle (indicating changes in the sarcolemma excitability) and impairments of the excitation-contraction coupling process, which may result from an impaired release and restoration of intracellular calcium from the sarcoplasmic reticulum and a reduced sensitivity between the contractile proteins and calcium (Bishop, 2012). Furthermore, increased blood acidity (Fernandez-del-Olmo et al., 2013) and hydrogen ion concentrations (Bishop, Edge, Davis, & Goodman, 2004), and muscle deoxygenation (Racinais et al., 2007), can impair metabolic pathways leading to increased neuromuscular fatigue within the muscle. However, there may be influences on the neural pathway from the motor cortex to the muscle (i.e. the corticospinal pathway) that may result in the central nervous system being unable to "drive" the muscle in generating the force it is capable of (i.e. central fatigue) (Gandevia et al., 1996). These influences include excitatory and inhibitory sensory feedback and alterations in motoneuron excitability making them more or less responsive to synaptic input (Rekling, Funk, Bayliss, Dong, & Feldman, 2000). This

degree of "drive" or voluntary activation of a muscle can be assessed, along with the peripheral properties, by evoking a muscle twitch during a maximal effort by applying a supramaximal electrical stimulation to the motor nerve and comparing it to an evoked twitch during rest (Behm, Whittle, Button, & Power, 2002; Gandevia et al., 1996; Merton, 1954). The technique most commonly employed to do such is known as the Interpolated Twitch Technique (ITT).

1.2. Assessment of Neuromuscular Fatigue: Interpolated Twitch Technique

The interpolated twitch technique (ITT) involves applying electrical stimulation to the motor trunk or muscle belly during and post isometric maximum voluntary contraction (MVC) in order to evoke a superimposed and potentiated twitch, respectively (Behm, St-Pierre, & Perez, 1996; Shield & Zhou, 2004). The amplitude of the superimposed twitch during the MVC is used to describe the central component of fatigue, while the loss of force of the potentiated twitch is used to describe the peripheral component of neuromuscular fatigue (Merton, 1954). Voluntary activation can then be quantified by expressing the superimposed twitch as a percentage of the potentiated twitch (see equation 1) (Behm et al., 1996; Shield & Zhou, 2004).

$$VA = \left(1 - \left[\frac{Superimposed\ Twitch}{Potentiated\ Twitch}\right]\right) \times 100\%$$

Equation 1: Equation used to estimate voluntary activation of a muscle.

As the intensity of a voluntary contraction increases, the neural drive to the muscle increases; therefore, more motor units are progressively recruited and the rate at

which they fire also increases (Behm, 2004). If a motor unit has not yet been recruited or is not firing at a rate fast enough to produce its maximal force, then the additional action potential created via eliciting a stimulus will evoke an increment in force from the muscle fibres within that motor unit (Taylor, 2009). In theory, as more motor units are recruited and their rate of firing increased, a reduction in the evoked superimposed twitch should be observed in comparison to when evoked during rest or at contractions of lesser intensity (Shield & Zhou, 2004). If the superimposed twitch becomes undetectable then it is hypothesized that the muscle is fully activated.

The ITT has been reported to be a reliable (Allen, Gandevia, & McKenzie, 1995; Behm, St-Pierre, & Perez, 1996) and valid (Behm et al., 1996; Taylor, 2009) measure for quantifying voluntary activation using both nerve stimulation and other methods such as transcranial magnetic stimulation (TMS) (Sidhu, Bentley, & Carroll, 2009a). In a study conducted by Allen et al. (1995), the authors had five subjects attend five separate sessions, where in each session the subjects completed ten MVCs with the ITT protocol. They demonstrated that the variability of MVC force, control twitch (i.e. potentiated twitch) and voluntary activation within an individual was similar from day to day; whereas, there are day to day differences between individuals. The theory of the ITT is supported by the inverse relationship between the amplitude of the evoked superimposed twitch and the intensity of the voluntary contraction at the moment of stimulation (Taylor citation). This relationship has been demonstrated in the adductor pollicis (Merton, 1954), quadriceps (Behm et al., 1996b), plantar flexors (Behm et al., 1996; Belanger & McComas, 1981), tibialis anterior (Belanger & McComas, 1981), and the biceps brachii (Allen et al., 1995) muscles. By definition, voluntary activation is the level of drive

during an effort (Gandevia, 2001). As previously described, this "drive" is facilitated by the CNS via the corticospinal pathway. When implementing the ITT via motor nerve stimulation the CNS is bypassed. Therefore, based on motor nerve stimulation the ITT does not provide a direct measure of the descending drive to the motonerons in order to contract the muscle; rather it quantifies the drive of the motoneurons to the muscle and how that impacts force production (Taylor, 2009). By using other techniques (e.g. TMS) in conjunction with the ITT, areas of the corticospinal tract (e.g. motor cortex) can be stimulated and central "drive" can be quantified as shown in the knee extensors (Sidhu et al., 2009a) and elbow flexors (Todd, Taylor, & Gandevia, 2003).

1.3. Can A Muscle Be Fully Activated?

A debated issue within the literature is whether or not individuals possess the ability to fully activate their muscles in order to produce maximum force. In a study conducted by Behm et al. (2002) the authors' objective was to determine if individuals could fully activate limb muscles of different function. They chose to assess the quadriceps, plantar flexors, dorsiflexors, and elbow flexors. The authors found that individuals were not able to fully activate their quadriceps (84.5%) in comparison to the near full activation of the plantar flexors (95.0%), dorsiflexors (95.0%) and elbow flexors (98.7%). Behm et al. (2002) attributed the results of this study to the differing reported fibre composition of the muscle groups. Fast twitch muscle fibres have greater recruitment thresholds; thus, muscles containing a greater fast fibre contribution (e.g., quadriceps) may just be more difficult to fully activate (Behm et al., 2002). As the authors

note, their findings were in conflict with other studies reporting full activation (Belanger & McComas, 1981, 1989). In both studies a similar experimental design using the ITT protocol was performed on pre- and post-pubertal males (Belanger & McComas, 1989) and on men and women (19 to 45 years old) (Belanger & McComas, 1981). Belanger and McComas (1989) showed that in pre-pubertal males eight out of ten subjects achieved at least 95% activation of the plantar flexors where six of them achieved 100%. In comparison, all eight post-pubertal subjects achieved at least 95% activation of the plantar flexors where seven of them achieved full activation. Meanwhile, Belanger and McComas (1981) demonstrated that in adult subjects full activation of the plantar flexors and dorsiflexors was achieved during MVCs. However, Behm et al. (2002) results were in accordance with even more studies that found similar results of muscle inactivation (Bulow, Norregaard, Danneskiold-Samsoe, & Mehlsen, 1993; Hurley, Jones, & Newham, 1994; Strojnik, 1995). The authors attributed these differences to differing muscle fibre composition of the muscle groups and methodological design. It has been reported (Johnson, Polgar, Weightman, & Appleton, 1973) that the mean percentage of type II fibres in the quadriceps is higher than that of the elbow flexors, dorsiflexors, and the plantar flexors. Type II fibres have the highest recruitment thresholds, according to the size principle of motor unit recruitment (Henneman, Clamann, Gillies, & Skinner, 1974); therefore, it would be expected that the quadriceps would be more difficult to fully activate. Earlier studies assess voluntary activation in muscle groups may not have had sensitive enough equipment with higher resolution techniques to be able to identify small changes in maximal torque force elicited via an interpolated twitch (Behm et al., 2002; Shield & Zhou, 2004). Along with inconsistencies of the stimulation intensities and

techniques used (Neyroud, Vallotton, Millet, Kayser, & Place, 2014) there may have been reports of overestimation of voluntary activation, as discussed below (Behm et al., 2002).

1.4. Technical and Practical Considerations with the ITT

1.4.1. Stimulation Intensity:

When eliciting electrical stimulation for the ITT there must be complete recruitment of the motor units within the muscle of interest (Neyroud et al., 2014). The intensity that corresponds to this is known as the optimal stimulation intensity (OSI). This is determined by gradually increasing the electrical stimulation until there is plateau in either the evoked twitch force or a plateau in the compound muscle action potential (M-wave), despite an increase in stimulation intensity. In unfatigued muscles, previous investigations of the ITT protocol have concluded that submaximal and maximal stimulation intensities have provided similar estimates of voluntary activation (Shield & Zhou, 2004). Despite the benefits of using submaximal stimulation (e.g., reducing discomfort of stimulation and risk of activating the antagonists) there are disadvantages associated with a reduced intensity. Repeated stimuli at a submaximal intensity may evoke responses in different portions of the muscle; resulting in inconsistent evaluation of the voluntary activation for that muscle as well as during fatiguing protocols the threshold of motor axons increases; thus, submaximal stimulations will progressively activate less motor units (Shield & Zhou, 2004). If either or both of these occur, then the superimposed twitch may diminish resulting in an overestimate of the voluntary activation. Regardless of how OSI is determined, there is little information regarding what stimulation intensity

is sufficient to have complete spatial recruitment while in the fatigued state. In a study conducted by Neyroud et al., (2014) they attempted to determine an appropriate stimulation intensity to adequately assess neuromuscular fatigue. Participants were asked to complete the ITT protocol with electrical stimulation evoked at three different intensities (100, 120, and 150 OSI) pre- and post- a fatiguing protocol. OSI for this experiment was determined by gradually increasing the electrical stimulation by 10 mA increments until there was no further increase in twitch force or M-wave amplitudes. The authors found that using a stimulation intensity of 100% OSI did not guarantee complete recruitment; whereas, using 150% OSI led to greater co-activation of agonist and antagonist muscles as well as discomfort. They concluded that using a stimulation intensity of 120% led to similar mechanical and electrical responses to 150% OSI without inducing greater co-activation or discomfort than 100% OSI. Thus, they recommend using 120% OSI when assessing components of neuromuscular fatigue.

1.4.2. Site of Stimulation:

Typically, electrodes are used to apply electrical stimulation over the nerve trunk that innervates the muscle of interest or the muscle belly. Regardless of electrode placement, it is possible that when the stimuli are applied both the agonist and the antagonist muscles will be activated (i.e., co-contraction) (Shield & Zhou, 2004). In most cases, if the electrodes are placed over the muscle belly near its motor point then the appropriate muscle should become more selectively stimulated. However, Shield and Zhou (2004) state that placing electrodes too far apart, too close to antagonists, using

large electrodes or high stimulation intensities may lead to greater activation of antagonists. Co-contraction of agonist and antagonist muscle groups should be of concern when assessing voluntary activation. The small twitch forces evoked in the antagonists may slightly reduce or mask the evoked twitch forces of the agonist, especially during MVCs. This can leave the impression that the muscle is fully activated when in fact it is not (Shield & Zhou, 2004).

1.4.3. The Number of Stimuli:

Originally, the ITT was performed with a single stimulus during and post voluntary contraction (Merton, 1954). However, when single stimuli are evoked during a contraction the force increments are variable and at times small, thus overestimating voluntary activation (Shield & Zhou, 2004). Therefore, for many researchers it has become common practice to elicit two or more stimuli during the ITT (Behm et al., 1996b; Behm et al., 2002; Hureau, Ducrocq, & Blain, 2016; Pearcey et al., 2015). More so, evidence has been shown that supramaximal twin (i.e., doublets), triple, and quadruple stimuli are less variable and produce larger, more readily detected responses over isometric contractions of 50% of maximum (Suter & Herzog, 2001). However, Behm et al. (1996) used doublets and quintuplets (5 stimuli) during their protocol and did not find an advantage in estimates with the quintuplets. Based on previous studies (Bigland-Ritchie, Furbush, & Woods, 1986; McKenzie, Bigland-Ritchie, Gorman, & Gandevia, 1992), it has been demonstrated that single stimuli are unsuited in studies that induce muscle fatigue because of alterations in the excitation-contraction coupling process.

