Validation of a dynamic local muscle function test and its response to local muscle temperature

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Dedication

To my abuela, Mamina. I hope my relentless pursuit of my research despite all of the challenges makes you proud.

Abstract

Current single joint isometric and isokinetic tests used to assess muscular performance are limited by the very testing-condition specific nature of the results, leading to a low to moderate correlation to in-vivo performance during sprinting and jumping. This study aimed to develop a single-joint plantarflexor test consisting of 30 s of maximal repeated dynamic contractions from 5° of dorsiflexion through 30° of plantarflexion at 70% of pre-testing isometric MVC. Twelve recreationally active healthy volunteers (9 male, 3 female) completed two different visits where they performed the isotonic plantarflexion test (IPFT). Session 1 aimed to establish its repeatability and variability during thermoneutral muscle temperature (36.6 ± 0.3°C). Across 4 bouts of IPFT with 30 min separation, no significant differences or pairwise differences were observed across measures of power, torque, work completed, time-to-peak torque or rate of torque development while average measures intraclass correlations ranged from 0.935 – 0.996. Session 2 assessed the sensitivity of the test to passive heating and cooling of the lower leg across thermoneutral (TN, 36.5 ± 0.3°C) Hot (38.3 ± 0.2°C) and Cold (33.3 ± 1.2°C) muscle temperatures. Aside from peak torque Hot had no effect on IPFT performance compared to TN, while Cold had a differential effect on IPFT performance. Compared to TN Cold led to significant increases in time-to-peak torque (Δ ↑ 0.14 s) and decreases in total work (Δ ↓ 10.02 J), average power (Δ ↓ 16.92 W) number of repetitions completed (Δ ↓ 4.90), average rate of torque development (Δ ↓ 65.30 $Nm \cdot s^{-1}$) and peak torque (Δ ↓ 6.40 Nm). Changes in performance in Cold were likely due to the effects of cooling on rate processes and contractile characteristics, while we may not have heated muscle enough to impact performance in Hot.

Keywords: Isotonic Plantarflexion Test, Muscle Temperature, Muscular Power, Performance

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I often look forward to the next pursuit without reflecting on what it took to reach a specific milestone. As I am reaching the end of this milestone, I am realizing how lucky I was to be able to pursue this degree and to work with everyone who helped and supported me along the way.

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List of Acronyms

Ca^{2+}: Calcium ions

C_aO_2: Arterial Oxygen Content

CNS: Central Nervous System

F_iO_2: Fraction of Inspired Oxygen

Hb: Hemoglobin

L_{SOL}: Lateral Aspect of the Soleus

MPO: Mean Power Output

MVC: Maximal Voluntary Contraction

M_{SOL}: Medial Aspect of the Soleus

O_2: Oxygen

PCO_2: Partial Pressure of Carbon Dioxide

PPO: Peak Power Output

RFD: Rate of Force Development

RTD: Rate of Torque Development

sEMG: Surface Electromyography

S_pO_2: Oxygen Saturation

TA: Tibialis Anterior

T_{core}: Core Temperature

T_{mu}: Muscle Temperature

T_{skin}: Skin Temperature

TSI: Tissue Saturation Index

TT: Time Trial

TTE: Time to Exhaustion

$T_{Tympanic}$: Tympanic Temperature

$\dot{V}O_2$: Consumption of Oxygen

$\dot{V}O_{2max}$: Maximal Consumption of Oxygen

°C: Degrees Celsius (temperature)

K^+: Potassium ions

Na^+: Sodium ions

SR: Sarcoplasmic Reticulum

MU: Motor Unit

RMP: Resting Membrane Potential

RSA: Repeated Sprint Ability

1. Introduction

1.1 Background

The field of exercise physiology has implemented many different exercise models to determine the impacts of various stressors on neuromuscular capacity. Neuromuscular capacity refers to the various qualities that may determine exercise performance with muscular strength, muscular power or muscular endurance, often the 3 main qualities analyzed. Of particular interest to both the field of exercise physiology and applied sports science is muscular power, defined as the product between force produced during a contraction and the velocity of said contraction [1,2], as it is the functional application of both strength and speed. Often when assessing the effects of various stressors on muscular performance whole-body dynamic exercise models are often used, with isolated muscle models used far less. This is exemplified by a meta-analysis looking at the effects of hyperoxia, where 45 studies implemented full-body endurance exercise models (time-to-exhaustion, time-trial or graded exercise tests) while only 6 studies implemented isolated muscle models, all of which used knee-extension modes [3]. Whole-body exercise models that aim to assess muscular power often adopt jump tests [4,5] or sprinting models [6], while isolated muscle models often utilize isometric maximal voluntary or stimulated contraction models [7]. It is important to assess single-joint performance as it provides an isolated look at the potential effects that various stressors may have on exercise performance through peripheral mechanisms rather than through central mechanisms. Isokinetic models are often used to assess effects of various stressors on neuromuscular capacity during single-joint dynamic exercise [3].

It is important to study exercise performance during single-joint exercise as it provides a

fuller understanding of neuromuscular capacity as it provides insights into the peripheral mechanisms by which performance is dictated. However, many of the commonly adopted exercise tests in the field are limited in providing a full understanding of neuromuscular capacity. While whole-body exercise can provide insights into the various aspects of neuromuscular capacity, results from these models cannot be translated to single-joint exercise models. This is because whole-body exercise performance is confounded by various psychophysiological variables and therefore cannot truly isolate for muscular function alone. During full-body exercise functional limitations may exist, such as fatigued muscles, but can be compensated for by surrounding agonistic musculature. Considering rowing as an example, one may compensate for the development of fatigue in the upper body musculature by adapting their technique to increasingly rely on lower body leg drive to maintain overall rowing performance (i.e., rowing speed, peak power output etc.) which would mask the significance of peripheral mechanisms which may impact performance during single-joint exercise tests. Single-joint exercise tests are often performed isometrically to assess neuromuscular capacity, in the form of voluntary or stimulated maximal involuntary contractions, and while they have their merits they are also limited in their application to dynamic exercise. While isometric models allow for greater insight into neuromuscular function, such as muscle fibre recruitment patterns and electromechanical delay, they do not necessarily account for the functional capacity of muscle during movement throughout its range of motion. This is because performance during voluntary and involuntary isometric contractions are specific to the joint angle that testing occurs in [8–12], and are limited in translation to dynamic performance as they do not account for the impact of changes in the force-length and force-velocity relationships that may occur in dynamic

contractions. Isokinetic models allow for the assessment of muscular strength and muscular endurance during either single-effort or repeated-effort exercise bouts but are limited as contractions during these tests are limited to a certain contraction speed regardless of the force applied by participants. This inherently limits the ability for isokinetic tests to assess muscular power, as a limited contraction speed limits the ability for muscular power to be fully expressed. Additionally, while isokinetic contractions are dynamic in nature they occur at a constant speed which limits the applicability of results from these tests to reflect true functional capacity when all movement characteristics are voluntarily controlled by the participants. There is therefore the need for a dynamic single-joint exercise test to be developed that assesses the neuromuscular capacity of skeletal muscle during continuous repeated efforts. The developed test should be dynamic in nature to consider the variety of factors that influence neuromuscular capacity such as the force-velocity and the force-length relationship; the velocity of contraction should also be controlled by the participants so as not impact performance by contractions occurring at a set contraction speed. Additionally, for the test to be able to assess muscular strength, endurance and power simultaneously the developed test should consist of multiple repeated contractions.

Beyond being repeatable and reliable, any potential exercise tests should be sensitive to potential experimental interventions. Temperature is a well-known stressor that impacts exercise performance. Whole-body endurance exercise may be augmented in cooler conditions with an inverted-U relationship between time-to-exhaustion performance and ambient temperatures being observed in literature [13–16], with specific temperature ranges depending on the specific context. Conversely, whole-body endurance is inhibited in hot environments [16–18]; this relationship between ambient temperature and endurance performance is exemplified in

marathon performance, where faster completion times occurring during cooler ambient wet bulb globe temperatures [19]. The opposite is true when looking at short-duration power dominant tasks such as sprinting, with peak and mean power output increasing in hot environments [6,20] while sprint speed and agility decreases in cool ambient environments [21]. Local level passive heating and passive cooling have also been shown to significantly influence exercise performance as lower leg passive heating, within a physiological safe temperature range from 34-42°C [22–24], may increase jump height by 2-5% for every 1°C increase in muscle temperature (T_{mu}) [5], while performance decreases by 2-10% for every 1°C increase in T_{mu} [25–27]. The temperature mediated changes in performance are seemingly due to alterations in muscle contractility and ultimately muscular power, with passive heating improving muscular power production and contractile function and passive cooling decreasing muscular power and contractility [25,26]. Chief among the changes in muscle contractility are thermal dependent changes in nerve conduction velocity. Nerve conduction velocity increases by ~5% per °C as nerve temperature increases [28–30], while it decreases by 1.5-2 m/sec for every 1°C drop in skin temperature [31]. The increase in nerve conduction velocity leads to faster and more forceful contractions [32], with the decrease in nerve conduction velocity leading to slower and weaker muscle contractions.

1.2 Purpose, Methods and Main Objectives

It was therefore the purpose of this study to develop a dynamic single-joint exercise test that may mimic the muscular demands of in-vivo performance while allowing for the full assessment of muscular performance during repeated dynamic contractions. To this end we conceptualized the isotonic plantarflexion test (IPFT) to meet the following criteria: i) contraction characteristics volitionally controlled by the participants ii) single-joint exercise test

as to allow for the assessment of dynamic muscular performance iii) repeated bouts as to assess functional capacity of muscle. The IPFT consisted of a 30-second bout of repeated cycles of active concentric plantar-and-dorsi flexions throughout a 35°range of motion (5 °dorsiflexion through to 30°plantarflexion) with the knee secured at 90°of flexion; plantarflexion resistance was 70% of the average peak torque achieved during 2 pre-experimental 5 second isometric MVCs with the ankle at 20°plantarflexion, while dorsiflexion resistance was set at the lowest possible resistance the dynamometer allowed (0.5 Nm). We assessed the repeatability, reliability and variability of the IPFT at thermoneutral by comparing performance across 4 different 30-second bouts separated by 30-minutes of rest performed within a single session. We also aimed to develop a test that would be sensitive to local thermal manipulation. We assessed the sensitivity of the IPFT to passive heating and cooling by comparing performance during 3 different 30-second bouts where the muscles of the lower leg were either maintained at thermoneutral (Soleus T_{mu} = ~36.6°C), passively heated (Soleus T_{mu} = ~38.3°C) or passively cooled (Soleus T_{mu} = ~33.3°C) prior to the IPFT. We selected an isotonic exercise model as the characteristics of each contraction, such as speed and complete range of motion, would be dependent on the volitional efforts of the participant rather than controlled by the dynamometer. We selected the plantar flexors as they are a relatively superficial muscle group which allowed our thermal manipulations to take effect within the 30-minute rest period within bouts. Additionally, we biased the soleus as it is more reliant on oxidative metabolism due to it being composed of more type-I muscle fibres when compared to the gastrocnemius; this in turn would mean that any of the potential effects of our thermal manipulation on blood flow and oxygen availability would impact the soleus more than the non-oxygen dependent gastrocnemius

muscle..

Therefore, the main objectives of this study were to:

1. Determine the repeatability and variability of the proposed IPFT as a single-joint exercise test of the plantar-flexor muscles
2. Determine the sensitivity of the IPFT to local thermal manipulation

2. Literature Review

2.1 Exercise Performance: How is it Measured and Neuromuscular Fatigue.

2.1.1 Basics of Performance: What is Performance and How is it Measured?

Looking at performance from the lens of evaluating skeletal muscle function, 3 macro-level categories are often used: 1) muscular strength 2) muscular power, and 3) muscular endurance. Muscular strength can classically be defined as the maximal amount of force a muscle can produce when contracting at a specific velocity [33], with further definitions pertaining specifically to the type of muscle action such as isometric, isokinetic and dynamic concentric/eccentric contractions; typically literature uses maximal voluntary contractions (MVCs) to assess muscular strength. From a purely neuromuscular perspective muscular power is defined as the product of a) the force produced by a contraction and b) the velocity of said contraction ($\frac{(Force\ x\ Distance)}{Time}$) [1,2] or ($\frac{Work}{Time}$), and is the functional application of both strength and speed. From an applied sports science perspective muscular power is often defined as the maximum amount of force a muscle can generate in a short period of time [34], or the ability to perform fast and forceful movements such as sprinting and jumping [35–38]. Jinha and Herzog [2] provide more context for the definition of muscular power and the potential issues with the definitions used in the applied sport science and strength and conditioning literature. The ability to express muscular power is underpinned by muscular strength, as well as the rate of force development (RFD)/rate of torque development (RTD). RTD/RFD is typically determined by the slope of the force/torque-time curve [39,40], with neuromuscular activation largely influencing early rate RTD development and muscle mass influencing late RTD [40]. Rate of torque/force development is an important measure as, in comparison to pure strength derived

from maximal voluntary contractions, it is better related to sport-specific and functional daily task performance [41,42] while being more sensitive to changes in neuromuscular function [43,44], and might be reliant on different mechanisms than MVC derived measures of maximal strength [45,46]. Additionally, measures of RTD/RFD provide insight into neuromuscular function, as early rate of torque development (~0-100ms) during voluntary contractions is more dependent on neural factors [40,46–48]. Muscular endurance refers to a muscles ability to avoid fatigue[34], and is often defined as the ability for muscle to sustain a given level of force production over time [49]; muscular endurance can be assessed by a)the ability to maintain a singular contraction until failure b) the amount of time a certain amount of force can be sustained before it declines or c) the maximum amount of repetitions that can be performed at a percentage of ones maximum capacity.