Bigland-Ritchie et al. (1986) had ten healthy adult subjects perform a fatiguing protocol of 6s contractions interspersed with 4s of rest until the subject's target force (i.e., 50% of control MVC) could no longer be maintained. They demonstrated that no superimposed twitch could be detected by using single pulse stimulations delivered during MVCs, even after the fatiguing protocol; indication full muscle activation. In another study conducted by McKenzie et al. (1992), the authors had the subjects perform a fatiguing protocol (ten MVCs of 10s separated by 10s of rest) where single- and twin-pulse stimuli were delivered. Results showed that there was greater declines in twitch amplitudes when evoked with a single pulse stimulus compared to a twin pulse stimulus; indicating that a greater level of fatigue had occurred when using single pulse stimuli. Based on these findings, doublets interspersed with 10ms are typically employed, especially during fatiguing protocols (Behm et al., 2002; McKenzie et al., 1992; Pearcey et al., 2016; Pearcey et al., 2015).

There are many critiques of using multiple stimuli to evoke a twitch response such as the increased discomfort involved; as well, with an increased number of stimuli used, spinal reflexes have more time to influence the superimposed twitch (Shield & Zhou, 2004b). Herbert and Gandevia (1999) used a computer model based on experimental data from the adductor pollicis muscle collected from three healthy male subjects to simulate twitch forces produced by the interpolated twitch stimulus in order to investigate factors that influence the amplitude of the interpolated twitch. When evoking a response in a muscle via electrical stimulation two impulses may occur: (1) orthodromic (axon conduction in the normal direction; soma to axon terminal) and (2) antidromic (axon conduction in the direction opposite to normal; axon terminal to soma) (Herbert &

Gandevia, 1999). Experimental simulations have demonstrated that antidromic impulses occur approximately 16% in the early recruited motoneurons and nearly 100% in the late recruited motoneurons (Herbert & Gandevia, 1999). Based on their simulations Herbert and Gandevia (1999) demonstrated that when the effects of antidromic impulses colliding with orthodromic impulses were eliminated the amplitude of the interpolated twitch was slightly greater and the time-to-peak force was extended at all contraction intensities. Their findings suggest that antidromic collisions and spinal effects (including reflexes) of an elicited stimulus can reduce the amplitude of interpolated twitches.

1.5. The Wingate Test

The Wingate Test (WT) is an "all out" maximal 30s cycling test. It involves pedalling or arm cranking at maximal speed against a constant force (i.e., resistance) for 30s (Bar-Or, 1987). It is predominately performed via anaerobic energy sources and induces a noticeable amount of fatigue (i.e., drop in mechanical power) within the first few seconds (Bar-Or, 1987). As discussed later, many fatiguing protocols use a modified WT in order to assess neuromuscular fatigue during leg cycling sprints.

When performing the WT under standardized environmental conditions it has been shown that test-retest correlation coefficients have ranged from 0.89 to 0.98 with the majority being higher than 0.94 (Bar-Or, 1987). One study (Tirosh, Rosenbaum, & Bar-Or, 1987) demonstrated that when the leg cycling WT was performed on two separate days, separated by seven to fourteen days, the test-retest r-value was 0.96 for peak power and mean power in children and adolescents (n = 38). Dotan and Bar-Or (1980) tested 28

children (10-12 years old) after 45 minutes of exposure to three different environmental conditions: neutral (22-23^{o}C, 55-60% relative humidity), hot (39-39^{o}C, 25-30% relative humidity), and humid (30^{o}C, 85-90% relative humidity). Correlation coefficient values ranged from 0.89 to 0.93 for mean power, demonstrating that the inter-environmental differences were insignificant. Based on the aforementioned studies, the WT has been shown to yield reproducible scores indicating that it is a highly reliable test (Bar-Or, 1987).

The validity of the WT was determined by comparing performance during this test to other indices of anaerobic performance as no 'gold standard' test exists (Bar-Or, 1987). In the review conducted by Bar-Or (1987), scores of the WT were compared to 17 studies that had subjects perform anaerobic tasks (e.g., sprinting and vertical jump) that required a supramaximal exertion but last only several seconds. Correlation coefficients ranged from 0.32 to 0.92. Bar-Or (1987) suggests that the correlation between performances is high, but the WT may not be used as a predictor of success in those tasks as level of skill plays an important role in successful execution.

1.6. Special Considerations when Implementing the Wingate Test

In order to optimize performance during the WT, several considerations must be made. Such factors would include: (1) the resistance, (2) the crank length, (3) use of toe stirrups, (4) time of day, and (5) practice effects. Dotan and Bar-Or (1983) had 18 female and 17 male physical education students complete five WTs on five separate days using a Fleisch cycle-ergometer. Each session consisted of using a different resistance in order to

test the optimal load for performance. The results suggested that the optimal load for a leg-cycling WT were 5.13 Joule/Rev/kilogram of body weight (KgB.W.) for males and 5.04 Joule/Rev/KgB.W. for females. Bar-Or (1987) later recommended that if using a Monark cycle-ergometer that 0.090Kp/KgB.W. be used in adult non-athletes and 0.100Kp/KgB.W. be used in adult athletes.

In the past, a convectional crank length of 17.5cm was used for all participants during the WT (Bar-Or, 1987). Inbar, Dotan, Trousil, and Dvir (1983) tested optimal WT performance by varying the length of the crank. Thirteen male students (22-27 years old) participated in five sessions, where five evenly spaced crank lengths centred around the convectional 17.5cm were used. Their results demonstrated that the optimal crank length was dependent on leg length. Interestingly, within ±5cm (i.e. two crank lengths) of the optimal crank length peak power did not vary by more than 1.24%. The authors concluded that in a heterogenous population, anthropometric measures must be taken into consideration; if not, performance during the WT may be greatly underestimated.

It was demonstrated by LaVoie, Dallaire, Brayne, and Barrett (1984) that the use of toe stirrups improved performance during the WT. The authors had 50 male subjects perform the WT with and without toe stirrups. Results showed that without the use of toe stirrups, participants' scores in 5-second peak power, 30-second anaerobic capacity, and percent fatigue were all lower than when subjects performed the WT with toe stirrups. The conclusion was that through the use of toe stirrups, participants are able to exert a pushing and pulling force on the pedal throughout the full cycle (Bar-Or, 1987).

The time of day has also been shown to have an effect on the performance of a WT. In two studies (Souissi et al., 2012; Souissi et al., 2007), with similar research designs, it was shown that in boys (mean age = 11.02 years) and healthy males (mean age = 21.8 years) that performance of the WT was better during the evening (17:00-18:00h) compared to when performed during the morning (06:00-07:00h). This was shown as a higher peak and mean power output during the evening session without a significant time effect for root mean square EMG of the vastus lateralis, rectus femoris, and vastus medialis muscles. The increased performance was attributed to better aerobic participation in energy production (Souissi et al., 2007) and improved muscle contractile properties (Souissi et al., 2012). These findings suggest that test-retest of the WT for a subject should be performed at the same time on each day.

Finally, it has been shown that when performing test-retest performance of the WT, there is a practice effect for participants (Barfield, Sells, Rowe, & Hannigan-Downs, 2002). Barfield et al. (2002) had 25 college men perform the WT twice, separated by seven days. Compared to the first trial, there was a significant increase in peak and mean power during the second trial. The authors recommend that at least one full practice WT be given prior to documenting performance for future studies and rehabilitation.

1.7. Neuromuscular Fatigue in Exercise

The type of exercise performed also influences the relative contribution of central and peripheral factors to neuromuscular fatigue (Taylor & Gandevia, 2008). Central and peripheral fatigue have been shown to occur following intermittent isolated muscle

contractions (Taylor, Allen, Butler, & Gandevia, 2000) and multi-joint exercises of long and short duration (Place, Lepers, Deley, & Millet, 2004; Sidhu, Bentley, & Carroll, 2009). Not only does the type of exercise play a vital role but also the intensity and duration of the exercise (Decorte, Lafaix, Millet, Wuyam, & Verges, 2012).

The majority of previous work aimed at assessing neuromuscular fatigue focuses on a pre-/post-exercise experimental design (Duffield, King, & Skein, 2009; Girard, Bishop, & Racinais, 2013b; Racinais et al., 2007). Few studies have attempted to investigate the time course of neuromuscular fatigue developed over the duration of an exercise intervention. In the studies that have examined the time course of neuromuscular fatigue, the results suggest that peripheral fatigue occurs in the early stages of exercise; whereas, central fatigue occurs towards the end of exercise (i.e., near the point of task failure) (Amann, 2011; Lepers, Maffiuletti, Rochette, Brugniaux, & Millet, 2002; Place et al., 2004; Sidhu et al., 2009b).

1.7.1. Constant Load exercise:

Several studies have examined the effect of long duration constant load exercise on neuromuscular fatigue. Lepers et al. (2002) demonstrated that MVC force of the knee extensors decreased after the first hour of constant load cycling and remained depressed even after 30-minutes post-exercise. This was accompanied by a decreased resting twitch torque but not a decrease in voluntary activation. Voluntary activation of the knee extensors was decreased after the fifth hour of cycling but recovered after 30-minutes of recovery, even though it did not return to pre-existing values. Based on these findings the

authors believed that peripheral fatigue developed within the early stages of exercise; whereas, central fatigue developed towards the end of exercise. Similarly, Decorte et al. (2012) showed that the knee extensor MVC force and potentiated twitch force were decreased half way through cycling to exhaustion and that the force and evoked twitch force did not recover within 30-minutes of exercise cessation. After the last quarter of exercise, decreases in voluntary activation were found. Thus, like the aforementioned study, these authors attributed their early findings of fatigue to peripheral fatigue whereas central fatigue played a prominent role in the fatigued experienced at the end of exercise. Sidhu et al. (2009b) used TMS to assess the output from the motor cortex to the knee extensors during a maximal aerobic cycling test. By comparing pre- to post-exercise, they found that the knee extensor MVC force, resting twitch forces, and voluntary activation using TMS and motor nerve stimulation were all decreased immediately and 45-minutes post-exercise. This led the authors to conclude that both peripheral and central fatigue occurred where the failure in voluntary activation was potentially mediated by intramuscular fatigue signals reducing the cortical output. Contrary to the aforementioned studies, Place et al. (2004) found that knee extensor MVC force was decreased only after the fourth hour of running and remained decreased even after 30-minutes of recovery. This was accompanied by a decreased in voluntary activation. However, the potentiated twitch was decreased only after the fifth hour but recovered to baseline measurements after 30-minutes of recovery. These authors attributed their findings of impaired neuromuscular function primarily to central fatigue.

1.7.2. High Intensity Sprint Exercise:

Many studies have attempted to assess neuromuscular fatigue of high intensity exercise using pre- to post-experimental measures (Billaut & Basset, 2007; Billaut et al., 2006; Billaut et al., 2013; Girard, Bishop, & Racinais, 2013a; Girard et al., 2013b; Hureau et al., 2016; Hureau, Olivier, Millet, Meste, & Blain, 2014b; Racinais et al., 2007). In three of these studies (Billaut et al., 2006; Girard et al., 2013a, 2013b; Hureau et al., 2014b), with similar experimental designs, the authors demonstrated that with a 10 x 6s (interspersed with 30s of recovery) repeated sprint protocol, fatigue had occurred. This was seen in the significant reductions in peak power output throughout the sprints, a reduction in potentiated twitch force, and a decreased quadriceps force output in both brief and sustained MVCs. It was even demonstrated by Billaut et al. (2006), that five minutes post completion of exercise that MVC was still significantly decreased. There were no reductions in voluntary activation during any of the MVCs when estimated using peripheral motor nerve stimulation. However, Girard et al. (2013b) also used TMS to evoked twitches during MVCs. The authors observed that exercise-induced reductions in cortical voluntary activation were seen during sustained but not brief MVCs. The studies concluded that repeated sprint exercise did not impair the corticospinal responsiveness of the motor neurons, suggesting that the observed quadriceps fatigue was mainly peripheral in origin. However, Hureau et al. (2014b) still suggest that central fatigue may have occurred. Central fatigue has been demonstrated to recover within three minutes post termination of exercise (Gandevia et al., 1996). Because these authors did not take the neuromuscular measurements until 3min 30s post completion of the repeated sprint protocol, any central fatigue that could have developed may have recovered.

In comparison, other studies (Billaut & Basset, 2007; Billaut et al., 2013; Goodall, Charlton, Howatson, & Thomas, 2015; Hureau et al., 2016) have demonstrated the development of both peripheral and central fatigue following a repeated sprint protocol. In all studies there was a significant decrease in quadriceps MVC force, potentiated twitch force, and voluntary activation. More so, two studies (Goodall et al., 2015; Hureau et al., 2016) went beyond the traditional pre-to-post measurement design and performed neuromuscular assessments throughout the sprint protocol. Hureau et al. (2016) had twelve healthy males attend five sessions randomly; where they performed 1-, 4-, 6-, 8-, or 10- 10s cycling sprints. The authors observed a significant decrease in MVC force and potentiated twitch after the fourth sprint indicating the development of peripheral fatigue. However, after the sixth sprint, they observed a further decrease in MVC force along with a decrease in voluntary activation indicating the development of central fatigue. The authors concluded that peripheral fatigue developed during the first half of exercise, whereas central fatigue developed during the second half in order to prevent excessive locomotor muscle fatigue. Likewise, Goodall et al. (2015) demonstrated reductions in quadriceps MVC force and potentiated twitch force after the second running sprint. Unlike the previous authors, Goodall et al. (2015) also observed reductions in voluntary activation through peripheral motor point electrical stimulation after the second sprint, demonstrating that peripheral and central fatigue developed early in the repeated sprint protocol. The differences observed in these studies suggest that there may be some differences in the fatigability of cycling versus running sprints. None the less, they both demonstrated that central and peripheral fatigue might play a role in the performance decrements seen during repeated sprint exercise.

The studies previously discussed have all attempted to quantify neuromuscular fatigue by using protocols with short recovery periods (i.e., ≤60s). Few studies have examined what happens during repeated sprint protocols with longer (i.e., >60s) recovery periods. Fernandez-del-Olmo et al. (2013) had ten young males (age 25 ± 3 years) complete two WT, separated by 30 minutes of recovery; whereby, they assessed neuromuscular fatigue using TMS. They found that after the first WT, participants' knee extensor MVC force, potentiated twitch force, and voluntary activation were all decreased. During the recovery period, participants MVC force and voluntary activation recovered within 15 minutes; however, potentiated twitch force did not recover. After the second WT MVC force, potentiated twitch force, and voluntary activation all decreased once more. Fernandez-del-Olmo et al. (2013) concluded that both central and peripheral fatigue played a role in the development of neuromuscular fatigue observed; however, the authors suspected that because MVC force and voluntary activation recovered whereas potentiated twitch force did not, central fatigue played a greater role.

Two studies have performed repeated sprint protocols using between 150s and 180s of recovery interspersed between sprints. In both studies, subjects have performed 10 10s sprints of the legs (Pearcey et al., 2015) and arms (Pearcey et al., 2016). Neuromuscular measurements were performed pre-, mid- (i.e., post-sprint 5), and post sprints (i.e., post-sprint 10). Pearcey et al. (2015) observed that after the fifth sprint there was a decrease in quadriceps MVC force and potentiated twitch force but no change in voluntary activation. After the completion of the tenth sprint, MVC force and potentiated twitch force were still decreased and voluntary activation had decreased. Likewise,

Pearcey et al. (2016) observed significant decreases in elbow flexor MVC force, potentiated twitch force, and voluntary activation. Unlike the previously mentioned study, these reductions were only observed after post-sprint 10. However, in both studies the authors concluded that peripheral and central fatigue occurred during the repeated-sprint exercise. Pearcey et al. (2016) had participants complete a second experiment in which they repeated the sprint exercise with measures of corticospinal excitability. They demonstrated that throughout the sprints: motor evoked potential (MEP) amplitude decreased, cervicomedullary evoked potential (CMEP) amplitude increased, and the MEP to CMEP ratio decreased. They authors suggest that the central fatigue observed during repeated arm-cycling sprint exercise is in part due to inhibition and disfacilitation at the motor cortex.

The findings of the aforementioned studies suggest that there may be no differences in the time course of neuromuscular fatigue of repeated sprint exercise with short and long duration recovery periods. However, no study to date has provided a direct comparison between difference recovery periods.

1.8. Pacing

When performing any type of exercise, including high intensity exercise or a MVC, pacing strategies may be utilized (Gibson et al., 2006). Pacing is the conscious and/or subconscious variations of workloads in order to distribute energy resources during exercise to limit premature fatigue (Billaut, Bishop, Schaerz, & Noakes, 2011; de Koning et al., 2011; Halperin, Aboodarda, Basset, Byrne, & Behm, 2014; Tucker &

Noakes, 2009). It has even been described by Gibson et al. (2006) as" a strategy employed in order to avoid catastrophic failure in the peripheral physiological system." It has been suggested that pacing strategies are established at the beginning of the exercise based on pre-exercise expectations of task duration and intensity (Gibson et al., 2006), intramuscular substrate availability (Lima-Silva et al., 2011), motivation (Blanchfield, Hardy, De Morree, Staiano, & Marcora, 2014), presence of competitors (Gibson et al., 2006), and knowledge of the end point (Billaut et al., 2011). Furthermore, the pacing strategy initially chosen is thought to be continuously regulated throughout the exercise in order to accommodate for changes within the external and internal environments (Tucker & Noakes, 2009). The mechanical outputs can then be adjusted in order to maintain performance (Gibson et al., 2006).

Interestingly, in a study conducted by Tucker et al. (2006), it was demonstrated that a global pacing strategy was present in all subjects. Eleven male cyclists performed a 20km self-paced cycling time trial. Power outputs were recorded every 200m. Results showed that peak power fluctuated for all participants. Typically, fluctuations in a signal would indicate 'noise' in the signal (Hu et al., 2004); however, non-random fluctuations have been demonstrated during activities of daily living (Hu et al., 2004), heartbeat dynamics (Ivanov et al., 1999), and gait stride (Hausdorff et al., 1996). Tucker et al. (2006) attributed the fluctuations to regulatory intrinsic biological processes, which may be located in the central nervous system, that respond to the changing afferent information from the peripheral physiological systems. Another interesting finding from the study is the presence of an end burst in peak power for all subjects during the last 2km; showing evidence of a pacing strategy. Previously, it has been shown that end bursts

tend to occur when the task is 90% complete, regardless of the length or time of the task (Catalano, 1973). The has lead to the belief that the central nervous system uses a scalar rather than an absolute time scale when creating a pacing strategy (Church, Meck, & Gibbon, 1994).

In many studies looking at exercise performance, including pacing studies, have provided participants with knowledge of how much work will be required to complete the study. Two studies (Billaut et al., 2011; Halperin et al., 2014) with similar experimental designs have assessed the affects of pacing on task performance when participants had no knowledge of the endpoint. In both studies subjects had randomly completed three sessions: (1) control (subjects were informed about the number of repetitions going to be completed), (2) deception (subjects were told they would complete half the number of repetitions then told they had to complete the other half), and (3) unknown (subjects had no knowledge of the number of repetitions but completed the same amount as the other trials). Billaut et al. (2011) and Halperin et al. (2014) demonstrated that when subjects were deceived about the number of leg-cycling sprints or isometric MVCs they had higher levels of peak power and elbow flexor force, respectively, compared to when they had no knowledge of the number of repetitions. Peak power was significantly higher in the deception group than the control group but MVC force was not. As well, in both studies the cumulative work completed and the average forces produced were not different between the control groups and the deception groups; however, both groups were significantly higher than the unknown groups. Both studies demonstrate the importance of knowledge of the endpoint on pacing strategies in exercise performance. Incorrectly informing the subject about the endpoint may lead to higher efforts initially,

possibly due to a more vigorous pacing strategy, but overall work may not be different than providing full disclosure of the endpoint. However, when withholding the endpoint, subjects may employ a reserve pacing strategy in which they produce less effort in order to maintain performance for the unknown duration (Halperin et al., 2014).

1.9. Conclusion

Performance decrements observed during an exercise task is due in part to neuromuscular fatigue, which had both peripheral and central mechanisms, involved in the impairment of the muscle and decreased voluntary drive, respectively. To date the technique most commonly employed by researcher to assess neuromuscular fatigue is the interpolated twitch technique. By evoking a twitch during and post a maximal voluntary contraction researchers are able to indirectly estimate the magnitude of fatigue incurred. As discussed in this review, there are several methodological considerations outlined in order to optimize the effectiveness of the interpolated twitch technique during. Many of the repeated sprint protocols discussed in this review, as well as the research project, are based off of the Wingate test. Like the interpolated twitch technique, this review discusses methodological considerations of the Wingate test in order to optimize its effectiveness and to ensure reproducible results.

It has been previously shown during long duration exercise that peripheral fatigue develops early into exercise; whereas, central fatigue develops close to the termination of exercise. It has been hypothesized that this decrease in motor drive to the muscle is a result of the central nervous system trying to prevent the excessive development of

peripheral fatigue of the active muscle, which may result in damage. However, it remains unclear how neuromuscular fatigue develops during a bout of repeated sprint exercise. Some researchers have shown that after a repeated sprint protocol only peripheral fatigue had developed. Other researchers using similar experimental designs have demonstrated the development of both peripheral and central causes to the developed neuromuscular fatigue. More so, the effect of recovery duration on the development of neuromuscular fatigue during a bout of repeated sprint exercise remains unclear. It has been thought that a longer duration recovery period would be sufficient in restoring energy stores, removing metabolites, and removing hydrogen ions in order to prevent the development of fatigue. Studies using longer recovery times (i.e., between 150 to 180s) have demonstrated similar results to that reported of studies using short recovery times (i.e., 30s), suggesting that long durations of recovery are still inefficient at preventing the development of neuromuscular fatigue. Despite these findings, to date there is no study that directly compared the effects of repeated sprints interspersed with short and long recovery times.

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Chapter 2: Co-authorship Statement

My contribution to this thesis are outlined below:

i) I recruited all participants and analyzed all data collected for this thesis.

ii) With the help of undergraduate students, Chris Compton and Joseph Yetman, I collected all experimental data for this thesis.

iii) I prepared the manuscript and thesis with the help and guidance of my supervisor Dr. Duane Button.

iv) Drs. Button and Kevin Power provided constructive feedback on the manuscript and Thesis.

Chapter 3: Neuromuscular fatigue of the knee extensors does not differ following repeated maximal intensity leg cycling sprints interspersed with 30s and 180s of rest.

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3.1. Abstract

Purpose: During maximal intensity leg cycling sprints central and peripheral fatigue develops and the development of this fatigue occurs with various (10-30s) short-duration recovery periods between sprints. The purpose of the current study was to compare the development of neuromuscular fatigue during maximal intensity lower-body sprints with short and longer duration recovery periods between sprints. *Methods:* Ten participants completed 10, 10s sprints interspersed with either 30s or 180s of recovery. Peak power outputs were measured for each sprint. Maximal force, voluntary activation (VA) and evoked contractile properties of the knee extensors were measured at pre-sprint 1, post-sprint 5 and post-sprint 10. Heart rate (HR) and perceived pain were also measured immediately following each sprint. *Results:* Peak power output was significantly lower by 16.1±4.2% (p<0.001) during sprint 10 with 30s compared to 180s of recovery. Irrespective of recovery time, maximal force, VA and potentiated twitch force decreased by 26.7±7.2 % (p<0.005), 5.8±1.2% (p=0.025), 38.7±6.1% (p=0.003) respectively, from pre-sprint 1 to post-sprint 10. HR and pain were significantly higher by 37.4±7.9% (p<0.001) and 54.5±9.5% (p=0.004) at post-sprint 10 with 30s compared to 180s of recovery. *Conclusion:* Although decreases in peak power, HR and pain were greater when sprints were interspersed with 30s compared to 180s of recovery, the development of neuromuscular fatigue of the knee extensors was similar. The results illustrate that peripheral fatigue developed early (sprints 1-5) whereas central fatigue developed later (sprints 6-10) in the sprint protocol, however the effect of recovery time on neuromuscular fatigue may be task specific.