2.1.2 Neuromuscular Fatigue

Neuromuscular fatigue, defined as the reduction in the force and/or power producing capabilities of skeletal muscles due to exercise [50,51], has both central and peripheral origins. Central fatigue is composed of all the spinal and supraspinal factors involved in the decrease in motoneuron excitation which ultimately leads to a decrease in force production. Central fatigue predominantly dictates fatigue during long-duration low-intensity exercise and can be characterized by decreased central command through alterations in the activity of cerebral neurotransmitters and muscular afferent fibres [52], however also observed during maximal activity such as sprinting [53]. Peripheral fatigue involves the processes that occur at the neuromuscular junction (NMJ), nerve endings, and within the muscle fibres themselves. Peripheral fatigue occurs with the impairment of muscle contraction with changes in calcium ion movement, the accumulation of metabolic byproducts from contraction, and a decrease in

intramuscular adenosine triphosphate (ATP) levels. Boyas & Guével [52] outlined the sites in which a decrease in force generation may occur and are as follows, with sites 1-3 being central sites and 4-9 being peripheral sites:

Central sites:

1) Activation of the primary motor cortex
2) Propagation of the command from the central nervous system (CNS) to motoneurons
3) Activation of motor units (MUs) and muscles

Peripheral sites:

4) Neuromuscular propagation (including propagation at the neuromuscular junction)
5) Excitation-contraction coupling
6) Availability of metabolic substrates
7) State of the intracellular medium
8) Performance of the contractile apparatus
9) Blood flow

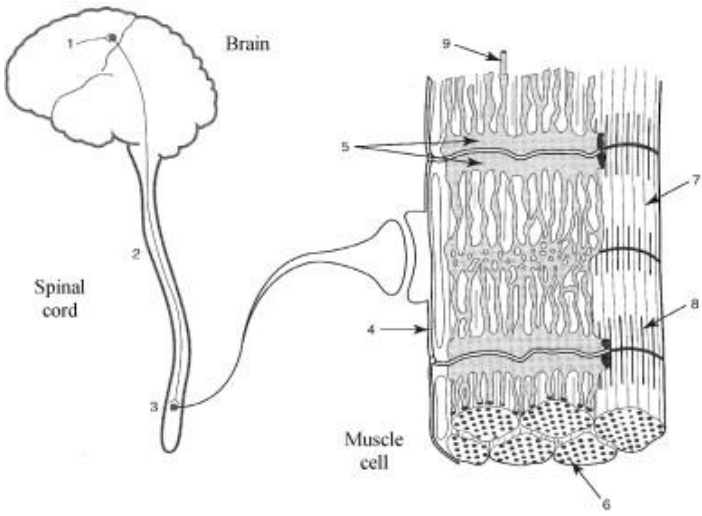

Figure 2.1: Central and Peripheral sites of Neuromuscular Fatigue [52].

The onset of fatigue occurs due to alterations in the mechanisms involved in force production (i.e. cross-bridge cycling, action potential propagation, clearing and metabolites), with the interaction between central and peripheral neuromuscular fatigue leading to a decrease in force production. The following section aims to outline both the central and peripheral factors

related to the development of neuromuscular fatigue.

Central Factors

Central fatigue can be defined as a progressive exercise-induced degradation of the voluntary activation of muscles [53,54], and encompasses all supraspinal and spinal physiological phenomena capable of decreasing motoneuron excitation. It primarily occurs due to a decrease in excitation supplied by the motor cortex, and a decrease in motoneuron activity [55]. Central fatigue significantly contributes to a decrease in performance during sustained contractions, with work from Gandevia's group demonstrating that ~25% of reductions in force during sustained MVCs occurring due to central fatigue[53,56,57]. Similarly, decreases in force generation during low intensity efforts is significantly dictated by central fatigue [58–61]. Again, work from Gandevia's group supports this, where sustained elbow flexion at 5% led to a 28% decrease in MVC torque and a 10-20% decrease in voluntary activation [61].The following section will explore the spinal and supraspinal factors involved in central fatigue.

Neurotransmitters such as serotonin play a significant role in supraspinal central fatigue, with early work suggesting that an increase in serotoninergic activity in the brain limits central command and the recruitment of motor units (MUs) [62]. The exercise induced elevation in tryptophan levels facilitates its passing into the brain, allowing for serotonin synthesis to occur which increases the concentration of serotonin, ultimately causing central fatigue and decreased performance[63,64]. Studies using serotonin agonist/antagonists demonstrate an inverse dose-dependent effect on exercise capacity, such that increases in serotonin concentration reduces the duration of physical activity [63,65]. Specifically, increases in exercise intensity correlates with an increase in serotonergic activity, leading to the feeling of lethargy, attenuated neural drive, and

a loss of motor unit recruitment [63].This tryptophan-serotonin model is supported by animal work, where the release and reuptake of serotonin leads to increases central fatigue [66]. Dopamine also influences the onset of fatigue, with the secretion of dopamine delaying the onset of fatigue [67,68]. Dopamine plays a pivotal role in motivation, memory, reward and attention [69–71], with increases in dopamine activity occurring with prolonged exercise suggesting that the increased activity allowed for the maintenance of exercise, and as such delaying the onset of fatigue [65]. Davis and Bailey [65] went on to build on the work from Newsholme et al. [62] suggesting that instead of central fatigue being solely mediated by the metabolism of serotonin it is an increase in the ratio between serotonin and dopamine that is important in accelerating the onset of fatigue. In general, it seems that the serotonin: dopamine ratio, along with other neurotransmitters (glutamate, acetylcholine, adenosine, gamma-aminobutyric acid) are more closely linked to central fatigue rather than the absolute levels of serotonin itself being the sole cause of central fatigue [63,66,72–74].

Cortical activity may be limited by certain muscle afferents, in turn contributing to supraspinal fatigue [75], where feedback about the biochemical status and force generating capacity at the muscle level leads to a reflex inhibition of motor neurons and a reduction in the excitatory signals sent to the muscles[53].This has been demonstrated in work utilizing ischemia to prevent recovery from fatigue. The metabolite-sensitive group III and IV muscle afferents continued to discharge as long as muscles were ischemic which allows for supraspinal fatigue to persist, with corticospinal and motoneuronal activity only returning to normal values once ischemia stopped [76,77]. There is also a reported decrease in excitation from the motor cortex that occurs with central fatigue demonstrated in studies using transcranial magnetic stimulation

(TMS) [55,78]. TMS involves the stimulation of neurons in the motor cortex, which causes the innervated motor units to contract as a result, with a decrease in excitation being quantified if there is an increase in force and EMG levels following the stimulation [52]. This has been demonstrated during sustained sub-maximal exercise, with decreases in excitation from the motor cortex occurring in isometric arm flexion at 15% and 30% MVC [79–81]. These findings collectively suggest that the group III and IV afferents contribute to limiting voluntary activation of MUs.

The group III and IV afferents also contribute to the spinal factors associated with central fatigue. Early literature demonstrated that the discharge rate of motoneurons is regulated by peripheral reflexes that are sensitive to the fatigue-induced metabolic changes in muscle [82]. Group III and IV muscle afferents are stimulated by ischemia, hypoxemia and the accumulation of potassium (K+) and lactate in extracellular fluid [83–86]. The stimulation of the group III and IV afferents during fatiguing exercise may specifically inhibit the activity of alpha-motoneurons [84,87–90]. Motoneurons are also inhibited by Renshaw cells, which are ironically stimulated by said motoneurons as well as other peripheral factors [91]. Several studies have used the indirect Hoffman reflex method to demonstrate that this inhibition of motoneurons increases with maximal efforts, but falls with submaximal contractions when central fatigue occurs [92–94]. Alpha motor neurons innervate skeletal muscle fibres and cause contractions by releasing acetylcholine into the neuromuscular junction ultimately leading to the propagation of an action potential to the innervated motor unit. It is logical to conclude that the inhibition of the alpha motoneuron would then lead to the inhibition of skeletal muscle contraction, ultimately leading to a negative impact on force production and contractile function of skeletal muscle.

Peripheral Factors

Peripheral fatigue can be defined as reduction in muscle function caused by a reduction in the efficacy of the neuromuscular junction as well as metabolic and biochemical changes within the muscle itself [95,96]. Peripheral fatigue occurs at the nerve endings, the NMJ, and within the muscle fibres themselves. Factors involved in peripheral fatigue include alterations in neuromuscular transmission, the propagation of muscle action potentials, excitation-contraction coupling and other related contractile mechanisms [52]. The following section will explore some of the key factors relating to peripheral fatigue.

Neuromuscular transmission can be defined as the transformation of the nerve action potential (AP) into a muscle action potential at the neuromuscular junction. The impairment of neuromuscular transmission can be demonstrated by eliciting electrically stimulated M-waves before, during and after a fatiguing contraction [52], with the M-wave corresponding to the electrical activity of muscle (indexed by EMG) in response to the electrical stimulation of the innervating nerve. A decrease in the M-wave amplitude is indicative as the degradation of one or multiple pathways involved in the conversion of the nerve action potential into the muscle action potential. A decrease in the quantity of neurotransmitters released by each nerve ending leads to a decrease in the amplitude of motor-endplate potentials, with the release of acetylcholine being pivotal in this process. Decreased release of acetylcholine may be related to a reduction in the number of exocytotic vesicles and/or a decrease in the amount of acetylcholine released per vesicle which has been demonstrated in animal models [97,98]. Additionally, neuromuscular transmission is also dependent on the sensitivity to acetylcholine of the post-synaptic membrane receptors, such that a decrease in sensitivity of the receptors

would lead to an impairment of neuromuscular transmission. Work in animal models have demonstrated that when cholinergic receptors are subjected to prolonged exposure to acetylcholine they become desensitized to the neurotransmitter, which increased the time that it takes for it to bind to the receptors [99].

Calcium plays an essential role in the mechanisms which lead to force generation, with early work using a prolonged isometric contractions showing a reduction in force correlated with a decrease in Ca^{2+} release from the sarcoplasmic reticulum (SR) [100]. The attenuation of the release of Ca^{2+} from the SR seems dependent on the oxidative capacities of the muscle fibres, such that it occurs later in type-I fibres and earlier in type-II fibres [101,102]. Ca^{2+} is critical in processes in contraction from the propagation of the muscle action potential along the sarcolemma and transverse tubules through to the binding to troponin to allow for cross-bridging [52], with changes in Ca^{2+} mechanics due to fatigue likely altering the ability for muscles to produce force [103]. Changes in inorganic phosphate (P_i) levels contribute to the impaired release of Ca^{2+}. Exercise causes the accumulation of P_i in muscles allowing for P_i to enter the SR, where P_i is then able to bind to Ca^{2+} which would limit the amount of Ca^{2+} capable of being released by the SR [104]. Adenosine tri-phosphate (ATP) levels seemingly impact the amount of Ca^{2+} released by the SR on multiple different levels [105]. The repeated propagation of APs Na+ and K+ concentrations become unbalanced on the respective sides of the cell membrane, where the hydrolysis of ATP supplies energy for the Na+/K+ cycling where 3 Na+ and removed from the cell and 2 K+ are into the cell in each cycle to maintain homeostasis. Therefore, a drop in ATP would alter this balance nature of the Na+/K+ cycling which would then attenuate the propagation of APs to the SR, and ultimately the release and reuptake of Ca^{2+} by the SR [106,107].

The opening of K+ channels is also impacted by the concentration of ATP [108], where the exercise evoked drop in ATP signals the channels with the same events previously outlined hypothetically can impact ATP and thus Ca^{2+} release [109]. The SR is also responsible for the reuptake of Ca^{2+}, therefore a decrease in the activity of the ATP-driven pumps that facilitate reuptake would logically decrease the amount of Ca^{2+} available for release. ATP is also critical to the impulse transmission between the transverse tubules and the SR and therefore also in the release of Ca^{2+}; a drop in ATP reserves associated with fatigue may attenuate phosphorylation in sites involved in AP propagation to limit Ca^{2+} [110]. The decrease in Ca^{2+} may also be impacted mitochondria [111], with skeletal muscle mitochondria containing increased concentrations of Ca^{2+} following exercise-fatigued muscles when compared to control muscles [112] suggesting that the mitochondrial uptake of Ca^{2+} contributes to the decrease in available Ca^{2+} [52].

The metabolic changes associated with exercise and metabolic substrate levels can influence performance during sustained exercise [113,114]. Increases in intracellular hydrogen ions (H+) and P_i underpin the reductions in force production seen with fatigue [105]. The hydrolysis of ATP and the production of lactic acid leads to the accumulation of H+, with the accumulation of H+ correlating with a decrease in intracellular pH, thus perturbing chemical reactions associated with muscle contraction. In general, early work looking at high-intensity intermittent efforts suggested that acidosis (decreases in intramuscular/intracellular pH) may be the cause of fatigue [115–119]. However, some literature has suggested that at a 'normal' body temperature the increased activity has a limited impact on force production capability of muscle [52,120]. It has subsequently been demonstrated that the accumulation of P_i is responsible for the decrease of force generation with peripheral fatigue. Work in animal models has both shown that increases

in intracellular P_i attenuates force production when intracellular fluid is saturated with Ca^{2+} in skinned muscle fibres [121], while reducing the concentration of P_i has been shown to increase force production [122]. P_i seemingly attenuates force production by decreasing Ca^{2+} sensitivity of myofibrils and acting directly on cross-bridges [123,124], with increased intracellular P_i during fatigue manipulating the contraction-relaxation cycles associated with cross-bridge cycling [104,123,125,126]. It is likely that both are true. During maximal intensity exercise, saturation of the ATP buffering systems causes the accumulation of H+ and P_i which impacts a) the number of cross-bridges and b) the affinity of actin for myosin; alongside an increase in ATP hydrolysis, there is ultimately a drop in muscle pH. These processes ultimately lead to a reduction in Ca^{2+} release from the SR leading to the consequent reduction in force production [127]. As a whole, these findings suggest that the attenuation of force production in the beginning phases of muscular contraction is due to the accumulation of P_i and other metabolic byproducts of the contraction process.

2.2 The Effects of Temperature on Performance

Temperature significantly impacts exercise performance, with the effects of temperature being dependent on the type of exercise (i.e., endurance events vs power/strength events), as well as the type of contraction (i.e., dynamic vs isometric). When performance is dictated by endurance cooling augments performance while heating hinders performance. When performance is reliant on power, heating may function as an ergogenic aid while cooling limits performance. A general explanation for this divergence is that exposure to heating accelerates while cooling slows metabolic and contractile processes. Exposure to heating and cooling may also affect muscle contractile characteristics. This section outlines the effects of different ambient temperatures, whole body thermal manipulation and local thermal manipulation on

muscular performance and will highlight some key mechanisms that may impact performance with thermal manipulations.

2.2.1 The Effects of Ambient Temperature on Performance

When comparing whole-body endurance performance in varying ambient temperatures cooler environments augment [13,15] while hot environments lead to decrements in performance [18]. This relationship is further exemplified by marathon performance, where faster completion times occurring during cooler ambient wet bulb globe temperatures [19]. Galloway & Maughan [13] found that demonstrated improved time-to-exhaustion performance within a temperature range of ~4-11°C and was worse when temperature strayed either side of the range, however performance at 4°C and 21°C had same effect. Similarly, Sandsund et al. [15] demonstrated that time-to-exhaustion was significantly longer in cool ambient temperature (-4°C and 1°C) when compared to cold (−14 and −9 °C) and warm (10 and 20 °C) when wearing cross-country skiing clothing. These findings exemplify the inverted U-relationship between ambient temperature and endurance performance, however optimal temperature(s) appear to be context specific. However, when ambient temperatures are too cold aerobic power, work time and time-trial performance all decrease [128,129]. In the cold performance may suffer due to reductions in movement economy and decreased force production [130,131], with decreases in hot environments occurring due to increased demands on the cardiovascular system to maintain exercise performance and thermoregulatory demands [132].