3.2. Key words

Power, force, recovery, voluntary activation, task-specific fatigue

3.3. Introduction

Sprinting is a maximal or near maximal intensity exercise of short duration. Typically, maximal intensity sprints are performed repeatedly. Most studies employ a leg cycling sprint protocol that encompasses 5-10 sprints with a set amount of recovery time between each sprint. The sprints are usually ≤ 15s in duration and are interspersed by ≤ 60s of recovery time (Girard et al., 2011). Each sprint imposes high demand on the neuromuscular system, which leads to neuromuscular fatigue and subsequently reduced leg cycling power (i.e. sprint performance) (Girard et al., 2013b; Hureau et al., 2016; Hureau, Olivier, Millet, Meste, & Blain, 2014a; Pearcey et al., 2016; Pearcey et al., 2015; Perrey, Racinais, Saimouaa, & Girard, 2010; Racinais et al., 2007). During a leg cycling sprint protocol, sprint performance decreases as the number of sprints completed increases (Girard et al., 2013a, 2013b; Hureau et al., 2016; Mendez-Villanueva, Hamer, & Bishop, 2007, 2008; Pearcey et al., 2015; Racinais et al., 2007). The decreased sprint performance occurs concomitantly with the development of central (i.e. brain and spinal cord) and/or peripheral (i.e. impairments in the excitation contraction coupling process) fatigue (Billaut et al., 2013; Girard et al., 2013a, 2013b; Hureau et al., 2016; Hureau et al., 2014a; Pearcey et al., 2015; Racinais et al., 2007).

Common measures of central and peripheral fatigue following a leg cycling sprint protocol are voluntary activation (VA) and evoked muscle twitch. VA of the knee extensors has been shown to decrease by ~3.5-11% (Billaut et al., 2013; Hureau et al., 2016; Racinais et al., 2007) indicating that impairments occur suraspinally or spinally. Evoked twitch force of the knee extensors have also been shown to decrease by ~10-50%

(Girard et al., 2013a, 2013b; Hureau et al., 2016; Hureau et al., 2014a; Pearcey et al., 2015; Racinais et al., 2007) indicating that impairments occur at the sarcoplasmic reticulum and contractile proteins (D. G. Allen, Lamb, & Westerblad, 2008; Glaister, 2005). Furthermore, lower muscle pH and increased metabolic by-products also occur (Billaut et al., 2006; D. Bishop, Edge, Davis, et al., 2004; Fernandez-del-Olmo et al., 2013; Gaitanos, Williams, Boobis, & Brooks, 1993; Glaister, 2005; Kinugasa et al., 2004; Mendez-Villanueva, Edge, Suriano, Hamer, & Bishop, 2012; Pearcey et al., 2015; Racinais et al., 2007), which may lead to impairments in twitch force. Some of the aforementioned studies have found that peripheral fatigue occurs early and lasts throughout the leg-cycling sprint protocol and it may play a greater role than central fatigue for the reduction in sprint performance (Girard et al., 2013b; Hureau et al., 2016; Hureau et al., 2014a; Pearcey et al., 2015; Perrey et al., 2010; Racinais et al., 2007). Others have found that central fatigue occurs towards the end of the sprint protocol (Hureau et al., 2016; Pearcey et al., 2015), which may act to limit peripheral fatigue from reaching a critical level (i.e. a safety mechanism) (Gibson, Lambert, & Noakes, 2001). One way to potentially offset the development of neuromuscular fatigue during a leg-cycling sprint protocol and enhance sprint performance is to increase the recovery time between sprints.

Recently, Hureau et al. (2016) demonstrated that sprint performance was gradually reduced over 10, 10s leg cycling sprints interspersed by 30s of recovery. When the participants performed the same sprint protocol with a reduced recovery time (10s), sprint performance was further reduced. Neuromuscular fatigue developed during their sprint protocol, however, it was similar regardless of recovery time (30s vs.10s). Glaister

et al. (2005) also found improved sprint performance and reduced metabolic demand when there was 30s as opposed to 10s of recovery between sprints. Billaut and Basset (2007) found that sprint performance increased as recovery time was increased between subsequent sprints. In each of these studies, recovery times were all less than 1 minute. A recovery time ≥2 minutes between sprints may reduce neuromuscular fatigue. For example, 2 minutes of recovery between sprints allows for an increased muscle buffering capacity and no change in sprint performance from sprint to sprint (Bishop & Claudius, 2005). There is also a significant recovery in skeletal muscle function 1-2 minutes following high intensity exercise (Froyd, Millet, & Noakes, 2013). Finally, neuromuscular fatigue, especially central fatigue, is minimal 2 minutes following the cessation of a leg cycling sprint protocol (Girard et al., 2013b; Hureau et al., 2014a; Perrey et al., 2010; Racinais et al., 2007). Thus, ≥2 minutes of recovery between leg cycling sprints may reduce neuromuscular fatigue thereby enhancing sprint performance compared to a shorter duration recovery.

There is one study (Pearcey et al., 2015) that demonstrated decreased sprint performance and the development of neuromuscular fatigue during a leg cycling repeated sprint protocol (10, 10s sprints) with 180s recovery time between sprints. However, the effect of 180s versus shorter recovery time between sprints on neuromuscular fatigue and sprint performance was not examined. Therefore, the purpose of this study was to determine whether or not neuromuscular fatigue and sprint performance are different when performing a leg cycling sprint protocol with a short (30s) and long (180s) recovery time between sprints. We hypothesized that: 1) neuromuscular fatigue and reduced sprint

performance would occur independent of recovery time and 2) a short compared to a long recovery time between sprints would lead to increased neuromuscular fatigue and greater reductions in sprint performance.

3.4. Methods

3.4.1. Participants:

Ten recreationally active (~10 hours of activity/week) male participants (187.1 ± 5.1 cm, 83.9 ± 10.6 kg, 23.8 ± 4.8 years) were recruited. In an attempt to remove any anticipatory inhibitory effects of evoked stimulation (Button and Behm 2008), all participants were accustomed to maximal bouts of exercise and had prior experience performing MVC with Interpolated Twitch Technique (ITT) protocol. All participants were instructed to refrain from heavy exercise 24 hours before testing and to follow the Canadian Society for Exercise Physiology (CSEP, 2003) preliminary instructions (no eating, drinking caffeine, smoking, or drinking alcohol for 2, 2, 2, or 6 hours, respectively) prior to the start of testing. All participants were also told that they would be completing 10 maximal intensity leg-cycling sprints that were 10s each. Participants were verbally informed of all procedures and read and signed a written consent form. The Memorial University of Newfoundland Interdisciplinary Committee on Ethics in Human Research approved the study (#20151742-HK) and was in accordance with the Tri-Council guidelines in Canada with full disclosure of potential risks to participants.

3.4.2. Leg-cycling Sprint Protocol:

Participants performed all sprints on a Monark ergometer (Monark 874E, Monark Exercise AB, Sweden). Saddle height was adjusted so that, with the crank position at bottom dead centre (face clock analogy - 6 o'clock position) and the foot secured to the pedal with toe clips, the knee joint was almost in full extension (approximately 170^{o}) and the sole of the foot was parallel to the ground. Each sprint was preceded by 10s of slow cycling at a self-selected pace. Immediately flowing the 10s of slow cycling (active rest), the participants began the sprint phase where the electromechanical brake applied a 10%, (Bar-Or, 1987) (of the participant's body weight) torque factor (i.e. resistance in which a subjected peddled against) at 100 rpm and participants were then given verbal encouragement to cycle as hard as they could for 10s. Sprints were interspersed with either 20s or 170s of passive rest. Thus total passive and active rest between sprints were 30s and 180s. The participants repeated this process 10 times. For all sprints, participants were told to accelerate following the initiation of the electromechanical break (i.e., to eliminate any over-estimates in power due to increased acceleration before break application). All power output data were recorded using Monark Wingate Software and stored on a computer. The peak power (watts) was measured during each sprint.

3.4.3. Heart Rate:

All participants were required to wear a Polar (T-31, PolarElectro, Kempele, Finland) heart rate monitor for the duration of their visit to the laboratory. Heart rate recordings were taken pre- and immediately post-sprints.

3.4.4. Perceived Pain:

Perceived pain was measured immediately following each sprint using a visual analogue scale (VAS) (Granges & Littlejohn, 1993). The VAS scale was a horizontal line with anchors at the ends indicating no pain (score of 0) and intolerable pain (score of 10) (Frey Law et al., 2008; Rolke, Andrews Campbell, Magerl, & Treede, 2005).

3.4.5. Knee Extensor Force:

To determine dominant leg knee extensor forces, participants sat on a custom built chair (Technical Services, Memorial University) in an upright position with hips and knees flexed at 90° with arms crossed in front of their body. The upper torso rested against the backrest and was secured with straps placed around the chest. The ankle of the dominant leg was inserted into a non-compliant padded strap, which was placed on the shank and attached by a chain that measured force using a load cell (Omegadyne Inc., Sunbury, OHIO). A load cell detected forces, which were amplified (x1000) (CED 1902, Cambridge Electronic Design Ltd., Cambridge, UK) and displayed on a computer screen. Data were sampled at 2000 Hz. During contractions, participants were instructed to maintain position. Verbal encouragement and visual feedback was given to all participants during each contraction.

3.4.6. Electromyography:

Electromyography activity was recorded from the vastus lateralis (VL) during the MVC with the ITT (MVC/ITT) protocol. Surface EMG recording electrodes (MediTrace Pellet Ag/AgCl electrodes, disc shape, and 10 mm in diameter, Graphic Controls Ltd., Buffalo, NY) were placed 2 cm apart (centre to centre) over the mid-muscle belly of the participant's dominant leg. A ground electrode was secured over the fibular head. Thorough skin preparation for all electrodes included shaving hair off the desired area, followed by cleansing with an isopropyl alcohol swab. An inter-electrode impedance of < 5 kOhms was obtained prior to recording to ensure an adequate signal-to-noise ratio. EMG signals were amplified (x1000) (CED 1902) and filtered using a 3-pole Butterworth with cutoff frequencies of 10-1000 Hz. All signals were analog-digitally converted at a sampling rate of 5 KHz using a CED 1401 (Cambridge Electronic Design Ltd., Cambridge, UK) interface.

3.4.7. Stimulation:

To evoke a maximal twitch force and maximal compound action potential (M_{max}) of the knee extensors, electrical stimulation was applied to the femoral nerve during rest via adhesive Ag-AgCl electrodes (diameter 10 mm) fixed to the skin over the inguinal triangle (cathode) and the greater trochanter (anode). Current pulses (200 μs in duration, 525-900 mA in amplitude) were delivered via a constant-current stimulator (DS7AH, Digitimer Ltd, Welwyn Garden City, UK). The electrical stimulation was gradually increased until the knee extensor twitch force and M_{max} plateaued. A supramaximal

stimulation current (i.e. 50% higher than that required to elicit maximum twitch force and M_{max}) was used for the remainder of the experiment.

3.4.8. Voluntary Activation:

The ITT was utilized as a measure of the central nervous system's ability to fully activate the contracting muscle and has been extensively described previously (Shield & Zhou, 2004). The ITT was performed with two evoked doublets (10 ms apart) at 4s intervals throughout a 10s data collection trial. Prior to performing a MVC, participants were administered an initial evoked singlet, relaxed, and then told to maximally contract their knee extensors for ~3-4s. During the MVC, participants received an evoked doublet (producing a superimposed twitch) and then were instructed to relax. Another evoked doublet (producing a potentiated twitch) was administered following the completion of the MVC. Doublets rather than single stimuli were used to increase the signal to noise ratio, (Behm, St-Pierre, & Perez, 1996a) and to overcome low-frequency muscle fatigue which is known to occur following dynamic exercise (Shield & Zhou, 2004).