The opposite relationship exists relative to ambient temperatures and performance in short-duration power dominant efforts (i.e., sprinting, jumping, throwing) with the historical analysis of the World Athletic Championship competitions suggesting that high temperatures lead to decreases in performance in events longer than 800m [133]. Work on cyclic-natured efforts like

sprinting in hot environments has demonstrated 8-15% increases in mean power output and 25% increases in peak power output [6,20]. 30 m sprint speed and agility performance in the Illinois Test were significantly worse in -5°C when compared to 10.5°C and 30°C ambient temperatures [21], with 15-minutes of exposure to a cool environment (6.1°C) significantly decreasing vertical jump performance [134]. Additionally, double poling sprint performance is significantly inhibited in cold (−14° C) ambient conditions in comparison to moderate conditions (6 °C), with significant reductions in power output during maximal sprint and exhaustion tests in well-trained skiers [135]. While not specifically assessing the effects of cold ambient temperatures on performance, 15 minutes of cold water immersion led to a 9.5 % and 13.7 % reductions in maximum power and average power in 30-second maximum effort cycle sprints [136]. As a collective these findings suggest that the impact of ambient temperature on performance is not only dependent on the degree of heating or cooling caused by the environment, but that the effects of ambient temperature may be specific to the type of exercise assessed.

2.2.2 The Effects of Whole-body Hyperthermia and Hypothermia on Performance

Prolonged exposure to hot environments may cause hyperthermia (37.5 - 40.0°C), which significantly impacts exercise performance and neuromuscular function. Passive hyperthermia is detrimental to performance as there is a resulting decrease in central neural drive to working muscles. Prior work which mainly used isometric performance tasks has pointed to hyperthermia directly impairing central nervous system activation irrespective of local skin or muscle temperature [137–139]. In these studies voluntary activation decreased as T_{core} increased and returned towards baseline levels as T_{core} decreased, suggesting an inverse relationship between voluntary activation and T_{core} irrespective of changes in skin [137] and muscle temperature [139].

However, data from isometric and maximal contractions may not translate to dynamic and/or submaximal contractions [140]. Work using isokinetic maximal voluntary knee extensions with the same passive heating and cooling protocol demonstrated that peak torque remained largely unaffected during full-body passive heating between a T_{core} range of 37.5°C to 39.5°C [141], but rather that a higher skin temperature was the primary driver for impairment. Supporting this work, Colletta et al. [142] demonstrated the effects of both T_{core} and T_{sk} on performance of a submaximal isometric force task and a dynamic iso-inertial position task. It was concluded that T_{core} was the primary thermal driver influencing isometric submaximal force performance, while T_{sk} was the primary driver relative to the position task with T_{core} having a negligible influence [142]. These findings suggest that the effects of whole-body passive heating on performance are dependent on the mode of the selected exercise task.

While mild cooling may improve endurance performance, prolonged exposure to cold causes hypothermia which negatively impacts performance. Mild hypothermia, which is indicated by a -0.5 to -2.0 ˚C decrease in T_{core} , clearly negatively impacts exercise performance [129,143,144]. Wallace et al. [144] demonstrated that decreasing T_{core} by 0.5 °C and 1.0 °C through prolonged exposure to a cold environment (116-160 minutes of exposure to 0 °C room with 0.8-1.2 m/s wind speeds while seated) reduced TTE performance ~ 61.4% (21.7 min vs 7.0 min) and ~71.6% (21.7 min vs 4.0 min) respectively. In a time-trial (TT) test, performance decreased following a 0.5 °C decrease in T_{core} with a 6% reduction in average wattage throughout the 15 km TT [129]. Hypothermia causes multiple responses in the body as the body aims to maintain T_{core} such as vasoconstriction and shivering (Figure 2.2). Shivering increases the metabolic demands on the body [145], and primarily occurs in the trunk and thigh muscles which may reduce

muscle capacity during exercise [130,146].

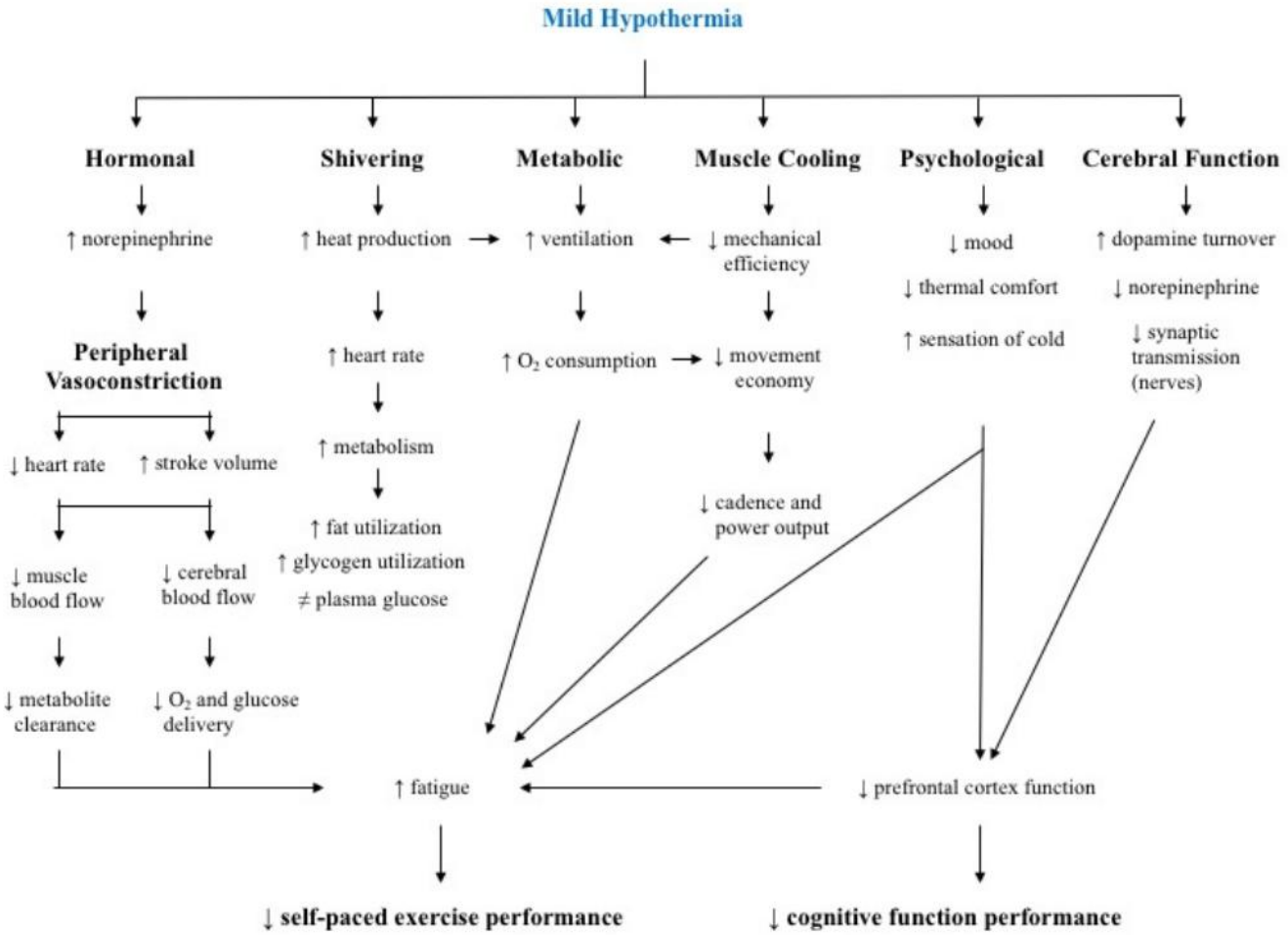

Figure 2.2: The psychophysiological responses to acute cold stress and mild hypothermia on self paced exercise and cognitive performance. Used with permission from the author [143].

2.2.3 The Effects of Targeted Segmental Thermal Manipulation on Performance

Local passive heating, where heating modalities are applied to specific segments or muscles to increase muscle temperature (T_{mu}) seemingly functions as an ergogenic aid to exercise performance. Lower leg passive heating demonstrated improvements in contractile properties and dynamic performance, with electrically stimulated time to peak torque decreasing (i.e., indicating a faster time to peak torque) and voluntary dynamic force production increasing [4]. A review of passive heating literature highlighted a dose-response relationship between T_{mu} and jump height, with jump height increasing 2-5% for every 1°C increase in T_{mu} [5]. The associated improvements in performance occur primarily due to increases in power production and contractile function [25] rather than significant increases in muscle force [147,148]. Improvements in contractile function due to passive heating can be quantified by improvements in the rate of force/rate of torque development observed during voluntary and evoked contractions that commonly occur following passive heating [7,149]. Rodrigues et al. [7] observed increases in RTD, decreases in half-relaxation time (HRT) and electromechanical delay (EMD) when leg T_{mu} increased. Increased RTD suggests improved contractility of the muscles, while decreases in HRT and EMD suggest that muscles should be able to relax and be recruited at a faster rate during dynamic exercise to further improve performance.

Local passive cooling, where cooling modalities are applied to specific segments or muscles to decrease T_{mu}, has an adverse impact on exercise performance. Reductions in T_{mu} have been associated with reductions in maximal dynamic strength and muscular power [4,25,31]. Isokinetic knee extension strength, total work, and power have been shown to decrease at faster speeds (180, 300 and 400°·s^{-1}) following immersion of the leg in 12 °C water when compared to 35.5°C water [150]. Recently the local cooling of the forearm muscles was shown to decrease

maximal voluntary force production while altering motor unit contractile characteristics and recruitment patterns [151].Ultimately there is seemingly a 2-10% decrease in performance per 1°C decrease in muscle temperature [27], with vertical jump performance sharing a 2-5% decrement per 1°C decrease in T_{mu} [5]. It appears that 10+ minutes of passive cooling is enough to reduce performance through a decrease in muscle contractility [26]. While performance correlates with muscle temperature, such that heating improves performance and cooling hinders performance, this is seemingly only the case for dynamic power dominant efforts. There is an inverse relationship between muscle temperature and isometric muscular endurance, with increases in muscular endurance being reported during leg extension and finger flexion tasks [148,152].

2.3 Underlying Mechanisms

While not a focus of this study, it is important to understand the role of oxygen in the function of skeletal muscle function as its availability is a critical factor in determining exercise performance. The main role of O_2 in muscle is to provide a baseline for energy production, specifically during oxidative phosphorylation which produces the ATP that is essential for skeletal muscle contraction. Overall, skeletal muscle performance is determined by O_2 delivery to the muscles, the diffusion of O_2 into cells, and the utilization of O_2 by skeletal muscle [153]. In general, increasing in O_2 availability via hyperoxia improves performance [3,154–158], with the inverse being true with decreasing in O_2 availability via hypoxia [154,156,159–163], suggesting a linear relationship between systemic O_2 availability and exercise performance [3,156]. Additionally, 5 weeks of knee-extensor training significantly increased thigh blood flow and thigh O_2 delivery alongside improvements in performance [164], suggesting that increases in blood flow may function to improve performance. Therefore, any impact that thermal manipulation may have on the delivery of oxygen to the muscle may in theory directly impact performance.

For muscle to contract a series of biochemical and mechanical processes need to occur which are explained by the sliding filament theory and the propagation of an action potential. Propagation of an action potential down the nerve fibres to the motor end plate and through the muscle fibres signals the contraction of muscle, with any temperature mediated changes in nerve conduction velocity and of resting membrane potential potentially impacting skeletal muscle contractility. Additionally, the underlying biochemical processes that control actin-myosin cross-bridge formation are ultimately mediated by the release and reuptake of calcium (Ca^{2+}) in and out of the sarcoplasmic reticulum. Therefore, any temperature mediated alterations in the nervous and biochemical processes that underpin the contractile processes of skeletal muscle would in turn impact performance as well.

2.3.1 Muscle Temperature, Muscle Blood flow and Muscle Oxygenation

Muscle oxygenation is seemingly related to muscle temperature, with passive heating increasing muscle oxygenation and passive cooling decreasing it [165,166]. A comprehensive study by Koch et al. [166] investigated the effects of whole-leg heating, upper-leg heating only and lower-leg only heating on muscle blood flow (mBF) and oxygen saturation (rSO2, %) of the legs. While they demonstrated that whole leg heating significantly increased both upper-and-lower leg rSO2, by 16 ± 9% and 24 ± 9% respectively, the authors importantly demonstrated that the increases in rSO2 only occurred in the directly heated segments and not in the segments that were not heated [166]. During exercise muscle oxygen saturation (StO_2) decreases, but local thermal manipulation may impact O_2 dynamics during exercise [165]. In this study, participants performed a 2-minute typing task after a 1-hour application of passive heating or passive cooling. When compared to the control conditions passive heating significantly attenuated the exercise induced decrease in StO_2 (49.3 vs 68.1) while passive cooling increased the magnitude

of the StO_2 decrease (55.1 vs 48.4) [165].These findings support a correlation between muscle temperature and muscle tissue oxygenation such that an increase in T_{mu} may yield increases in muscle oxygenation [166–169] when the tissues are directly heated with passive cooling potentially having the opposite effect[170–172].

The effects of passive heating and passive cooling on muscle oxygenation may be in part due to the impacts of thermal manipulation on mBF. Local direct-passive heating where heating is applied only to specific segments, but not full-body passive heating, may significantly increase mBF [173]. This was demonstrated by Heinonen et al. [173], who performed a passive heating protocol where only the right-calf was heated (i.e., local passive heating) or full-body passive heating occurred with the left-calf being excluded from the heating. In response to the passive heating, which also yielded a ~ 4°C increase in muscle temperature, mBF increased by ~45 % in the directly heated calf while the excluded calf saw no changes in mBF despite a ~1°C increase in T_{core} with full-body heating [173]. Additionally, the increase in mBF due to passive heating may be proportional to the increase in T_{mu}, such that a larger increase in T_{mu} may lead to a larger increase in mBF [174]. By adopting a leg heating model where the whole leg, upper leg only or lower leg only was passively heating Koch Esteves et al. [166] not only furthered the work by Heinonen et al. [173] by demonstrating a causal link between mBF and local T_{mu}, but also that the increases in mBF lead to segment specific responses. Taken as a whole, these findings suggest that a passive heating mediated increase in mBF may serve one of the underlying mechanisms by which passive heating augments performance.

Three potential mechanisms may contribute to the increase in blood flow following passive heating. Firstly, T_{mu} increases may directly stimulate vasodilation within the muscle

microvasculature as a thermoregulatory response. This initial vasodilatory response occurs when temperature-sensitive receptors located in cutaneous sensory nerves detect heat resulting in the release of vasodilatory neurotransmitters [175–177]. This is then sustained through nitric oxide (NO) diffusing into the smooth muscle layer of the endothelium and stimulating their relaxation [175,178,179]. The increase in blood vessel diameter would decrease the resistance provided to blood flow, thus allowing for an increase in blood flow to the heated area [166,173]. Secondly, metabolic reaction rates increase with higher temperatures with the Q_{10} effect, and the elevated oxygen consumption by the heated tissues may in turn elicit a metabolically induced vasodilation. Finally, heating may elicit the release of ATP from red blood cells (RBCs), which in turn signals endothelium-dependent vasodilation [180–183]. When RBCs are heated, ATP is released into the extracellular space and broken down into adenosine, which subsequently binds to adenosine receptors and stimulates cAMP production and vessel wall relaxation. Increases in arterial ATP have been observed during heating, with strong correlations between arterial ATP plasma concentration, vascular conductance, and muscle temperature [174].

Exposure to the cold, whether through cold environments or by direct passive cooling, has the opposite effect and may elicit vasoconstriction. In a cold environment peripheral vasoconstriction occurs as a thermoregulatory response to shift blood from the peripheries to the core to maintain core temperature at ~37°C [184–187], resulting in decreases in mBF [188–190]. Relative to direct local passive cooling, 20 minutes of ice applied to the right forearm was sufficient to reduce radial artery blood flow by 48% [191]. While not a direct measure of mBF, forearm blood flow was reduced from 16 ± 6 L·min-1 to 8 ± 2 L·min-1 following the circulation

of ~13°C through Tygon tubes wrapped around the forearms for 25-minutes [151]. The effects of passive cooling on mBF varies between muscles as blood flow increased to the deep muscles and decreased to the superficial muscles in responses to water temperatures between 8-15°C [192]. During exposure to the cold, either in a cold environment or with direct local passive cooling, thermoreceptors in the skin detect the reduction in skin temperature and stimulate the sympathetic nervous system. The stimulation of the sympathetic nervous system drives the release of norepinephrine, neuropeptide-Y, and RhoA-ROCK to shift blood from the limbs to the core to attenuate heat loss from the peripheries[193]. During local cooling the local vasoconstriction response occurs in part by the activation adrenergic nerves [194], the of α_{2c}-adrenoceptors and the inhibition of the nitric oxide system (NOS) [195]. Additionally the exposure to cold may have a direct effect on the smooth muscle cells of the blood vessel walls which causes them to contract and narrow further [196,197], while endothelial cells may release potent vasoconstrictions such as endothelin in response to cold which may cause further vasoconstriction [198,199].

Beyond directly impacting mBF passive heating/cooling may impact muscle oxygenation, and subsequentially exercise performance, by altering the oxygen hemoglobin disassociation curve (ODC). The ODC describes the reversible binding of four molecules of oxygen to Hb [200], and is sensitive to temperature. At higher temperatures hemoglobin's affinity for oxygen decreases, meaning that hemoglobin (Hb) is more likely to unload oxygen (O_2) releasing it to the surrounding tissue causing a rightward shift in the ODC and increasing the availability of oxygen to be used by the surrounding tissue. Additionally increases in temperature lead to an increase in increase in O_2 consumption [174,201], indicating that an increase in temperature not only

leads to an increase in the release of O_2 to the surrounding tissue, but also the increased uptake and utilization of oxygen by skeletal muscle as well. Inversely, at lower temperatures hemoglobin's affinity for oxygen increases, meaning that Hb is less likely to unload O_2 to the surrounding tissues causing a leftward shift in the ODC and ultimately decreasing the amount of oxygen available to be used by the surrounding tissue [202]. Alongside the cold induced vasoconstriction and resulting decrease in oxygen availability the leftward shift in the ODC caused by cooling may contribute to a reduction in exercise performance that occurs with passive cooling.

2.3.2 Muscle temperature, Neuromuscular function and Skeletal Muscle Contractility

Temperature has a profound effect on the peripheral nervous system and in turn skeletal muscle contractility. The excitability of neurons is dependent on membrane potentials [203], with any potential increases or decrease in resting membrane potential (RMP) in theory making the neurons either more or less excitable. Mammalian RMP shares a linear relationship with temperature, such that RMP increases by 1.0-1.3 mV per °C as temperature increases and decreases by 1.1–1.4 mV per °C when temperature decreases [204]. While not large changes in magnitude, RMP deviations either side of the -70mV thermoneutral rest point means that either a larger or smaller impulse is required to cause full depolarization and therefore increasing or decreasing excitability[31,32]. The relative refractory period (RRF), which is the time between repeated depolarizations, is also temperature sensitive with a 20.09 ms time at 20°C but a 3.19 ms time at 35°C [30]; a shorter RRF means that the time between repeated depolarizations is shorter subsequentially decreasing the time between action potentials and ultimately contractions[31]. Muscle contractility may also be impacted by temperature, as nerve conduction velocity exhibits a high thermal dependence [205]. Nerve conduction velocity increases as nerve

temperature increases [28–30] and decreases with reduction in nerve temperature [31], in turn either leading to faster or slower contractions [32]. Cooling may have a larger impact on nerve conduction velocity than heating does, with a 10°C increase in forearm temperature increasing conduction velocity by ~10% [206] and a 10°C decrease in arm temperature decreasing conduction velocity by ~20% [205]. It appears the thermal effects of nerve conduction velocity occur due to changes in sodium channel kinetics, with faster sodium channel gating occurring with heating [207] and slower sodium channel gating occurring with cooling [208].

2.3.4 Muscle Temperature and Biochemical Processes

For muscle to contract the cross-bridging of myosin and actin needs to occur, and this process is heavily determined by the release and reuptake of calcium (Ca^{2+}) by the sarcoplasmic reticulum (SR) [209]. Increases in T_{mu} lead to the shortening of muscle contraction and relaxation characteristics [210–213] and is associated with faster release and reuptake of calcium [214,215]. It has been suggested that passive heating can increase intracellular Ca^{2+} and cause myoplasmic Ca^{2+} accumulation [216]. Passive heating has also been shown to increase intracellular Ca^{2+} by increasing myotube formation via Trpv1 channels in a temperature-dependent manner [217,218].Additionally, ryanodine receptors (RyR) release Ca^{2+} in response to the depolarization of the sarcolemma, however there is not enough literature to conclude whether or not passive heating also increases this method of Ca^{2+} release [219]. Decreases in T_{mu} lead to the lengthening of muscle contraction and relaxation characteristics [4,31], which may be in part with the slower release and reuptake of calcium as calcium release from the SR [220,221] and calcium sensitivity are reduced with cooling [222].

The rate of cross-bridge cycling is a critical determinant of skeletal muscle contractile velocity, and therefore directly impacts contractile force and RFD/RTD [209,223], with passive

heating and passive cooling increasing and decreasing cross-bridge cycling rate respectively. The changes in calcium cycling rate that occur with increases or decreases in temperature largely contribute to altered cross-bridge cycling rate. Passive heating leads to a decrease in half-relaxation time (HRT) [4,7,224] suggesting that Ca^{2+} reuptake through the SERCA channel is accelerated to facilitate the decreased recovery time [219]. In contrast, HRT is prolonged with cooling, with a Q_{10} effect of 1.7 demonstrated when cooling the first dorsal interosseus muscle from 35°C to 25°C [212] and a 132% longer HRT occurring during isometric contractions when the plantar flexors were cooled to 22°C [225]. With cooling, ATP is slower to bind to the myosin head causing a slower detachment of the myosin-actin cross-bridges [226], while SR ATPase activity is slowed which delays the re-uptake of calcium which in turn prolongs contraction time [220,225]. Ultimately, as cross-bridge cycling rate increases the frequency of myosin-actin power strokes increases, leading to a faster contraction velocity [227] and an increase in RTD/RFD. In theory during a time-constrained exercise task, such as a 30-second bout of maximal voluntary work, more work (e.g., more contractions completed) could potentially be performed when compared to a thermoneutral muscle. In theory, the opposite effect should be true with passive cooling, as a decrease in the frequency of myosin-actin power strokes would lead to a reduction in contraction velocity and in the RTD/RFD. A byproduct of this increase in Ca^{2+} cycling and cross-bridge cycling rate is an associated increase in ATP production. This increase occurs due to an increase in enzymatic activity, such as an increase in CK which turns ADP to ATP, and provides the system with enough ATP to maintain the increased rate of Ca^{2+} cycling [228].

2.4 Gaps in the Literature and Future Directions

While there is an extensive body of literature examining the effects of passive heating and

passive cooling on exercise performance, to our knowledge only a few studies that have looked at the effects of both passive heating and cooling on single-joint submaximal repeated dynamic performance. Isometric and isokinetic single-joint models are often used to assess neuromuscular capacity, and subsequently the effects of passive heating and cooling on single-joint exercise. However, these models are limited in their translation to muscular function in vivo and during sporting events. This is because isometric models do not account for the changes in the force-velocity and force-length relationships as the contractions do not include movement of the joint. Isokinetic models are dynamic in nature, addressing the highly specific nature of isometric performance, but are performed at pre-determined contraction velocities which limit the translation to 'real' performance where movement is often fully dependent on the individual rather than artificially constrained. To our understanding there is no single-joint exercise test that closely mimics the volitional and repeated dynamic contractile demands placed on muscle during movements such as sprinting or repeated jumping. As a result, it is unclear whether the widely reported impacts of passive thermal manipulation would translate to single-joint repeated submaximal dynamic performance. We believe there is therefore the need for a dynamic single-joint exercise test to be developed that assesses the neuromuscular capacity of skeletal muscle during continuous repeated efforts.

3. Objectives and Hypotheses

3.1 Objectives

The objectives of this study were to:

1. Determine the repeatability and variability of the proposed IPFT as a single-joint exercise test of the plantar-flexor muscles
2. Determine the sensitivity of the IPFT to local thermal manipulation

3.2 Hypotheses

The hypotheses of this study were:

1. Thermoneutral IPFT performance during Session 1 will be unchanged from IPFT-1 through IPFT-4, such that there will be no significant difference between measures of work completed (i.e., total work, average work, number of repetitions), torque (i.e., peak torque, average torque per contraction), power (i.e., average power) and contractility (i.e., RTD, time-to-peak torque).
2. Total work performed during the 30-second IPFT would be in relation to muscle temperature, such that passive heating would yield an increase in total work and passive cooling would yield a decrease in total work. The same response should also occur with the number of repetitions completed.
3. Muscular power (i.e., average power) and contractility (i.e., RTD and time-to-peak torque) will relate to muscle temperature such that it is improved by passive heating and inhibited by passive cooling.

4. Materials and Methods

4.1 Isotonic Plantarflexion Test

The isotonic plantarflexion test (IPFT) was designed to be a volitional maximal effort single-joint dynamic test of plantar flexors. The IPFT consisted of 30-seconds of repeated concentric plantarflexion and dorsiflexion with the dynamometer set to isotonic mode. Isotonic mode was selected to allow for full volitional control of contractile characteristics when compared to other dynamic modes such as isokinetic where contractile velocity is fixed. Ankle range of motion (ROM) during the IPFT was limited to a total of 35°, starting at 5° of dorsiflexion to the ending at 30° of plantarflexion. Experimental ROM was set within these limits as it allowed the plantar flexors to go through the range of motion where it has the most force-generating capacity without entering full range of motion to reduce the possibility of fatigue impacting subsequent performance [229,230]. Resistance during the plantarflexion portion of the IPFT was set as 70% of the average peak torque produced during the 2 pre-experimental isometric maximal voluntary contractions (MVCs, see 4.3.1 for further explanation) rounded to the nearest 5 or 10 Nm value. Resistance during the dorsiflexion portion of the IPFT was set at 0.5 Nm which was the lowest permissible resistance in order to prevent fatigue of the dorsiflexors from limiting plantarflexion performance during the IPFT. Prior to each IPFT participants were given 3 practice repetitions to familiarize the participants with the full experimental range of motion, while also 'warming' up the dynamometer to ensure that any potential lag between the movement of the motor axis and the application of force from the participants. Participants were instructed to "contract as hard as you can, as fast as you can, throughout the whole range of motion for the full 30-seconds" and were given a verbal 3-second countdown prior to the beginning of each 30-second bout. During the IPFT participants

provided standardized strong verbal encouragement throughout the IPFT that did not provide specific instruction so as to not impact performance. We validated the IPFT in 2 different sessions. Session 1 established if there was a familiarization effect, an effect of fatigue, and the repeatability of performance during the IPFT. Session 2 established the test-retest variability of the IPFT and the sensitivity of the IPFT to passive heating and passive cooling of the lower leg.

Neuromuscular Testing Set-Up - All neuromuscular testing (isometric and dynamic contractions) were performed on the right leg using a dynamometer (Biodex 3, Biodex Corp., USA). The seat and foot pedal of the dynamometer was adjusted for each participant to ensure that the right malleolus was aligned to the motor axis of the dynamometer, and that the participants were able to comfortably maintain 90° knee and hip angles and a flat foot on the foot pedal. The participants were secured to the dynamometer's chair with a cross-hip and 2 diagonal shoulder straps, while the upper leg was secured to a support cushion by a cross-quadricep strap to prevent the upper leg from contributing to performance. The foot was secured to the dynamometer foot pedal with a strap across the forefoot, with a second strap across the midfoot used to attempt to limit the amount of heel lift during plantarflexion.

4.2 Participants

Twelve healthy volunteers (9 males, 3 females, age: 24.3 ± 2.2 years; height: 1.7 ± 0.1 m, body mass: 79.8 ± 15.5 kg, right lower leg circumference: 38.5 ± 3.3 cm, right lower leg skin-fold thickness: 22.3 ±5.9 mm, 12 right-foot dominant), with no history of skeletal, neuromuscular, or circulatory disorders, contraindications to high intensity exercise and/or direct heating/cooling, not pregnant, and no recent history of musculoskeletal injury of the lower leg that may limit the range of motion or performance during repeated ankle plantar-and-dorsi-flexion were recruited from the university and community population. All volunteers were

informed about the nature, aims, and risks associated with the experimental procedure before giving their verbal and written consent. The study was approved by the Brock University Research Ethics Board (REB 23-325 CHEUNG) and conformed to the latest revisions of the Declaration of Helsinki.

4.3 Experimental Design

Participants visited the laboratory on two separate occasions separated by 72-hours to minimize fatigue and allow for sufficient recovery. During the first visit, participant's anthropometrics were recorded, and they performed the novel isotonic plantar flexion test (IPFT) 4 times to determine the familiarization effect and variability in performance and performed maximal voluntary contractions before and after the neuromuscular test batteries to quantify fatigue. During the second visit participants performed 3 IPFTs, first a thermoneutral condition (TN), followed by either a passively heated (Hot) or cooled (Cold) condition in a counterbalanced order to determine the sensitivity of the IPFT to thermal manipulation. Participants were instructed to refrain from vigorous physical activity and from consuming alcohol in the 24 hours preceding each visit and to avoid caffeine on the day of each visit. Participants were asked to maintain their regular dietary and exercise behavior outside of these instructions.

4.3.1 Session 1: Familiarization Effect and Repeatability of the IPFT

Figure 4.1 schematically outlines the protocol for the first visit, with the main objective of this first visit being to test the learning effect and repeatability of the IPFT. Upon arrival to the laboratory participant anthropometric measurements of height (cm), mass (kg) skin-fold thickness (mm) of the medial gastrocnemius using manual skinfold calipers (Harpenden, Bay International, United Kingdom) and circumference (cm) of lower leg (calf) at its widest girth

were recorded. Next participants were landmarked and instrumented and wrapped in Tygon tubing (for thermal manipulation of muscle temperature). Participants were seated in and fitted to the dynamometer and performed a 30-minute thermoneutral wash-in (see details below), pre-experimental maximal voluntary contractions MVCs), 4 IPFT (separated by 30-minutes of rest where the lower leg was maintained at TN), and post-MVCs.