3.4.9. Experimental Procedure:

Participants randomly completed two experimental sessions; 1) 30s or 2) 180s of recovery between sprints on 2 separate days (~1.5 – 2 h each) with a least 48h between sessions. Participants began each experimental session with a 5 min warm up on the cycle ergometer at a self-selected pace. Following the warm up participants were prepared for EMG. Next, maximal twitch force and M_{max} were obtained through femoral nerve stimulation. Participants were then instructed to complete a knee extension MVC/ITT

protocol. Following the MVCs, participants completed 5, 10s leg cycling sprints on the cycle ergometer. Each sprint was interspersed by either 30s or 180s of recovery. Immediately, following the fifth sprint, participants were instructed to get off the cycle ergometer and complete the MVC/ITT protocol and then return to the cycle ergometer to finish the remaining duration of their rest period. Participants then proceeded to complete the 5 remaining sprints. Immediately post-sprint 10, participants were again instructed to get off the cycle ergometer and complete the MVC/ITT protocol. The transition time from the cycle ergometer to the start of performing the MVC/ITT protocol post-sprint 5 and post-sprint 10 was less than 10s. It was important to minimize this transition time, because significant recovery of skeletal muscle function occurs 1 – 2 min following exercise (Froyd et al., 2013). Thus, following sprint 5 participants received ~20s and 170s of recovery as opposed to the 30s or 180s of recovery following all other sprints. Heart rate was recorded prior to and post-sprints and pain was also recorded following each sprint (see Figure 1A, B and C for experimental set-up).

3.4.10. Data Analyses:

The ITT was utilized as a measure of the central nervous system's ability to fully activate the contracting muscle (Shield & Zhou, 2004a). VA was calculated by comparing the amplitude of the superimposed twitch force with the potentiated twitch force with the following equation: % voluntary activation = [1 - (superimposed doublet force/potentiated doublet force) X 100]. The potentiated twitch forces were also used to examine changes in peak twitch force. To determine the changes in central activation

failure, the root mean square (RMS) of VL EMG was determined for 500 ms prior to the superimposed twitch during the MVC. A ratio of the RMS EMG and M_{max} amplitude (peak-peak) was calculated and compared at the 3 time points (pre-sprint, post-sprint 5 and post-sprint 10). Knee extensor MVC force, VA and EMG data were measured offline using Signal 4.0 software (Cambridge Electronic Design Ltd., Cambridge, UK).

3.4.11. Statistical Analysis:

All statistics were performed on SPSS (SPSS 18.0 for Macintosh, IBM Corporation, Armonk, New York, USA). Assumptions of sphericity were tested using Mauchley's test and if violated, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity. Two separate two-way repeated measures ANOVAs including recovery time (30s and 180s) X sprint number (1-10) and secondly recovery time (30s and 180s) X time (pre-sprint and post-sprints 5 and 10) were used to determine between and within condition effects for all dependent variables. A Bonferonni Post Hoc test was performed to test for significant differences between interactions. *F*-ratios were considered statistically significant at the $p < 0.05$ levels. Descriptive statistics in text and Table 1 includes mean ± SD and the figures include mean ± SE.

3.5. Results

3.5.1. Peak Power:

All peak power raw data are reported in Table 1. There was a significant main effect for group ($F_{(1, 8)} = 22.2$, $p = 0.002$), sprint number ($F_{(2, 16)} = 18.7$, $p < 0.001$) and a significant interaction ($F_{(2, 16)} = 24.5$, $p < 0.001$) between recovery time and sprint

number on peak power. Peak power was significantly ($p = 0.002$) different with 30s (903.9±103.6 Watts) compared to 180s (1032.2±158.0 Watts) of recovery. Irrespective of recovery time, peak power was significantly ($p < 0.006$) different between sprint 1 (1090.3±173.8 Watts) and sprints 5 (941.5±115.6 Watts) and 10 (875.4±151.2 Watts). Peak power was significantly different between 30s and 180s of recovery during sprint 5 (840.5±86.7 vs. 1023.5±137.2 Watts, $p < 0.001$) and sprint 10 (782.1±113.2 vs. 968.7±162.4 Watts, $p = 0.001$) (Fig. 2).

3.5.2. Heart rate:

There was a significant main effect for recovery time ($F_{(1, 8)} = 15.4$, $p = 0.006$) and sprint number ($F_{(2, 16)} = 49.1$, $p < 0.001$), but no significant interaction ($F_{(2, 16)} = 2.5$, $p = 0.119$) between recovery time and sprint number on post-sprint HR. Post-sprint HR was significantly ($p = 0.006$) different with 30s (164.2±13.9 bpm) compared to 180s (155.7±13.5 bpm) of recovery. Irrespective of recovery time, HR was significantly ($p < 0.001$) different between post-sprint 1 (147.4±15.1 bpm) and post-sprints 5 (166.2±14.5 bpm) and 10 (166.3±13.3 bpm) (Fig. 3A).

There was a significant main effect for recovery time ($F_{(1, 8)} = 76.4$, $p < 0.001$), sprint number ($F_{(2, 16)} = 278.7$, $p < 0.001$) and a significant interaction ($F_{(2, 16)} = 49.4$, $p < 0.001$) between recovery time and sprint number on pre-sprint HR. Pre-sprint HR was significantly ($p < 0.001$) different with 30s (140.8±12.3 bpm) compared to 180s (111.4±17.4 bpm) of recovery. Irrespective of recovery time, HR was significantly ($p < 0.001$) different between pre-sprint 1 (87.9±12.6 bpm) and pre-sprints 5 (144.5±18.3

bpm) and 10 (146.8±14.5 bpm). HR was significantly different between 30s and 180s of recovery at pre-sprint 5 (164.7±13.8 vs. 124.2±22.4 bpm, $p < 0.001$) and pre-sprint 10 (169.1±12.8 vs. 123.0±17.6 bpm, $p < 0.001$)(Fig. 3B).

3.5.3. Pain:

There was a significant main effect for recovery time ($F_{(1, 9)} = 11.7$, $p = 0.008$), sprint number ($F_{(2, 18)} = 28.4$, $p < 0.001$), and a significant interaction ($F_{(2, 18)} = 7.6$, $p = 0.004$) between recovery time and sprint number on post-sprint pain. Post-sprint pain was significantly ($p = 0.008$) different with 30s (2.9±1.8) compared to 180s (1.9±1.7) of recovery. Irrespective of recovery time, pain at post-sprint 1 (0.58±0.8), post-sprint 5 (2.5±2.0) and post-sprint 10 (4.2±2.5) were significantly ($p < 0.012$) different from one another. There was a trend for pain to be significantly different between 30s and 180s of recovery at post-sprint 5 (2.9±2.1 vs. 2.1±2.1, $p = 0.053$) and pain was significantly different post-sprint 10 (5.1±2.7 vs. 3.3±2.4 bpm, $p = 0.004$)(Fig. 4).

3.5.4. MVC force, VA, Twitch Force and Central Activation Failure:

There was a significant main effect for recovery time ($F_{(1, 8)} = 5.6$, $p = 0.05$) and time ($F_{(2, 16)} = 34.9$, $p < 0.001$), but no significant interaction ($F_{(2, 16)} = 1.3$, $p = 0.297$) between recovery time and time on MVC force. Knee extensor MVC force was significantly ($p = 0.05$) different with 30s (577.8±152.1 N) compared to 180s (536.6±149.1 N) of recovery. Irrespective of recovery time, knee extensor MVC forces at

pre-sprint 1 (652.4±187.4 N), post-sprint 5 (540.5±136.4 N) and post-sprint 10 (479.7±120.7 N) were significantly ($p < 0.003$) different from one another (Fig. 5A).

There was a significant main effect for time ($F_{(2, 16)} = 5.8$, $p = 0.048$), but no significant effect for recovery time ($F_{(1, 8)} = .80$, $p = 0.436$) or a significant interaction ($F_{(2, 16)} = 1.5$, $p = 0.283$) between recovery time and time on VA. Irrespective of recovery time, knee extensor VA was significantly ($p < 0.003$) different between pre-sprint 1 (87.8±5.5 %) and post-sprint 10 (82.0±6.5 %)(Fig. 5B).

There was a significant main effect for time ($F_{(2, 16)} = 64.7$, $p < 0.001$) but no significant effect for recovery time ($F_{(1, 8)} = 0.7$, $p = 0.789$) or a significant interaction ($F_{(2, 16)} = 2.4$, $p = 0.117$) between recovery time and time on potentiated twitch force. Irrespective of recovery time, knee extensor potentiated twitch forces at pre-sprint 1 (257.0±74.6 N), post-sprint 5 (199.4±101.0 N) and post-sprint 10 (155.9±58.9 N) were significantly ($p < 0.002$) different from one another (Fig 5C).

There was a significant main effect for time ($F_{(2, 16)} = 3.5$, $p < 0.043$) but no significant effect for recovery time ($F_{(1, 8)} = 2.9$, $p = 0.128$) or a significant interaction ($F_{(2, 16)} = 0.3$, $p = 0.728$) between recovery time and time on EMG/M_{max}. Irrespective of recovery time, EMG/M_{max} was significantly ($p < 0.003$) different between pre-sprint 1 (0.98±0.03) and post-sprint 10 (0.80±0.02).

There were no significant main effects for recovery time ($F_{(1, 8)} = 0.04$, $p = 0.853$), time ($F_{(2, 16)} = 2.1$, $p = 0.112$) or a significant interaction ($F_{(2, 16)} = 1.2$, $p = 0.905$) between recovery time and time on M_{max}.

3.6. Discussion

In the present study, peak power output decreased and HR and perceived pain increased throughout the sprint protocol but to a greater extent with 30s compared to 180s of recovery time between sprints. The sprint protocol decreased knee extensors; MVC force, VA, EMG/M_{max}, and evoked twitch force and the decrease in these variables were independent of recovery time. The time course of the changes in these measurements illustrated that peripheral fatigue occurred early and increased throughout the sprints, whereas central fatigue occurred towards the end of the sprint protocol. Although 30s of recovery time between sprints lead to greater reductions in sprint performance and HR recovery and increased perceived pain compared to 180s of recovery time, the time course and extent of neuromuscular fatigue of the knee extensors when measured from knee extension MVCs was similar between 30s and 180s of recovery time.

3.6.1. Leg cycling sprint performance, HR and perceived pain:

Similar to other studies (Girard et al., 2013a, 2013b; Hureau et al., 2016; Mendez-Villanueva et al., 2012; Pearcey et al., 2015; Racinais et al., 2007), during the repeated sprint protocol, the peak power output during each sprint deceased as the number of sprints performed increased. In fact, the decrease in peak power output from sprints 1-10 with 30s and 180s of recovery between sprints as shown in the current study closely resemble those found by Hureau et al. (2016)(see Figure 5A, 30s recovery) and Pearcey et al. (2015)(see Table 1, 180s recovery), respectively. The rate of decline in peak power during the first half of the sprints (i.e., sprints 1-5) was greater than that during the last

half (i.e., 6-10) and this rate of decline was dependent upon recovery time between sprints. Based on the current results and others (Billaut & Basset, 2007; Glaister et al., 2005; Hureau et al., 2016), there is a preservation of peak power as recovery time between sprints increases during a repeated sprint protocol. A longer recovery time between sprints may allow for further restoration of phosphocreatine and adenosine triphosphate contents (Billaut et al., 2013; D. Bishop & Claudius, 2005; D. Bishop, Edge, & Goodman, 2004; Mendez-Villanueva et al., 2012; Racinais et al., 2007) and release of blood lactate (Glaister et al., 2005), which subsequently improves sprint performance.

HR recorded immediately post-sprint (i.e. indicator of sprint intensity) were similar for both recovery times. HR gradually increased from sprints 1-5 and then plateaued thereafter, which is a similar trend to that reported elsewhere (Foster, Taylor, Chrismas, Watkins, & Mauger, 2014; Glaister et al., 2005; Hureau et al., 2016). The HR recorded immediately pre-sprint (i.e. HR recovery following 30s and 180s of rest) also gradually increased over the first few sprints and plateaued, however HR was much lower following 180s than 30s recovery time from sprints 2-10. During similar sprint protocols, HR recovery was much higher following 30s than 10s of recovery time (Glaister et al., 2005; Hureau et al., 2016). Thus, similar to peak power, heart rate recovery increases as recovery time between sprints increases during a repeated sprint protocol.