Maximal Voluntary Contractions – Isometric MVCs were performed by the participants before the start of the IPFT battery and after completion of the IPFT battery to quantify the potential fatigue caused by the multiple IPFTs during each session. The MVCs were performed in the thermoneutral condition, which was achieved by a 30-minute wash-in period prior to starting the MVCs. The ankle was set to 20° of plantarflexion, the knee at 90° of flexion, and the dynamometer set to isometric mode. Participants were instructed to "contract as hard as you can, and to sustain your maximal effort for the whole time" prior to each MVC and were given a 3-second countdown prior to the beginning of each MVC. Researchers provided standardized strong verbal encouragement (i.e., "Push Push Push or Go Go Go") throughout the MVC that did not provide specific instruction as to not impact performance. Participants were not provided with any performance feedback (e.g., torque produced). If participants began the MVC prior to the end of the count down the attempt was not counted, and they rested for 5 minutes before re-attempting the MVC. The MVC battery consisted of 4 total isometric MVCs separated by 5-minutes of rest, 2 plantarflexion MVCs and 2 dorsiflexion MVCs. While we did not analyze dorsiflexion performance, we performed the MVCs to allow for the normalization of the tibialis anterior sEMG data. An average of the raw peak torque provided from the dynamometer during the 2 plantar flexion MVCs was used to determine the 70% resistance that

would be applied to plantarflexion during the IPFT.

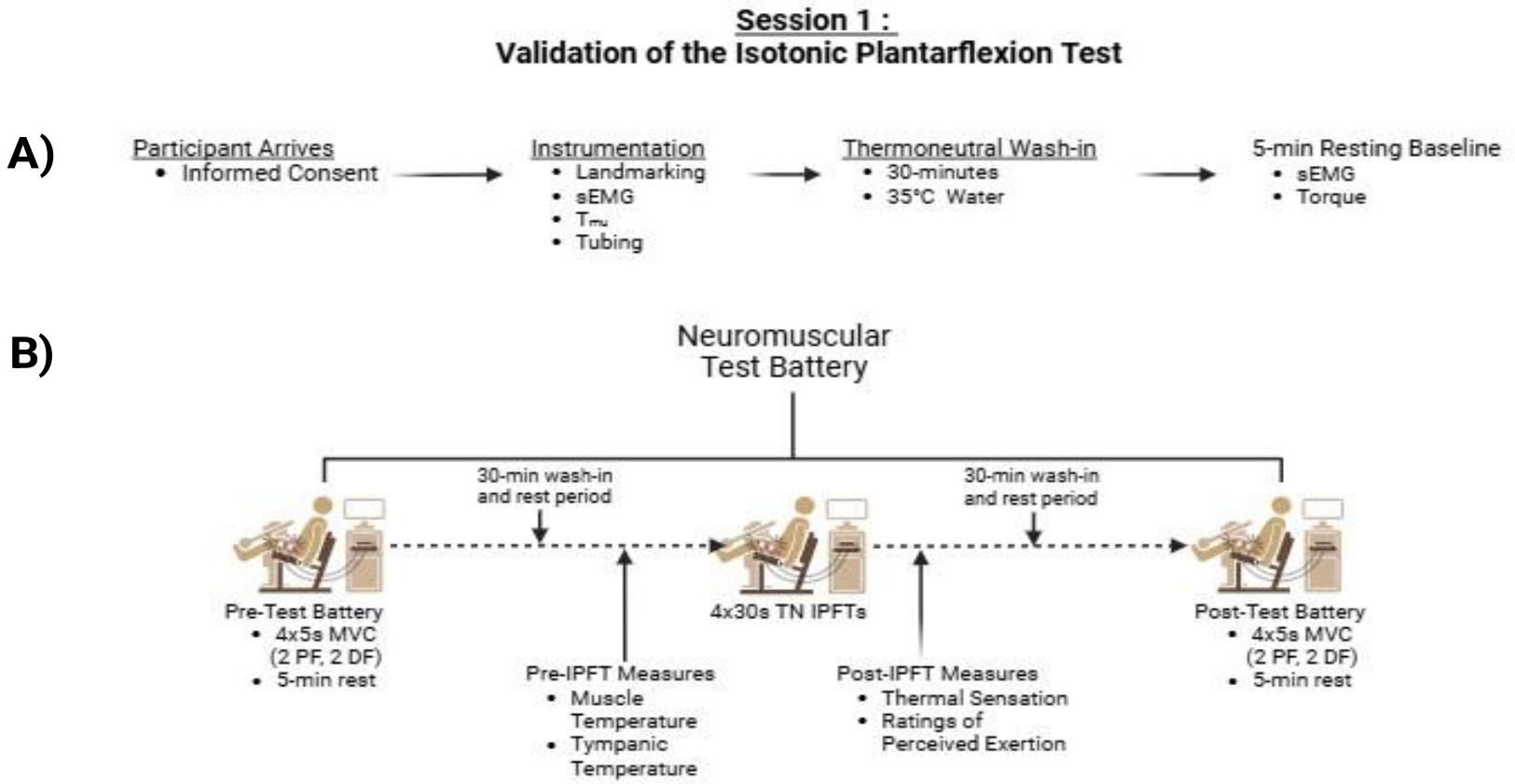

Figure 4.1:Outline of the protocol performed during Session 1. A) All of the events that occurred prior to the beginning of data collection, including the landmarking and instrumentation of surface electromyography (sEMG) and muscle temperature (Tmu). B) The neuromuscular test battery, consisting of pre-test and post-test maximal voluntary contractions (MVCs) for both plantarflexion (PF) and dorsiflexion (DF), and 4 bouts of the isotonic plantarflexion test (IPFT) with thermoneutral muscle temperature. Created in https://BioRender.com

4.2.2 Session 2: Sensitivity of the IPFT to Passive Heating and Cooling

Figure 4.2 schematically outlines the protocol for the second session. This session had two purposes: i) test the test-retest variability of the IPFT by comparing performance from Session 1 to the thermoneutral IPFT during this session, and ii) determine the sensitivity of the IPFT to passive heating and cooling of the muscles in the lower leg. Upon arrival participants were instrumented identical to Session 1. Following instrumentation, participants performed a 30-minute thermoneutral wash in, followed by pre-experimental MVCs, the TN IPFT, then either a Hot IPFT or Cold IPFT in a counterbalanced order, and a post-experimental MVCs. Each of the IPFTs were separated by a ~30-45-minute rest period rest (TN = 30.0 ± 0 minutes, Hot = 39.0 ± 2.8 minutes, 43.0 ± 2.9 minutes) where muscle temperature was manipulated to match the respective condition. Following the completion of the last IPFT, another 30-minute thermoneutral wash-in occurred prior to the post-experimental MVCs to return muscle temperature to the TN condition where at which point participants performed the post-experimental MVCs as they did in Session 1.

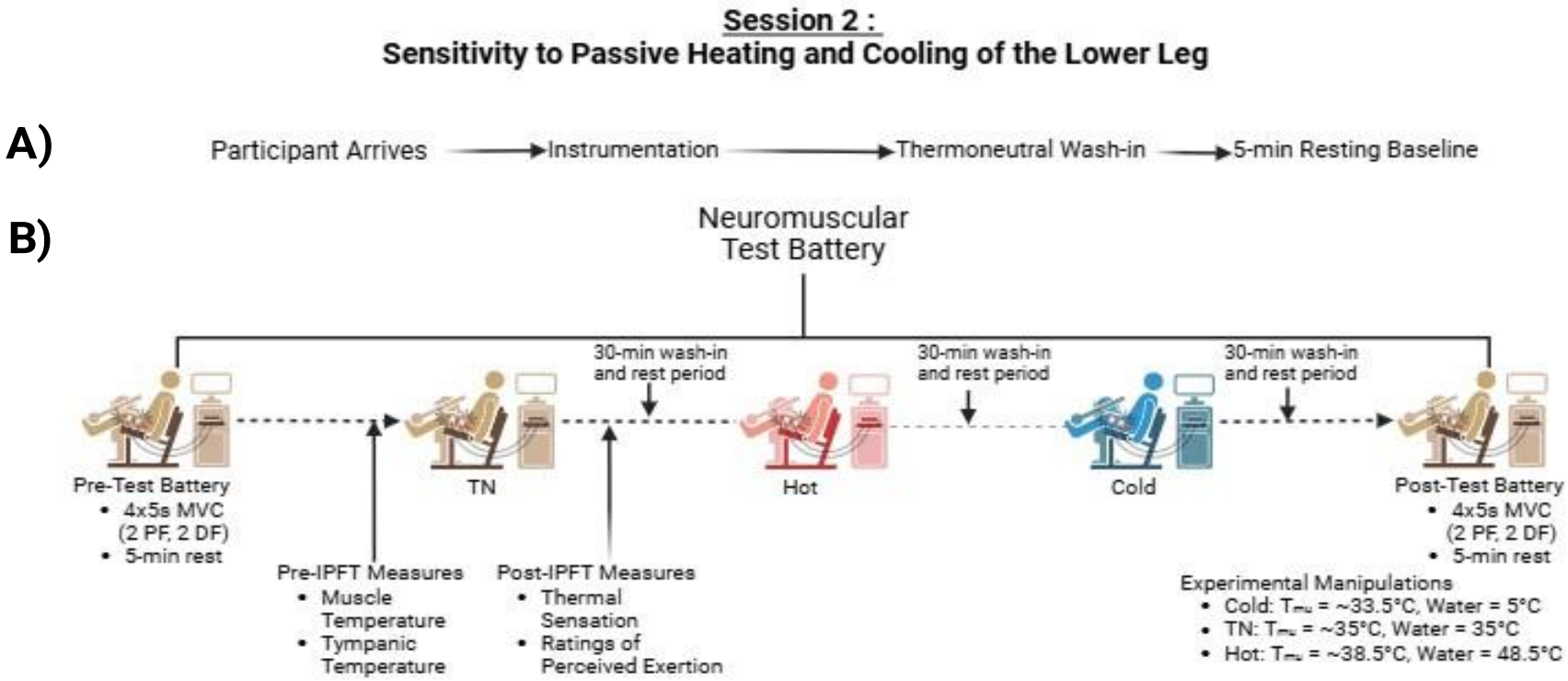

Figure 4.2: Outline of the protocol performed during Session 2. A) Pre-Neuromuscular testing processes as performed during Session 1 including the landmarking and instrumentation of surface electromyography (sEMG) and muscle temperature (T_{mu}). B) The neuromuscular test battery performed, consisting of pre-test and post-test maximal voluntary contractions (MVCs) for both plantarflexion (PF) and dorsiflexion (DF), and 3 bouts of the isotonic plantarflexion test (IPFT) in 3 different muscle temperatures. Created in https://BioRender.com

4.3.3 Equipment and Procedures

Instrumentation

Participants were seated with their right knee at 90° and the tibialis anterior (TA), medial soleus (M_{SOL}) and lateral soleus (L_{SOL}) muscle bellies were located through palpation while asking participants to plantarflex and dorsiflex as required. Next, the skin was shaved, abraded (Nuprep, Weaver and Company, USA), and cleaned with 70% isopropyl. The sEMG electrodes (Bagnoli Surface EMG Sensor, Delsys Inc., USA) were affixed to the surface of the skin on the M_{SOL} and TA muscles following SENIAM guidelines, which were marked with permanent marker (Figure 4.3) with adhesive double-sided tape with an interelectrode distance of 1 cm connected to Bagnoli EMG system hardware (Bagnoli EMG system, Delsys Inc., USA). The L_{SOL} was used to record T_{mu} and was landmarked using common anatomical landmarks and palpation. A permanent marker was used to mark the site for repeatability of the measure (Figure 4.4). Muscle temperature (°C) was measured using a hypodermic needle microprobe (MT-26/4HT, Physitemp Instruments, USA) plugged into a digital thermometer (Thermalert TH-5, Physitemp Instruments, USA). The muscle temperature probe was inserted 3 cm into the muscle belly of L_{SOL} and held in place for ~3-5 seconds to allow for the measurement to stabilize and was subsequently removed. During piloting, we confirmed that our landmarking was accurate and that the muscle temperature probes were accurately inserted into the lateral soleus via ultrasound imaging with the help of a trained technician (Figure 4.5). Tympanic body temperature was measured via a tympanic thermometer ($T_{tympanic}$, °C) inserted into the right ear of the participant. Temperature measurements were taken at the end of each wash-in period prior to performing the IPFT.

Figure 4.3: Landmarked sites for the surface electromyography (sEMG) sensors on the medial aspect of the soleus (MSOL) and the tibialis anterior (TA) muscles. Landmarking was performed by following SENIAM guidelines.

Figure 4.4: Landmarked window for site of recording muscle temperature (T_{mu}) with tubing wrapped around the lower leg.

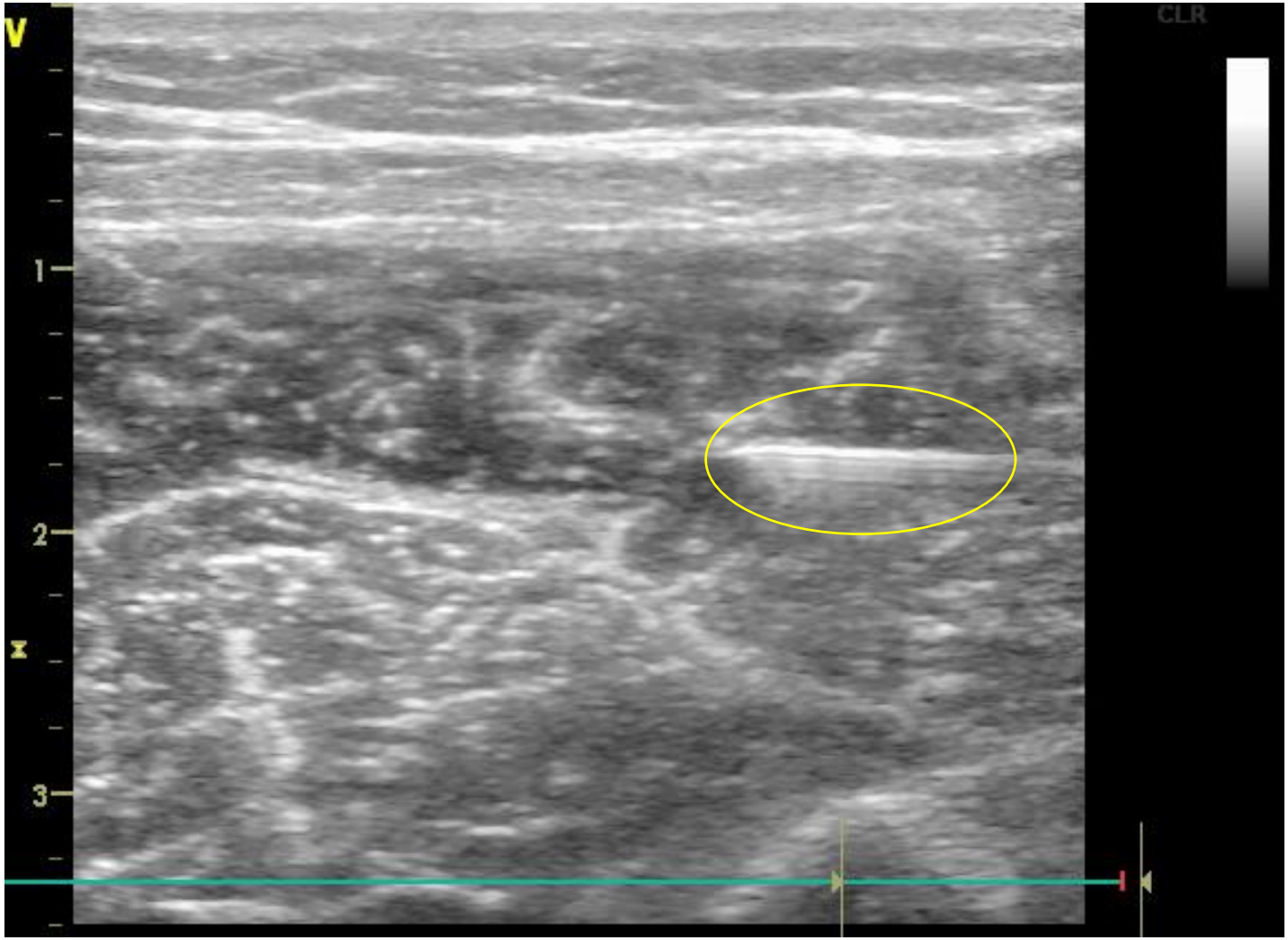

Figure 4.5:Ultrasound confirmation of the muscle temperature (T_{mu})probe in the soleus taken from the lateral aspect of the lower leg. Yellow circle is highlighting the T_{mu} probe (bright white line) inside the muscle.