Perceived pain gradually increased throughout the sprint protocol from sprints 1-10 regardless of the amount of recovery time between sprints. However, a reduced recovery time between sprints led to greater perceived pain by sprint 10. Although studies have not measured perceived pain in relation to repeated sprint protocols, they have measured rating of perceived exertion (RPE). Pain and RPE during high intensity cycling

exercise are moderately correlated (Borg, Ljunggren, & Ceci, 1985; Cook, O'Connor, Eubanks, Smith, & Lee, 1997). During a repeated sprint protocol, RPE has been shown to increase as sprint number increases (Glaister et al., 2005; Hureau et al., 2016; Pearcey et al., 2016) and that RPE increases as recovery time between sprints decreases (Glaister et al., 2005; Hureau et al., 2016). The current and aforementioned studies indicate that the recovery time-dependent decreases in repeated sprint peak power outputs might be, in part, due to increased pain.

3.6.2. Neuromuscular fatigue:

Typically peripheral fatigue has an early onset (i.e. with in the first 5 sprints) and remains the same or increases further until the end of the repeated sprint protocol (Girard et al., 2013b; Hureau et al., 2016; Hureau et al., 2014a; Pearcey et al., 2015; Perrey et al., 2010; Racinais et al., 2007), whereas central fatigue is associated with the end of the sprint protocol when peripheral fatigue may be reaching a critical threshold (Amann, 2011; Hureau et al., 2016; Pearcey et al., 2015). Similar to the aforementioned studies, the current study showed that peripheral fatigue developed early and increased towards the end of the sprint protocol, thus playing a significant role in reducing MVC force and sprint performance. Because there were no changes in M_{max} from pre- to post-sprints 5 and 10, the peripheral fatigue occurred beyond the sarcolemma. The fatigue may have led to decreased sensitivity of calcium to contractile protein interactions and/or altered release and restoration of intracellular calcium from the sarcoplasmic reticulum thereby altering the excitation-contraction coupling process within the muscle. Increased blood

lactate (Fernandez-del-Olmo et al., 2013; Glaister et al., 2005; Hureau et al., 2016; Kinugasa et al., 2004; Pearcey et al., 2015; Thomas, Sirvent, Perrey, Raynaud, & Mercier, 2004), muscle deoxygenation (Racinais et al., 2007), PCr breakdown (Glaister, 2005) and H^+ (D. Bishop, Edge, Davis, et al., 2004), all of which affect excitation contraction coupling process, also occurs during repeated sprint protocols.

In the present study, increased central fatigue occurred towards the end of the repeated sprint protocol. Others have shown increased central fatigue following repeated sprint (Goodall et al., 2015; Hureau et al., 2016; Pearcey et al., 2016; Pearcey et al., 2015) and Wingate test (i.e. 30s sprint) (Fernandez-del-Olmo et al., 2013) protocols. Central fatigue (as shown by decreased VA) may be due to reduced supraspinal and/or spinal drive to the muscles while performing the sprint(s) and MVC. Recording motor evoked potential (MEP) amplitudes (i.e. corticospinal excitability) via transcranial magnetic stimulation from a muscle of interest is one way to measure supraspinal fatigue. It has been found that MEP amplitudes of the VL do not change following lower body repeated sprint (Girard et al., 2013b; Goodall et al., 2015) or 30s sprint (Fernandez-del-Olmo et al., 2013) protocols suggesting that the occurrence of supraspinal fatigue during sprinting is not due to changes in corticospinal excitability. Interestingly, Fernandez-del-Olmo et al. (2013) showed a decrease in cortical VA of the knee extensors during a MVC following a 30s sprint, which indicates that supraspinal fatigue may be generated upstream to the motor cortical neurons. An overall change or lack thereof in corticospinal excitability may be due to changes in the spinal cord. Spinal excitability measurements were not taken in either of the aforementioned studies. However, Pearcey et al. (2016) measured MEP and cervicomedullary MEP (CMEP) (i.e. spinal excitability) amplitudes from the

biceps brachii following repeated arm-cycling sprints and found a decrease in supraspinal excitability in conjunction with an increase in spinal excitability. Thus, the reduction in sprint performance and MVC force shown here and elsewhere is, in part, due to changes in supraspinal and/or spinal fatigue.

Another measure of central fatigue is central activation failure as measured by EMG/Mmax (Billaut et al., 2013; Girard, Lattier, Maffiuletti, Micallef, & Millet, 2008). In the present study, central activation failure occurred towards the end of the sprint protocol. Several studies have also shown central activation failure following a repeated sprint protocol (Hureau et al., 2016; Hureau et al., 2014a; Racinais et al., 2007). Other studies have also found reduced quadriceps EMG activity (Billaut et al., 2013; Mendez-Villanueva et al., 2007, 2008; Pearcey et al., 2015; Racinais et al., 2007) or no change in muscle compound action potential amplitudes (Hureau et al., 2016; Pearcey et al., 2015) during and following repeated sprint protocols. Thus, the decrease in the central activation ratio may be due to reduced supraspinal and/or spinal activity and hyperpolarization of the motor axons (Kiernan, Lin, & Burke, 2004; Vagg, Mogyoros, Kiernan, & Burke, 1998), but not excitation of the sarcolemma.

Repeated leg-cycling sprints have been shown to increase pain (Foster et al., 2014). Muscle pain leads to increased group III/IV afferent feedback. Feedback from the peripheral (working) muscles via group III/IV afferents provide inhibitory input to the motor cortex and spinal cord, thus limiting the central motor drive to both exercising and non-exercising limbs during intense, fatiguing exercise (Amann, 2011; Amann et al., 2011; Kennedy, McNeil, Gandevia, & Taylor, 2013, 2014; Martin, Weerakkody, Gandevia, & Taylor, 2008; Sidhu et al., 2014). Increased activity of the group III/IV

afferents may be reflected through participants' own perceptions of pain and increased peripheral fatigue. The perception of pain and peripheral fatigue increased throughout the sprint protocol with concomitant decreases in sprint performance and MVC force. The increase in perceived pain and peripheral fatigue (increased group III/IV afferent activity) from pre-sprint to post-sprint 5 might not have been strong enough to impede central motor drive, thus not affecting VA (i.e. central motor drive). However, post-sprint 10 there were further increases in perceived pain and peripheral fatigue (i.e. even greater group III/IV activity) compared to post-sprint 5. By the end of the sprint protocol VA significantly decreased, illustrating that group III/IV activity and peripheral fatigue may have reached a critical threshold and were subsequently governed by central fatigue and the subsequent inhibition of central motor drive.

3.6.3. Task-specific neuromuscular fatigue:

We found a greater preservation of peak power output during sprints when the sprints were interspersed with 180s compared to 30s of recovery time. Others (Glaister et al., 2005; Hureau et al., 2016) showed similar results when sprints were interspersed with 30s compared to 10s of recovery time. Also, as recovery time between leg cycling sprints was increased throughout a repeated sprint protocol, sprint performance increased (Billaut & Basset, 2007). On the other hand, recovery time had no effect on the development of neuromuscular fatigue in the current study as determined via knee extensor MVCs performed pre- and post-sprints 5 and 10. Our results were surprising since there is a significant recovery in skeletal muscle function 1-2 minutes following high intensity

exercise (Froyd et al., 2013) and neuromuscular fatigue (all measured during knee extension or plantar flexion MVCs), especially central fatigue, is minimal 2 minutes following the cessation of a leg cycling sprint protocol (Girard et al., 2013b; Hureau et al., 2014a; Perrey et al., 2010; Racinais et al., 2007). Recently, Hureau et al. (2016) also showed similar results as the current study ((i.e. difference in peak power but not neuromuscular fatigue (as determined by via knee extensor MVCs)) when sprints were interspersed with 30s compared to 10s of recovery time. Based on our findings and others (Hureau et al., 2016), we suggest that the effects of recovery time on neuromuscular fatigue are task dependent.

There are a number of factors that potentially influence the amount of neuromuscular fatigue resulting from a task. Such factors include: the type (i.e. dynamic versus isometric contraction) and intensity of the contraction, the muscle groups involved, and the physical environment in which a given task is performed (Enoka & Duchateau, 2008; Hunter, 2009; Hunter, Duchateau, & Enoka, 2004). Previous studies have shown that there are significant differences in activation of the quadriceps between dynamic and isometric contractions (Babault, Desbrosses, Fabre, Michaut, & Pousson, 2006; Babault, Pousson, Ballay, & Van Hoecke, 2001) and corticospinal excitability of the biceps brachii also differs between arm cycling and an intensity matched tonic contraction (Forman, Raj, Button, & Power, 2014). These findings suggest that activation levels during isometric contractions may not represent those that may be occurring during dynamic movements. Therefore, the use of a single-joint isometric contraction may not be sufficient enough to assess the overall development of neuromuscular fatigue during high intensity multi-joint

dynamic movements such as repeated sprints, especially when determining the effects of recovery time between sprints on neuromuscular fatigue.

3.7. Conclusion

We investigated the effects of recovery time between sprints on neuromuscular fatigue and sprint performance. Regardless of recovery time between sprints, peripheral fatigue occurred early and increased throughout the sprint protocol, while central fatigue occurred towards the end of the sprint protocol. The development of both types of fatigue lead to decreased sprint performance. Although it was not directly measured, part of the neuromuscular fatigue was probably due to increased activity of type III/IV afferents. The participants perceived the sprints as being painful and previous research have shown that type III/IV afferents affect central motor drive. Finally, 180s of recovery helped to preserve some of the decreased sprint performance compared to 30s of recovery, but had no impact on the overall neuromuscular fatigue as determined by knee extension MVCs. Based on the findings from this study and others it appears that the effect of recovery time on neuromuscular fatigue may be task-specific. However, weather or not the development of neuromuscular fatigue during a sprint protocol has a greater impact on sprint performance or an MVC remains unknown.

3.8. Acknowledgements

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3.10. Tables

Table-1: Average peak power (W) raw data outputs during sprints 1-10.

	Recovery Time	
Sprint #	30s	180s
1	1077.2 ± 153.9	1103.4 ± 185.2
2	1009.9 ± 121.8	1103.8 ± 187.6
3	914.6 ± 120.6	1065.7 ± 146.5
4	870.8 ± 90.2	1062.2 ± 164.2
5	840.5 ± 86.7	1023.5 ± 137.2
6	823.1 ± 114.2	1008.2 ± 135.1
7	810.4 ± 84.1	960.4 ± 154.8
8	798.7 ± 85.9	968.3 ± 210
9	801.4 ± 91.6	980.9 ± 159.3
10	782.1 ± 113.2	968.7 ± 162.4

3.11. Figures

Fig 1. Experiment setup. (A) Illustrations of the experimental setup for knee extensors maximum voluntary contraction (MVC), voluntary activation (VA), and electromyography (EMG) (left) and the leg-cycling sprints (right). (B) Timeline of the experimental protocol. Light grey bars represent submaximal intensity cycling that each participant performed prior to the maximal intensity cycling and the white bars represent passive rest in a seated position. Arrows pointing downward indicate when force and VA measures were taken at pre-sprint 1, post-sprint 5, and post-sprint 10. (C) Stimulation protocol used to estimate VA. The single triangle represents a single stimulus, whereas the double triangles represent doublets of stimuli. The grey box represents a knee extensor MVC.