Thermal Manipulations

The right lower leg was wrapped in 1/4-inch diameter vinyl tubing connected to a submersion pump (300508Z, BURCAM, Canada) with a flow rate of 5.3 $L \bullet min^{-1}$ submerged in a water bath controlled by a water circulator (MX-C, VWR North America, USA) (Figure 4.6). Prior to each IPFT, water was circulated for 30-min at ~35°C for thermoneutral (Session 1 = 35.0°C ± 0.1°C; Session 2 = 35.1°C ± 0.1°C) (TN, prior to all IPFTs in Session 1), ~48.5°C (Hot = 48.5°C ± 0.1°C), or ~5°C (Cold = 5.1°C ± 0.1°C). The thermoneutral temperature was selected as the standardized 'thermoneutral' starting temperature because when resting T_{mu} was compared to the average resting T_{core} it sits slightly lower (~35°C vs ~37°C) [14,231]. Muscle and tympanic temperature were taken prior to every IPFT. Water temperature was circulated at target temperature throughout the IPFTs. The experimental conditions were:

Thermoneutral Muscle (TN): In this condition, T_{mu} was maintained at thermoneutral (~36.6°C) by circulating 35°C water over the lower leg. This condition functioned as our control condition.

Hot Muscle (Hot): In this condition T_{mu} was elevated to ~38.3°C by circulating 48.5°C water over the lower leg for 30-minutes before the IPFT. This condition allowed us to assess the sensitivity of the IPFT to passive heating.

Cold Muscle (Cold): In this condition T_{mu} was decreased to ~33.3°C by circulating 5°C water over the lower leg for 30-minutes. This condition allowed us to assess the sensitivity of the IPFT to passive cooling.

Figure 4.6: The thermal manipulation set up with ¼ inch vinyl tubing wrapped around the lower leg during a thermoneutral wash-in period. Elastic bandage was also wrapped around the lower leg but was removed for this image to allow for clear view of the tubes.

4.3.4 Data Reduction and Processing

Performance Data - Each contraction over the 30-second IPFT was windowed and isolated into: full contraction (plantar flexion + dorsi flexion), plantar flexion only, and dorsi flexion only (see Figure 4.7 for conceptualization) offline in Matlab (MatLab R2024a, MathWorks Inc, USA). Raw torque (mV) was collected directly through the Biodex at 2000 Hz through EMGworks Acquisition (EMGworks Acquisition, Delsys Inc., USA) and then converted offline into newton meters (Nm) using the following equation: $Torque\ (Nm)\ =\ (V * 153.89)\ +\ z$, where *z* is the offset value which was calculated in MatLab based on the resting data recorded by the dynamometer during the resting baseline where the foot was affixed to it but no force was being applied. The converted data was then filtered with a 15 Hz low-pass Butterworth digital filter. Peak torque, average torque, average RTD, and # of contractions were calculated from the torque signal. Total work performed (J) was determined through the use of a trapezoidal function to calculate the areas under the curve of the rectified torque signal.

sEMG Data - The raw sEMG data was pre-amplified and filtered (bandwidth 20 – 450 Hz, gain = 1,000, sampling frequency 2000 Hz) by the Bagnoli EMG system and was recorded and stored using EMGworks Acquisition and Analysis software (Delsys Inc., USA). The raw sEMG data was then debiased, full-wave rectified, and low-pass filtered at 6 Hz using a dual passed second-ordered Butterworth filter. All sEMG data was normalized to activity recorded during the pre-experimental MVCs. Peak and mean sEMG activity of the TA and M_{SOL} during each IPFT in the following windows was determined through MatLab: a) throughout the full 30-second IFPT b) for the plantar-flexion only portion of each contraction (Figure 4.7).

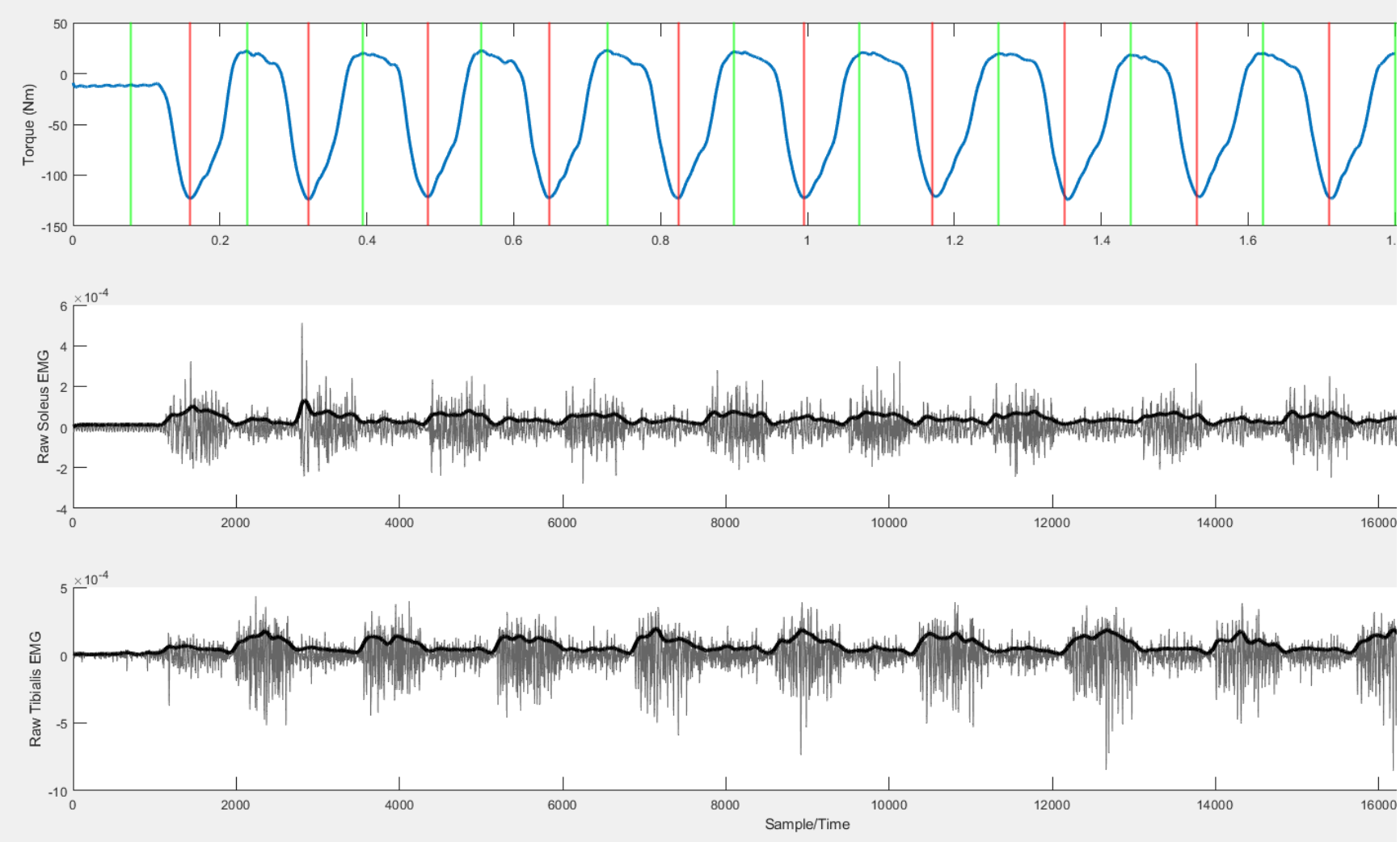

Figure 4.7: Visualization of the windows where Torque and sEMG data is being analyzed. Each segment of the contraction was windowed as follows for further analysis: A) Green-to-Green = Full Contraction, B) Green-to-Red = Plantarflexion only, C) Red-to-Green = Dorsiflexion only

4.3.5 Statistical Analysis

All physiological and performance variables are expressed as means ± SD and perceptual data expressed as median (quartile 1 – quartile 3). To analyze physiological and performance data from Session 1, 1 x 4 (IPFT-1 vs. IPFT-2 vs. IPFT-3 vs. IPFT-4) repeated measures ANOVAs were performed. To analyze the potential differences in average muscle activity during Session 1, 2 (muscle) x 4 (IPFT-1 vs. IPFT-2 vs. IPFT-3 vs. IPFT-4) repeated measures ANOVAs were performed. To compare variability between days, paired samples t-test were used to compare performance from IPFT-4 from Session 1 to TN from Session 2, and to compare experimental load from Session 1 to Session 2. To assess the consistency of our measures an intraclass correlation was run on all data from Session 1. To compare the variability, the % change in performance was calculated for all IPFTs compared to IPFT-1 and each IPFT compared to the previous IPFT in Microsoft Excel. To compare the effects of temperature, 1 x 3 Temperature (TN vs. Hot vs. Cold) repeated measures ANOVAs or linear mixed model repeated measures ANOVAs were performed (missing performance, neuromuscular, and EMG variables from 1 participant in TN due to data corruption). Data was assessed for normality through visual inspection of box and Q-Q plots and the Shapiro-Wilks test (not normal if $p < 0.05$). If normality was violated a non-parametric Friedman's ANOVA was used. The Greenhouse-Geisser correction was used for all tests. If necessary, pairwise comparisons were performed using a Bonferroni post-hoc test corrected for multiple comparisons. All statistical analyses were performed with IBM SPSS Statistics for Windows (version 28.0; IBM Corp., USA) and GraphPad Prism (V.8.3.0 GraphPad Prism, USA). Statistical significance was set at $p < 0.05$.

5. Results

5.1 Session 1 Thermal Manipulation and Perceptual Data

The thermal protocol was successful in maintaining pre-IPFT T_{mu} at a 'thermoneutral' temperature (average of 36.6 ± 0.3°C across IPFTs) without significantly altering $T_{tympanic}$ with no differences across the 4 IPFTs in Session 1 (Table 5.1). Participants perceived the temperature as 'neutral' with thermal sensation of 4 (4-4) prior to each IPFT bout with no difference across IPFT bouts ($p < 0.001$). There was a significant effect for ratings of perceived exertion ($p < 0.001$), where ratings did not differ between IPFT-1 at 16 (16-17) and IPFT-2 at 16 (16-18, $p = 0.581$), while ratings were higher in IPFT-3 at 18 (16-19, $p < 0.001$ vs IPFT-1, $p > 0.05$ vs IPFT-2) and IPFT 4 at 18 (16-19, $p < 0.001$ vs IPFT-1, $p > 0.05$ vs IPFT-2).

5.2 Session 1 Familiarization Effect, Fatigue Effect, and Variability in Performance

Peak torque during plantar flexion MVCs was not significantly different between the pre-test and post-test batteries (Figure 5.1-A, $p = 0.229$). Total and average work performed across the 4 IPFT bouts were not statistically different (Table 5.1, all $p > 0.05$). Furthermore, there were no differences (all $p > 0.05$) for # of repetitions completed, peak torque, average torque, time-to-peak-torque, or average rate of torque development (Table 5.1). While there was a significant main effect for average contraction time ($p = 0.019$), there were no significant pairwise differences (all $p > 0.05$). Average variability in IPFT performance compared to IPFT-1 across measures of work, torque and power ranged from -8.36 ± 11.72 % to 11.38 ± 20.67 % depending on the individual variable (see Table 5.3), while average variability when comparing each IPFT to the previous bout ranged from -2.81 ± 10.02 % to 3.94 ± 15.64 % depending on the individual variable (see Table 5.4). Average measures intraclass correlations ranged from 0.935 (0.841 - 0.979) to 0.996 (0.989 - 0.999) throughout Session 1 depending on the individual

variable (see Table 5.6, all $p < 0.05$). Muscle activity (sEMG) was not different across Session 1 in measures of peak activity, total activity, average activity relative to the pre-test MVCs (all $p > 0.05$). Additionally, there were no significant differences in average activity relative to the pre-test MVC between the soleus and tibialis anterior for both full contractions and only during plantarflexions (both $p > 0.05$).

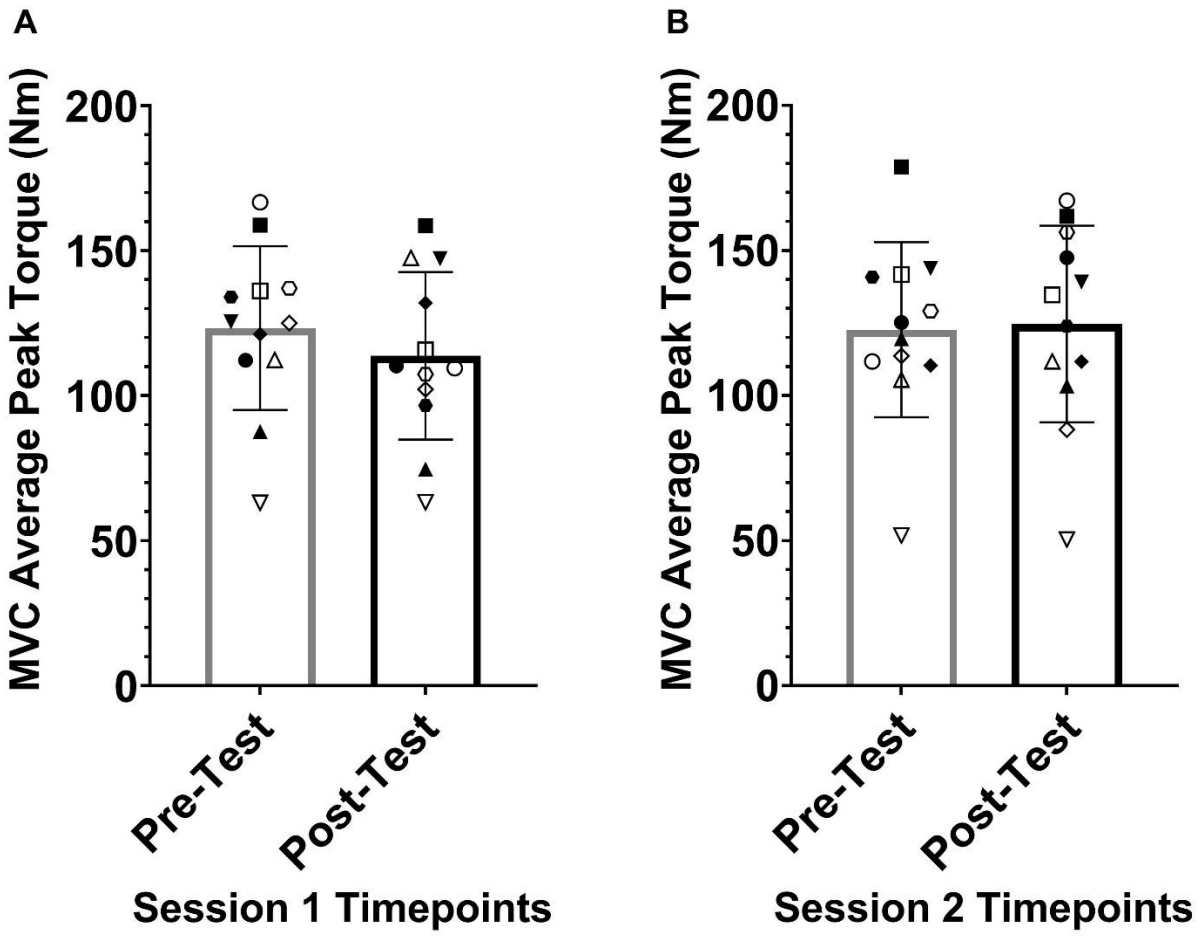

Figure 5.1: Pre-Test and Post-Test average peak torque during session 1 and session 2. Data is presented as mean ± standard deviation.

Variable	IPFT-1	IPFT-2	IPFT-3	IPFT-4	Main effect p value
T_{mu} (°C)	36.4 ± 0.4	36.6 ± 0.4	36.9 ± 0.2	36.6 ± 0.3	0.274
$T_{tympanic}$ (°C)	36.7 ± 0.2	36.6 ± 0.4	36.5 ± 0.3	36.6 ± 0.3	0.274
Total Work (J)	1026.0 ± 312.7	1119.0 ± 280.8	1110.0 ± 331.8	1105.0 ± 306.3	0.219
Average Work (J)	39.23 ± 6.47	40.58 ± 8.60	36.68 ± 8.45	38.78 ± 8.20	0.409
Average Power per Contraction (W)	35.58 ± 10.48	38.62 ± 9.56	38.03 ± 10.81	38.12 ± 10.40	0.238
Repetitions (#)	26 ± 6	28 ± 5	28 ± 5 [1]	28 ± 4	0.005
Peak Torque (Nm)	132.7 ± 21.3	133.4 ± 22.4	134.1 ± 21.8	133.9 ± 22.5	0.646
Average Torque (Nm)	45.3 ± 11.9	42.8 ± 8.9	42.0 ± 8.3	41.9 ± 8.0	0.074
Average Contraction Time (s)	1.20 ± 0.30	1.10 ± 0.19	1.06 ± 0.18	1.07 ± 0.17	0.019 *
Time-to-Peak-Torque (s)	0.6 ± 0.1	0.6 ± 0.1	0.6 ± 0.1	0.6 ± 0.1	0.077
Average Rate of Torque Development ($Nm \cdot s^{-1}$)	566.1 ± 120.9	573.4 ± 102.4	603.1 ± 113.0	599.6 ± 111.7	0.108

*Table 5.1: Thermal and performance results of the IPFT from session 1 presented as mean ± standard deviation. Significant main effects are identified with a *. Post hoc pairwise comparisons using a Bonferroni correction factor can be interpreted as: [1] different from IPFT-1, [2] different from IPFT-2, [3] different from IPFT-3, [4] different from IPFT-4. Significance set at $p \leq 0.05$.*

5.3 Session 1 IPFT-4 vs Session 2 TN

Day-to-Day IPFT performance remained stable, indicating we were successful in developing a repeatable exercise test. In comparison between IPFT-4 from Session 1 to TN of Session 2, there was no significant difference in T_{mu}, $T_{tympanic}$, total work, average work, average power per contraction, repetitions completed, peak torque, or average torque (Table 5.2, all $p > 0.05$). Additionally, there were no significant differences in time to peak torque or in average rate of torque development (Table 5.2, all $p > 0.05$). Absolute plantarflexion resistance in Session 1 was significantly different than resistance in Session 2 (Session 1: 98.9 ± 16.8 Nm vs Session 2: 105.0 Nm ± 18.3 Nm, $p = 0.04$). Significant average measures intraclass correlations ranged from 0.874 (0.564 - 0.964) to 0.945 (0.749 - 0.985) throughout when comparing IPFT-4 to TN depending on the individual variable (see Table 5.8, all $p < 0.05$); average measures intraclass correlation was insignificant for average torque 0.653 (- 0.291 - 0.907, $p = 0.055$).

Variable	Session 1 IPFT-4	Session 2 TN	Main effect p value
T_{mu} (°C)	36.6 ± 0.3	36.5 ± 0.3	0.234
$T_{tympanic}$ (°C)	36.6 ± 0.3	36.6 ± 0.4	0.902
Total Work (J)	1105.0 ± 306.3	11146.0 ± 343.6	0.429
Average Work (J)	41.63 ± 11.08	38.78 ± 8.28	0.111
Average Power per Contraction (W)	38.12 ± 10.40	39.99 ± 12.12	0.268
Repetitions (#)	28 ± 4	27 ± 3	0.176
Peak Torque (Nm)	132.8 ± 23.3	136.8 ± 24.4	0.416
Average Torque (Nm)	41.7 ± 8.4	44.5 ± 7.0	0.190
Time-to-Peak-Torque (s)	0.58 ± 0.1	0.57 ± 0.1	0.514
Average Contraction Time (s)	1.07 ± 0.17	1.08 ± 0.12	0.777
Average Rate of Torque Development ($Nm \cdot s^{-1}$)	250.7 ± 49.9	262.0 ± 62.8	0.176

*Table 5.2: Thermal and performance measures compared between IPFT-4 from Session 1 and TN IPFT from Session 2 using a paired samples t-test presented as mean ± standard deviation. A * indicates a significant difference. Significance set at $p \leq 0.05$.*

Variable	IPFT-2 vs IPFT-1	IPFT-3 vs IPFT-1	IPFT-4 vs IPFT-1	Session 1 Grouped Average Variability
Total Work (J)	12.62 ± 20.00 %	11.19 ± 24.00 %	10.34 ± 19.00 %	11.38 ± 20.67 %
Average Work (J)	3.74 ± 14.00 %	- 1.21 ± 14.00 %	- 1.20 ± 12.00 %	0.44 ± 12.99 %
Average Power per Contraction (W)	11.32 ± 18.00 %	9.26 ± 21.00 %	9.13 ± 18.00 %	9.90 ± 18.45 %
Repetitions (#)	8.03 ± 13.00 %	11.33 ± 13.00 %	11.31 ± 16.00 %	10.23 ± 14.71 %
Peak Torque (Nm)	0.50 ± 3.00 %	1.15 ± 4.00 %	0.89 ± 4.00 %	0.85 ± 3.49 %
Plantarflexion Peak Torque (Nm)	0.58 ± 3.00 %	1.26 ± 4.00 %	0.96 ± 3.00 %	0.93 ± 3.53 %
Average Torque (Nm)	-3.79 ± 10.00 %	-5.50 ± 10.00 %	5.86 ± 10.00 %	-5.05 ± 9.62 %
Time-to-Peak-Torque (s)	-3.55 ± 9.00 %	-6.03 ± 7.00 %	-2.53 ± 8.00%	-4.04 ± 8.01 %
Average Contraction Time (s)	- 6.36 ± 11.00 %	- 9.80 ± 12.00 %	- 8.91 ± 12.00 %	- 8.36 ± 11.72 %
Average Rate of Torque Development ($Nm \cdot s^{-1}$)	4.88 ± 10.00 %	7.67 ± 9.00 %	4.88 ± 10.00 %	5.83 ± 9.50 %

Table 5.3:Variability in IPFT performance when compared to IPFT 1 in Session 1. All data is presented as mean ± standard deviation.

Variable	IPFT-2 vs IPFT-1	IPFT-3 vs IPFT-2	IPFT-4 vs IPFT-3	Session 1 Grouped Average Variability
Total Work (J)	12.62 ± 20.00 %	- 1.09 ± 12.0 %	0.30 ± 10.0 %	3.94 ± 15.64 %
Average Work (J)	3.74 ± 14.00 %	- 4.47 ± 8.0 %	0.56 ± 9.0 %	- 0.06 ± 10.92 %
Average Power per Contraction (W)	11.32 ± 18.00 %	- 1.65 ± 11.0 %	0.87 ± 11.0 %	3.51 ± 14.56 %
Repetitions (#)	8.03 ± 13.00 %	3.35 ± 7.00 %	-0.15 ± 5.00 %	3.74 ± 9.58 %
Peak Torque (Nm)	0.50 ± 3.00 %	0.65 ± 3.00 %	-0.22 ± 3.00 %	0.31 ± 2.75 %
Plantarflexion Peak Torque (Nm)	0.58 ± 3.00 %	0.69 ± 3.00 %	-0.26 ± 3.00 %	0.33 ± 2.79 %
Average Torque (Nm)	-3.79 ± 10.00 %	-1.69 ± 4.00 %	-0.31 ± 5.00 %	-1.93 ± 6.66 %
Time-to-Peak-Torque (s)	-3.55 ± 9.00 %	-2.07 ± 9.00 %	3.99 ± 9.00%	-0.54 ± 9.27 %
Average Contraction Time (s)	- 6.36 ± 11.00 %	- 3.35 ± 10.00 %	1.28 ± 7.00 %	- 2.81 ± 10.02 %
Average Rate of Torque Development ($Nm \cdot s^{-1}$)	4.94 ± 10.00 %	3.29 ± 11.00 %	-2.27 ± 9.00 %	-1.99 ± 10.55 %

Table 5.4:Variability in IPFT performance when compared to the previous IPFT in Session 1. All data is presented as mean ± standard deviation.

Variable	Session 1 Thermoneutral Variability (%)	Hot vs TN % Change	Cold vs TN % Change
Total Work (J)	3.94 ± 15.64 %	- 1.57 ± 12.24 %	- 39.85 ± 12.00 %
Average Work (J)	- 0.06 ± 10.92 %	- 5.03 ± 10.50 %	-24.49 ± 8.00 %
Average Power per Contraction (W)	3.51 ± 14.56 %	- 0.93 ± 11.52 %	- 38.00 ± 7.00 %
Repetitions (#)	3.74 ± 9.58 %	3.65 ± 5.56 %	-16.39 ± 5.00 %
Peak Torque (Nm)	0.31 ± 2.75 %	1.17 ± 1.62 %	-4.19 ± 1.00 %
Average Torque (Nm)	-1.93 ± 6.66 %	-2.18 ± 5.67 %	-2.13 ± 9.00 %
Time-to-Peak-Torque (s)	-0.54 ± 9.27 %	-2.78 ± 4.86 %	23.90 ± 13.00%
Average Contraction Time (s)	- 2.81 ± 10.02 %	3.94 ± 15.64 %	3.94 ± 15.64 %
Average Rate of Torque Development ($Nm \cdot s^{-1}$)	-0.54 ± 9.27 %	- 2.43 ± 7.12 %	22.64 ± 6.00 %

Table 5.5: Mean variability in Session 1 IPFT performance and % change in IPFT performance during Session 2 compared to TN. All data is presented as mean ± standard deviation. Participant 8's cold data was excluded due to being a systemic outlier which affected all statistical outcomes.

Variable	Average Measures ICC	Lower Bound	Upper Bound	Sig
Total Work (J)	0.960	0.904	0.987	< 0.001
Average Work (J)	0.956	0.893	0.945	< 0.001
Average Power per Contraction (W)	0.962	0.908	0.988	< 0.001
Repetitions (#)	0.967	0.921	0.989	< 0.001
Peak Torque (Nm)	0.996	0.989	0.999	< 0.001
Plantarflexion Peak Torque (Nm)	0.996	0.989	0.999	< 0.001
Average Torque (Nm)	0.972	0.932	0.991	< 0.001
Time-to-Peak-Torque (s)	0.967	0.920	0.989	< 0.001 < 0.001
Average Contraction Time (s)	0.934	0.841	0.979	< 0.001
Average Rate of Torque Development ($Nm \cdot s^{-1}$)	0.966	0.919	0.989	< 0.001

Table 5.6:Average measures of intraclass correlation in IPFT performance across Session 1. 95% confidence interval was used.

5.4 Session 2 Thermal Manipulation and Perceptual Data

The thermal protocol successfully altered peripheral muscle temperature without significantly altering $T_{tympanic}$. The thermal manipulations prior to each IPFT significantly ($p < 0.001$) changed soleus muscle temperature, with T_{mu} increasing following passive heating (38.3 ± 0.2°C) and significantly decreasing following passive cooling (33.3 ± 1.2°C, Figure 5.2A, all $p < 0.001$). Tympanic temperature prior to the IPFT was not different ($p = 0.061$) across the 3 temperature conditions (Figure 5.2B). Thermal sensation was significantly altered by the thermal manipulations ($p < 0.001$) and was 4 (4-4) in TN, increasing to 6 (6-6) in Hot ($p =0.014$ versus TN), and decreasing to 1 (1-2) in Cold ($p < 0.001$ versus TN and Hot). Ratings of perceived exertion were statistically significant ($p = 0.048$), however, pairwise comparisons revealed no differences (all $p > 0.05$) between TN (16 (16-18)), Hot ((18 (17-18)) or Cold (18 (17-19)).