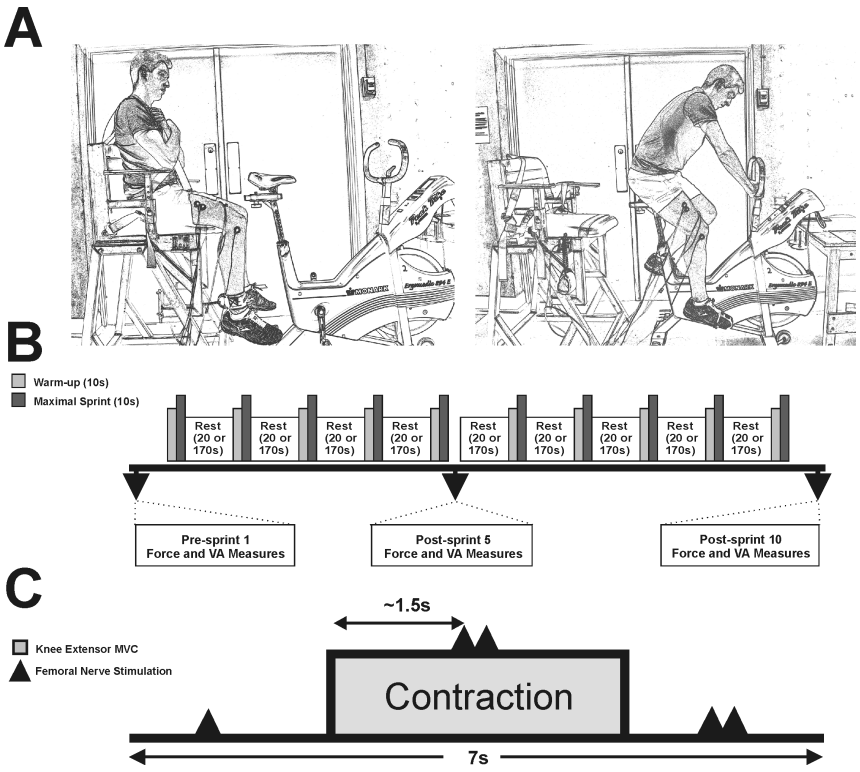

Figure 1: Experimental Setup

Figure 2. Normalized peak power output profiles throughout the 10 sprints. White dots represent a recovery time of 30s and black dots represent a recovery time of 180s between sprints. * Significant ($p < 0.05$) time effect compared to sprint 1 and ** significant ($p < 0.05$) time effect compared to sprint 5; # Significant ($p < 0.05$) differences between groups. Each point represents the group mean ± SE.

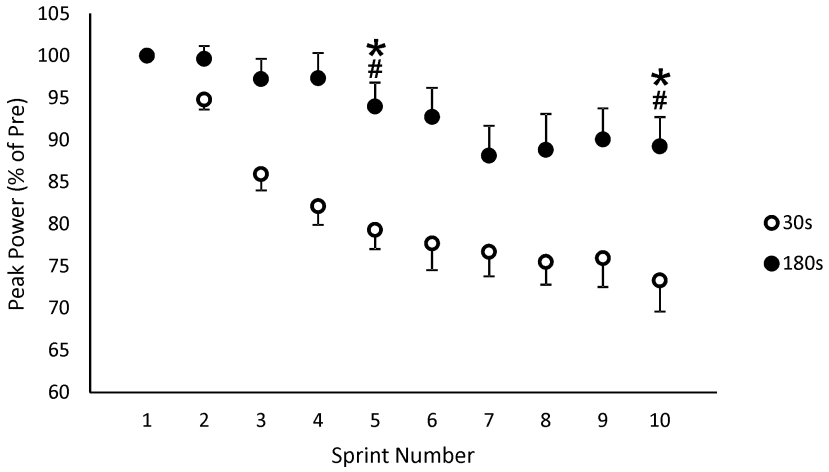

Figure 2: Normalized peak power output profiles through the 10 sprints.

Figure 3. Profile of heart rate (HR) recovery throughout the 10 sprints. Average HR (A) post-sprint and (B) pre-sprint. White dots represent a recovery time of 30s and black dots represent a recovery time of 180s between sprints. * Significant ($p < 0.05$) time effect compared to sprint 1. # Significant ($p < 0.05$) differences between groups. Each point represents the group mean ± SE.

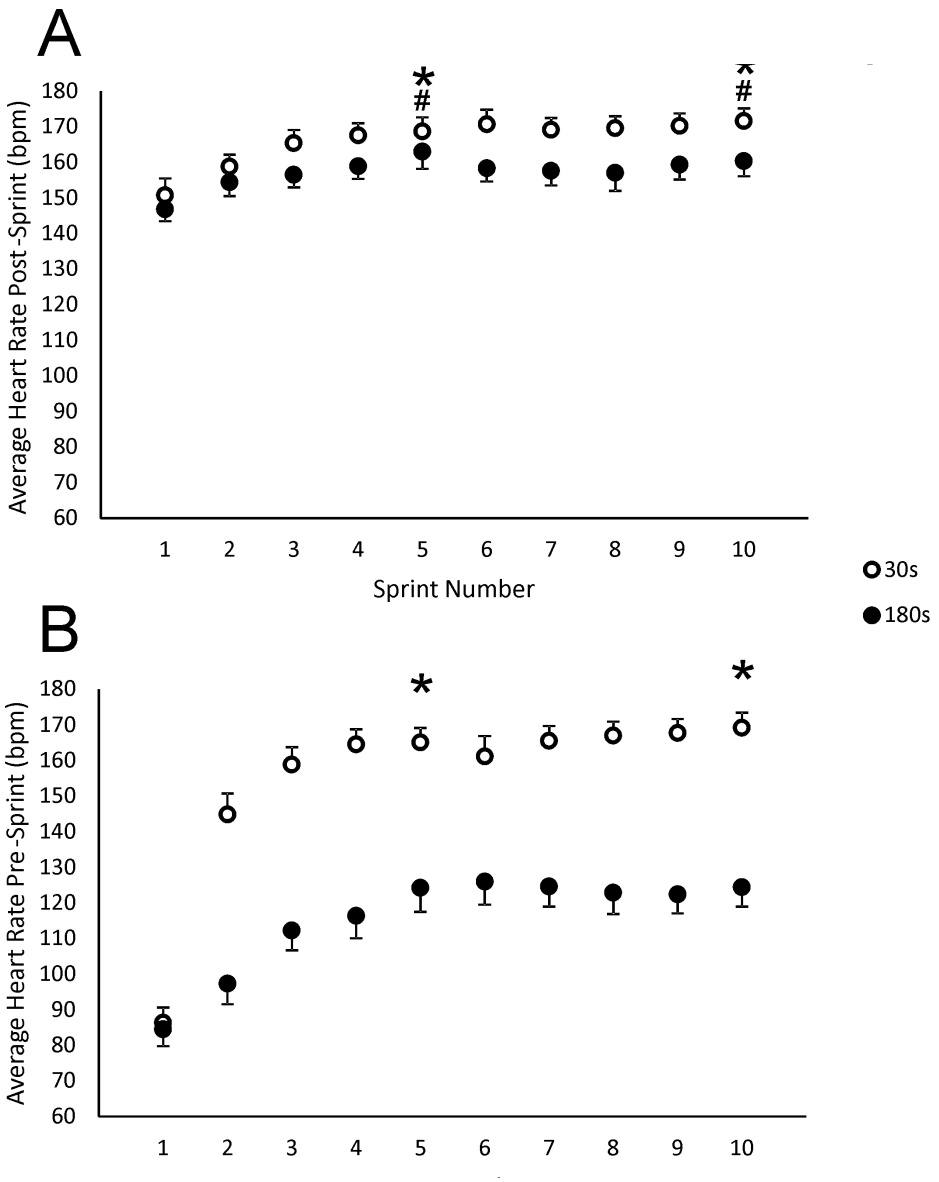

Figure 3: Profile of heart rate (HR) recovery throughout the 10 sprints.

Figure 4. Changes of perceived pain throughout the 10 sprints. White dots represent a recovery time of 30s and black dots represent a recovery time of 180s between sprints. * Significant ($p < 0.05$) time effect compared to sprint 1 and ** significant ($p < 0.05$) time effect compared to sprint 5. # Significant ($p < 0.05$) differences between groups. Each point represents the group mean ± SE.

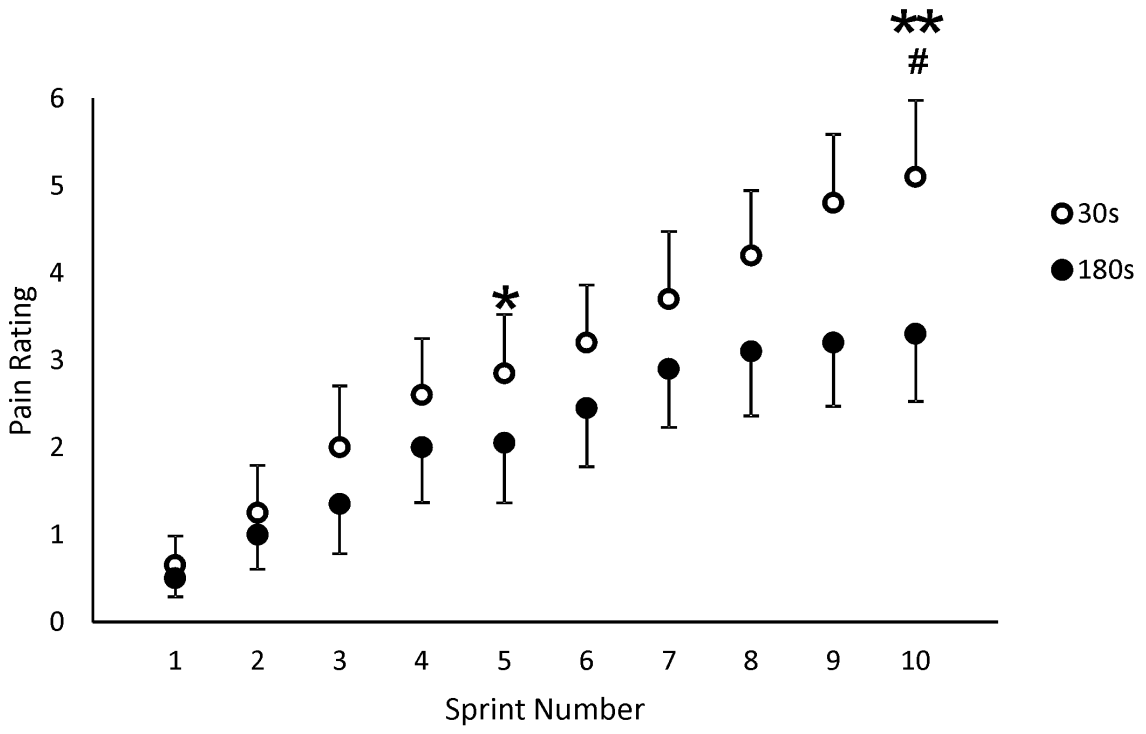

Figure 4: Changes in perceived pain throughout the 10 sprints.

Figure 5. Normalized knee extension (A) maximum voluntary contraction (MVC), (B) voluntary activation (VA) and (C) potentiated twitch force from pre-sprint 1 to post-sprint 10. White dots represent a recovery time of 30s and black dots represent a recovery time of 180s between sprints. * Significant ($p < 0.05$) time effect compared to sprint 1 and ** significant ($p < 0.05$) time effect compared to sprint 5. Each point represents the group mean ± SE.

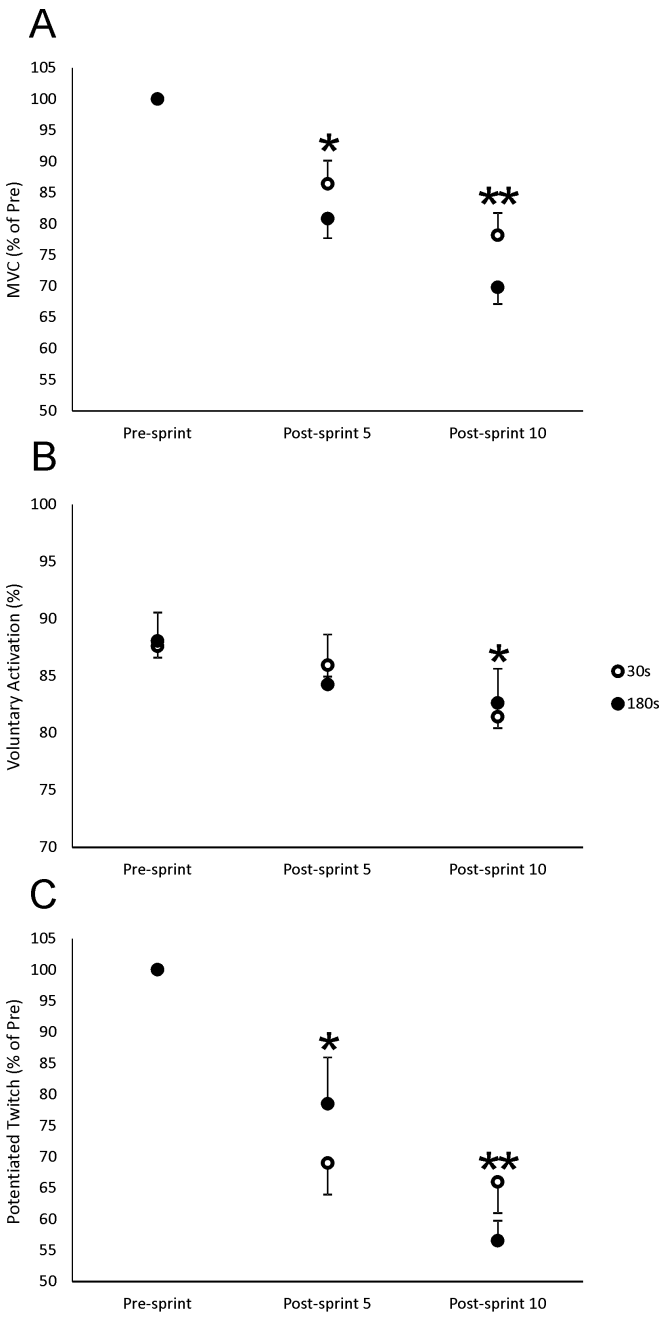

Figure 5: Normalized knee extension (A) maximum voluntary contraction (MVC), (B) voluntary activation (VA) and (C) potentiated twitch force from pre-sprint 1 to post-sprint 10.

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Appendix A: Par-Q+

CSEP approved Sept 12 2011 version

PAR-Q+

The Physical Activity Readiness Questionnaire for Everyone

Regular physical activity is fun and healthy, and more people should become more physically active every day of the week. Being more physically active is very safe for MOST people. This questionnaire will tell you whether it is necessary for you to seek further advice from your doctor OR a qualified exercise professional before becoming more physically active.

SECTION 1 - GENERAL HEALTH

Please read the 7 questions below carefully and answer each one honestly: check YES or NO.		YES	NO
1.	Has your doctor ever said that you have a heart condition OR high blood pressure?	☐	☐
2.	Do you feel pain in your chest at rest, during your daily activities of living, OR when you do physical activity?	☐	☐
3.	Do you lose balance because of dizziness OR have you lost consciousness in the last 12 months? Please answer NO if your dizziness was associated with over-breathing (including during vigorous exercise).	☐	☐
4.	Have you ever been diagnosed with another chronic medical condition (other than heart disease or high blood pressure)?	☐	☐
5.	Are you currently taking prescribed medications for a chronic medical condition?	☐	☐
6.	Do you have a bone or joint problem that could be made worse by becoming more physically active? Please answer NO if you had a joint problem in the past, but it does not limit your current ability to be physically active. For example, knee, ankle, shoulder or other.	☐	☐
7.	Has your doctor ever said that you should only do medically supervised physical activity?	☐	☐

If you answered NO to all of the questions above, you are cleared for physical activity.

Go to Section 3 to sign the form. You do not need to complete Section 2.

- Start becoming much more physically active – start slowly and build up gradually.
- Follow the Canadian Physical Activity Guidelines for your age (www.csep.ca/guidelines).
- You may take part in a health and fitness appraisal.
- If you have any further questions, contact a qualified exercise professional such as a CSEP Certified Exercise Physiologist® (CSEP-CEP) or CSEP Certified Personal Trainer® (CSEP-CPT).
- If you are over the age of 45 yrs. and NOT accustomed to regular vigorous physical activity, please consult a qualified exercise professional (CSEP-CEP) before engaging in maximal effort exercise.

If you answered YES to one or more of the questions above, please GO TO SECTION 2.

Delay becoming more active if:

- You are not feeling well because of a temporary illness such as a cold or fever – wait until you feel better
- You are pregnant – talk to your health care practitioner, your physician, a qualified exercise professional, and/or complete the PARmed-X for Pregnancy before becoming more physically active OR
- Your health changes – please answer the questions on Section 2 of this document and/or talk to your doctor or qualified exercise professional (CSEP-CEP or CSEP-CPT) before continuing with any physical activity programme.

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Appendix B: Free and Informed Consent Form

Informed Consent Form

Title:	Examining neuromuscular fatigue of the upper and lower body during submaximal and maximal cyclical and isometric outputs
Researcher(s):	Mr. Michael Monks, Mr. Joeseph Yetman, Mr. Chris Compton, and Mr. Devin Philpott, Ms. Carla Chaytor
Supervisor(s):	Dr. Duane Button and Dr. Kevin Power

You are invited to take part in a research project entitled *"Examining neuromuscular fatigue of the upper and lower body during submaximal and maximal cyclical and isometric outputs."*

This form is part of the process of informed consent. It should give you the basic idea of what the research is about and what your participation will involve. It also describes your right to withdraw from the study. In order to decide whether you wish to participate in this research study, you should understand enough about its risks and benefits to be able to make an informed decision. This is the informed consent process. Take time to read this carefully and to understand the information given to you. Please contact the researcher, *Michael Monks*, if you have any questions about the study or would like more information before you consent.

It is entirely up to you to decide whether to take part in this research. If you choose not to take part in this research or if you decide to withdraw from the research once it has started, there will be no negative consequences for you, now or in the future.

Introduction:
As part of my Master's Thesis I am conducting research under the supervision of Dr. Duane Button. We are conducting an experiment to assess the impact of rest periods (30- and 180-seconds) on the fatiguing effects of very short (10-seconds) cycling sprints of the upper and lower limbs in people above the age of 18. To our knowledge, scientific research has not examined a direct effect of these differences of rest periods in the upper and lower body. We want to see 1) if there are any differences in the fatigue profile following different rest periods of the upper and lower body, and 2) if there are any differences of the fatigue profile of the upper body compared to that of the lower body following different rest periods.

Purpose of study:
The purpose of this study is to determine (1) the effects of different rest periods on the fatigue profile of upper and lower cycling and (2) to compare the effects of upper body cycling sprints with the lower body.

What you will do in this study:
You will be asked to come to the laboratory for 5 separate sessions for approximately 60 minutes per session. The first session is a familiarization session where you will become introduced to the protocols and techniques used in this study. You will also be required to sign all forms to ensure physical clearance to participate in the study. In the remaining sessions you will either perform multiple high intensity cycling bouts of 1) the upper limbs, or 2) the lower limbs with varying rest periods of 1) 30 seconds, or 2) 3 minutes. The resistance of the bike will be set at a selected percentage of your body weight that will be taken each session. Resistance of the lower body sprints will be set at 10% body weight and the resistance of the upper body will be based on a ratio of peak power output of the upper body compared to the lower body determined during the first session. Prior to the first and following the fifth, final, and 5 and 10 minutes post final bout of cycling, you will be asked to perform a maximal isometric contraction of the arm or leg, in which you will be given electrical stimuli. The stimuli will either be applied to the brachial plexus and biceps brachii muscle (upper body) or the femoral nerve (lower body). This will be used to assess the amount of voluntary force you are able to produce. After each sprint, you will be asked to provide a rating of your perceived exertion and any perceived pain. This will allow the research team to quantify how hard a participant is exercising and if the protocol needs to be stopped at any point.

Length of time:
You will be asked to come to the laboratory for 5 separate sessions for approximately 60 minutes per session.

Withdrawal from the study:
At any point in time throughout the study you may withdrawal from the study by indicating to a member of the research team. By withdrawing from the study any data that has been collected will be destroyed up until the results of the study have been published (~1 year post study completion). There will be no consequences if you choose to withdrawal from the study.

Possible benefits:

1. Participating in this study will allow you to learn about various ways to assess neuromuscular fatigue from maximal intensity exercise.
2. You will be able have your peak power output of your elbow flexors and knee extensors determined and interpreted in comparison to population norms.

Possible risks:

1) You will have electrodes placed on the front and back of your arm and leg. These electrodes have an adhesive that has a tendency cause redness and minor irritation of the skin when the electrodes are removed. This mark is temporary (usually fades within 1-2 days) and is not generally associated with any discomfort or itching.

2) The electrical stimulations will cause twitching of the muscles and mild discomfort, but is not painful. The sensation has been described as if someone flicked your neck and arm muscles firmly with a finger. The sensation will be very brief (less than a second) and will in no way result in any harm to either the muscles or underlying skin.

3) Muscle soreness similar to that following an exercise routine may be experienced by you following this experiment.

4) Transcranial magnetic stimulation used to assess motor cortex excitability is applied at the top of the skull (most individuals do not experience any discomfort). To ensure that there are no contradictions while performing this technique, you will be required to complete and sign a TMS safety checklist.

5) The stimulators used for the experiment are designed for human research, are completely safe and have been used extensively by Dr. Button for many years.

Confidentiality:

The ethical duty of confidentiality includes safeguarding participants' identities, personal information, and data from unauthorized access, use, or disclosure.

Although the data from this research project will be published and presented at conferences, the data will be reported as sample means, so it will not be possible to identify individuals. Moreover, any forms requiring a signature will be stored separately from any data collection sheets used, so that it will not be possible to associate a name with any given set of data. Please do not put your name or other identifying information on any set of data collection sheets used.

Anonymity:

Anonymity refers to protecting participants' identifying characteristics, such as name or description of physical appearance.

Your participation in this study will not be made known to anyone except researchers who are directly involved in this study. Every reasonable effort will be made to ensure your anonymity; and you will not be identified in publications without your explicit permission.

Recording of Data:
There will be no video or audio recordings made during testing.

Storage of Data:

a. All data collected for this study will be kept in a secured location for 5 years, at which time it will be destroyed. Paper based records will be kept in a locked cabinet in the office of Dr. Button while computer based records will be stored on a password protected computer in the office of Dr. Button. The only individuals who will access to this data are those directly involved in this study.

b. Data will be retained for a minimum of five years, as per Memorial University policy on Integrity in Scholarly Research after which time it will be destroyed.

c. The data collected as a result of your participation can be withdrawn from the study at your request up until the point at which the results of the study have been accepted for publication (~1year post study).

Reporting of Results:

a. The thesis will be publically available at the QEII library.

b. Results of this study will be reported in written (scientific article) and spoken (local and national conferences and lectures) forms. For both forms of communication only group average data will be presented. In cases where individual data needs to be communicated it will be done in such a manner that you confidentiality will be protected (i.e. data will be presented as coming from a representative subject).

Sharing of Results with Participants:
Following completion of this study please feel free to ask any specific questions you may have about the activities you were just asked to partake in. Also if you wish to receive a brief summary of the results then please indicate this when asked at the end of the form.

Questions:

You are welcome to ask questions at any time before, during, or after your participation in this research. If you would like more information about this study, please contact: *Michael Monks* at mmonks@mun.ca or *Dr. Duane Button* at dbutton@mun.ca.

The proposal for this research has been reviewed by the Interdisciplinary Committee on Ethics in Human Research and found to be in compliance with Memorial University's ethics policy. If you have ethical concerns about the research, such as the way you have been treated or your rights as a participant, you may contact the Chairperson of the ICEHR at icehr@mun.ca or by telephone at 709-864-2861.

Consent:
Your signature on this form means that:

- You have read the information about the research.
- You have been able to ask questions about this study.
- You are satisfied with the answers to all your questions.
- You understand what the study is about and what you will be doing.
- You understand that you are free to withdraw participation in the study without having to give a reason, and that doing so will not affect you now or in the future.
- You understand that if you choose to end participation **during** data collection, any data collected from you up to that point will be destroyed.
- You understand that if you choose to withdraw **after** data collection has ended, your data can be removed from the study up to the results of the study being accepted for publication (~1 year).

By signing this form, you do not give up your legal rights and do not release the researchers from their professional responsibilities.

Your signature confirms:

☐ I have read what this study is about and understood the risks and benefits. I have had adequate time to think about this and had the opportunity to ask questions and my questions have been answered.

☐ I agree to participate in the research project understanding the risks and contributions of my participation, that my participation is voluntary, and that I may end my participation.

☐ A copy of this Informed Consent Form has been given to me for my records.

_____ _____

Signature of participant Date

Researcher's Signature:
I have explained this study to the best of my ability. I invited questions and gave answers. I believe that the participant fully understands what is involved in being in the study, any potential risks of the study and that he or she has freely chosen to be in the study.

Signature of Principal Investigator	Date