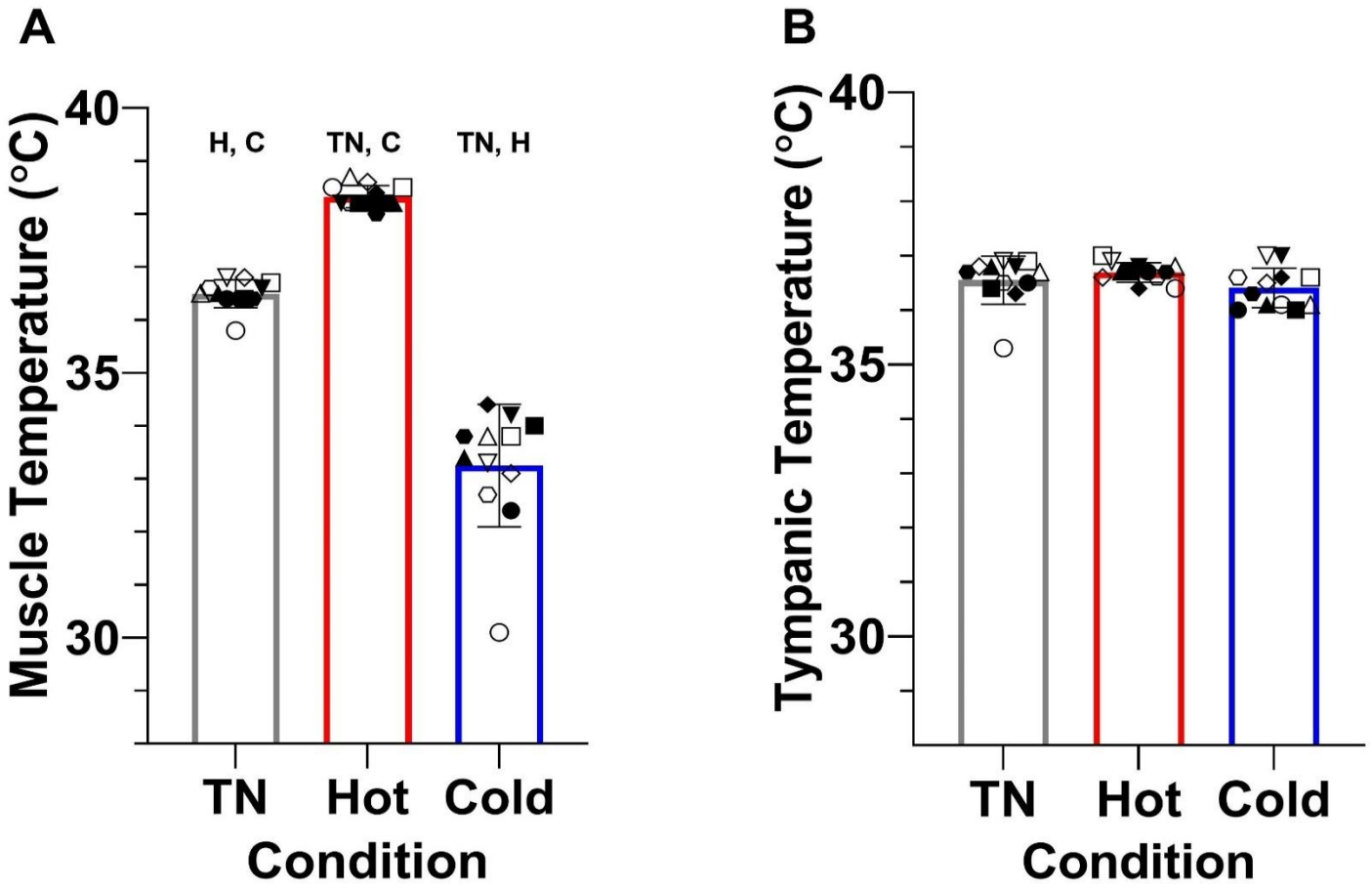

Figure 5.2: Soleus (A) and tympanic (B) temperature responses to thermal manipulations during Session 2 presented as mean ± standard deviation. If significant, pairwise comparisons can be interpreted as: TN different from TN, H different from Hot, C different from Cold. Significance set a $p \leq 0.05$

5.5 Session 2 Contractile properties

We excluded participant 8's data from all Cold results as they were a systemic outlier which skewed results. The thermal manipulations had a differential effect in altering muscle contractile properties indicative of changes in local muscle temperature. Temperature impacted average contraction time where there was no difference between TN (1.08 ± 0.12 s) or Hot (1.05 ± 0.13 s, $p = 0.825$), while it was significantly longer in Cold (1.33 ± 0.09 s, $p < 0.001$ vs both). Temperature also impacted time to peak torque (Figure 5-3A, $p < 0.001$), with no difference between TN (0.57 ± 0.07 s) or Hot (0.56 ± 0.08 s, $p = 0.862$); however, it was significantly longer in Cold (0.71 ± 0.18 s, $p < 0.001$ vs both). Similarly, cooling significantly affected concentric average rate of torque development (Figure 5.3B, $p < 0.001$), with no difference between TN (262.0 ± 62.8 $Nm \cdot s^{-1}$) and Hot (268.0 ± 65.7 $Nm \cdot s^{-1}$, $p = 0.784$) and decreased in the Cold (196.7 ± 49.9 $Nm \cdot s^{-1}$, $p < 0.001$ vs both). Measures of sEMG were taken on a subset of participants (N=6). Temperature statistically altered total soleus muscle activity (Table 5.6, $p = 0.032$), however, post-hoc comparisons revealed no significant differences across conditions (all $p > 0.05$). Temperature affected total tibialis anterior muscle activity (Table 5.6, $p = 0.038$) with post-hoc comparisons revealing that total activity was significantly higher in the Cold compared to Hot ($p = 0.023$ vs Hot) but not TN ($p = 0.169$), with no difference between Hot and TN ($p = 1.00$). Temperature affected average soleus muscle activity relative to activity during the pre-experimental isometric MVCs (Table 5.6, $p = 0.003$) with post-hoc comparisons showing that average activity was significantly higher in the Cold when compared to the Hot ($p < 0.001$). Similarly, temperature affected average tibialis anterior muscle activity relative to the pre-experimental isometric MVCs (Table 5.6, $p = 0.010$) with post-hoc comparisons identifying an increase in average activity in the Cold when compared to

the Hot ($p < 0.001$), with no difference between Cold and TN, nor Hot and TN (both $p > 0.05$). Additional sEMG activity variables are presented in Table 5.7.

Variable	TN	Hot	Cold	Main effect p value
Sum SOL sEMG	4163.7 ± 866.8	3565.7 ± 773.5	5536.0 ± 1577.1	0.034
Sum TA sEMG	3213.4 ± 2435.8	3381.9 ± 929.3 [C]	4887.3 ± 1723.7 [H]	0.038
Peak SOL sEMG	194.2 ± 30.9	175.1 ± 47.2	272.6 ± 132.3	0.069
Peak TA sEMG	147.5 ± 88.7	169.5 ± 149.5	194.2 ± 30.9	< 0.001
Avg Peak SOL sEMG	142.3 ± 24.6	116.6 ± 27.7 [C]	231.8 ± 78.8 [H]	0.024
Avg Peak TA sEMG	110.0 ± 77.5 [C]	109.0 ± 24.7 [C]	201.8 ± 69.6 [TN, H]	0.002
Avg % MVC SOL sEMG	60.4 ± 10.7	50.3 ± 17.6 [C]	80.4 ± 24.4 [H]	0.003
Avg % MVC TA sEMG	45.5 ± 29.3	46.4 ± 9.9 [C]	69.8 ± 18.1 [H]	0.011
Concentric Avg % MVC SOL sEMG	71.0 ± 17.5	58.8 ± 8.1 [C]	98.6 ± 33.6 [H]	0.007
Concentric Avg % MVC TA sEMG	44.8 ± 32.8	42.8 ± 14.0 [C]	65.1 ± 23.4 [H]	0.010

Table 5.7: Soleus (SOL) and tibialis anterior (TA) surface electromyography (sEMG) measures from session 2. Pairwise comparisons can be interpreted as: [TN] different from TN, [H] different from Hot, [C] different from Cold. Significance set at $p \leq 0.05$. All sEMG measures were normalized to activity recorded during the pre-experimental MVCs.

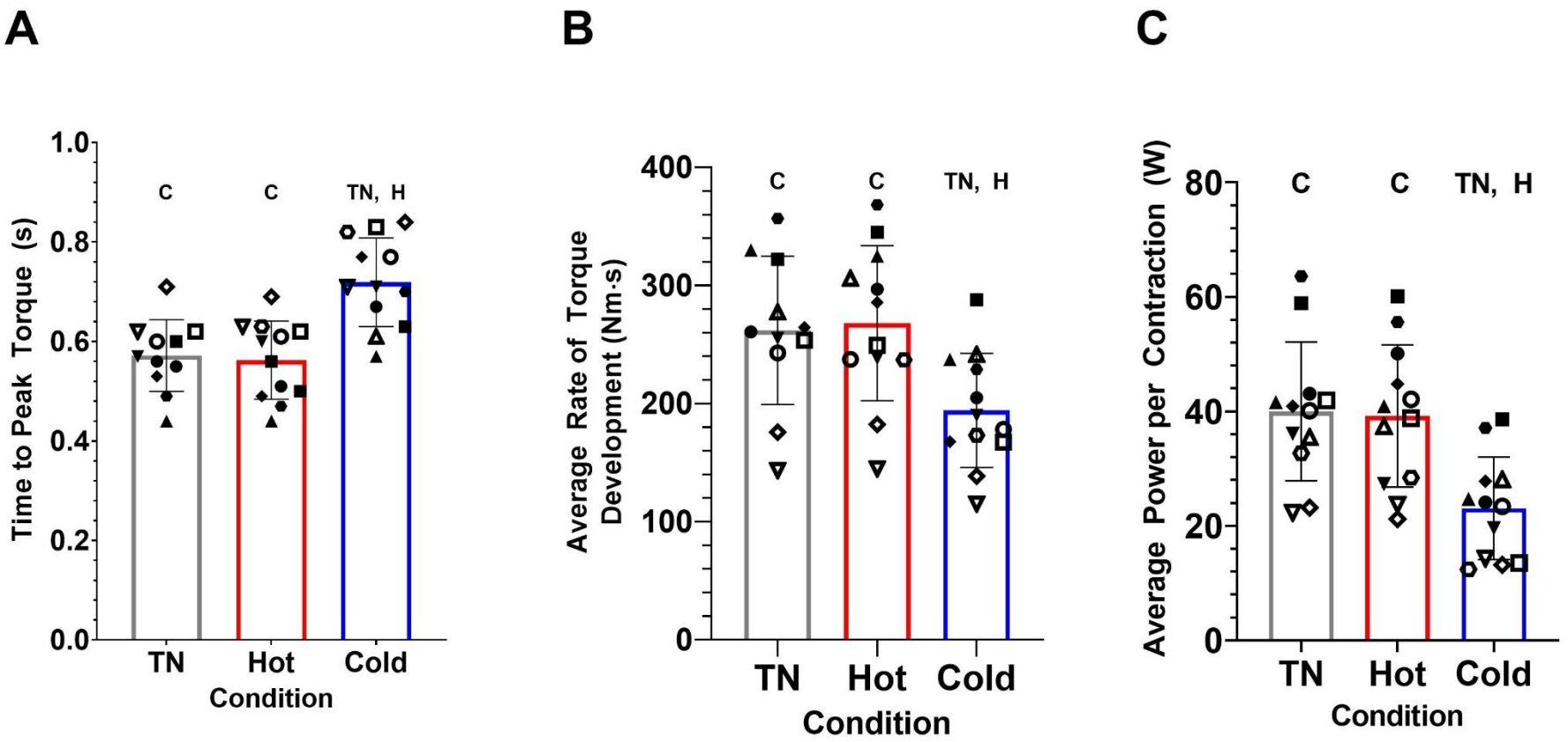

Figure 5.3: Time to peak torque (A) average rate of torque development (B) and (C) average power per contraction during session 2. If significant, pairwise comparisons can be interpreted as: TN different from TN, H different from Hot, C different from Cold. Significance set at $p \leq 0.05$

5.6 Session 2 Neuromuscular Capacity

We excluded participant 8's data from all Cold results as they were a systemic outlier which skewed results. The thermal manipulations had a differential effect in altering neuromuscular capacity. Temperature had a main effect on average power (Figure 5.3C, $p < 0.001$), with no differences between TN and Hot (TN: 39.99 ± 12.12 W, Hot: 39.20 ± 12.41 W, $p > 0.05$) while Cold led to a significant reduction (Cold: 23.94 ± 8.84 J, $p < 0.001$ vs both). Temperature significantly affected total work performed across the whole 30-second IPFT bouts (Figure 5.4-A, $p = 0.670$); average work per contraction was also significantly lowered by Cold (TN: 41.63 ± 11.08 J, Hot: 39.29 ± 10.48 J, Cold: 31.61 ± 9.86 J, $p < 0.001$ vs both) with no difference between TN and Hot ($p = 0.210$). There was a temperature effect ($p < 0.001$) for # of repetitions completed (Figure 5.4-C), where individuals completed fewer repetitions in Cold (22.6 ± 2.7 repetitions) compared to TN (27.5 ± 3.0 repetitions) and Hot (28.3 ± 3.5 repetitions, both $p < 0.001$), with no differences between TN and Hot ($p = 0.307$). Temperature had a differential effect on peak torque (Figure 5.5A, $p < 0.001$), with post-hoc comparisons revealing that peak torque was the highest in Hot (138.7 Nm ± 23.3 Nm, $p = 0.031$ vs TN, $p < 0.001$ vs Cold) when compared to TN (136.8 ± 24.4 Nm) and Cold, and was the lowest at Cold (130.4 ± 24.4 Nm, both $p < 0.001$). Temperature did not significantly affect average torque (Figure 5.5-B, $p = 0.372$, TN: 44.5 ± 7.0 Nm, Hot: 43.7 ± 6.6 Nm, Cold: 44.6 ± 8.0 Nm). Temperature had a differential effect on average peak plantarflexion torque (Figure 5.5-C, $p < 0.001$), which was lower in Cold (125.4 ± 23.0 Nm) compared to TN (131.0 ± 24.4 Nm) at TN and Hot (132.2 ± 22.9 Nm, both $p < 0.001$), but not between TN and Hot ($p = 0.677$). Average peak torque from the isometric plantarflexion MVCs was not significantly different between the Pre-test battery and Post-test battery (Pre: 112.7 ± 30.2 Nm vs Post: 124.7 ± 33.9 Nm, $p = 0.771$).

Variable	Average Measures ICC	Lower Bound	Upper Bound	Sig
Total Work (J)	0.925	0.740	0.978	< 0.001
Average Work (J)	0.908	0.679	0.973	< 0.001
Average Power per Contraction (W)	0.935	0.775	0.981	< 0.001
Repetitions (#)	0.913	0.678	0.977	< 0.001
Peak Torque (Nm)	0.881	0.558	0.968	0.001
Plantarflexion Peak Torque (Nm)	0.876	0.538	0.967	0.001
Average Torque (Nm)	0.653	-0.291	0.907	0.055
Time-to-Peak-Torque (s)	0.918	0.694	0.978	< 0.001
Average Contraction Time (s)	0.874	0.564	0.964	< 0.001
Average Rate of Torque Development ($Nm \cdot s^{-1}$)	0.945	0.794	0.985	< 0.001

Table 5.8:Average measures of intraclass correlation between IPFT-4 from Session 1 to TN from Session 2. 95% confidence interval was used.

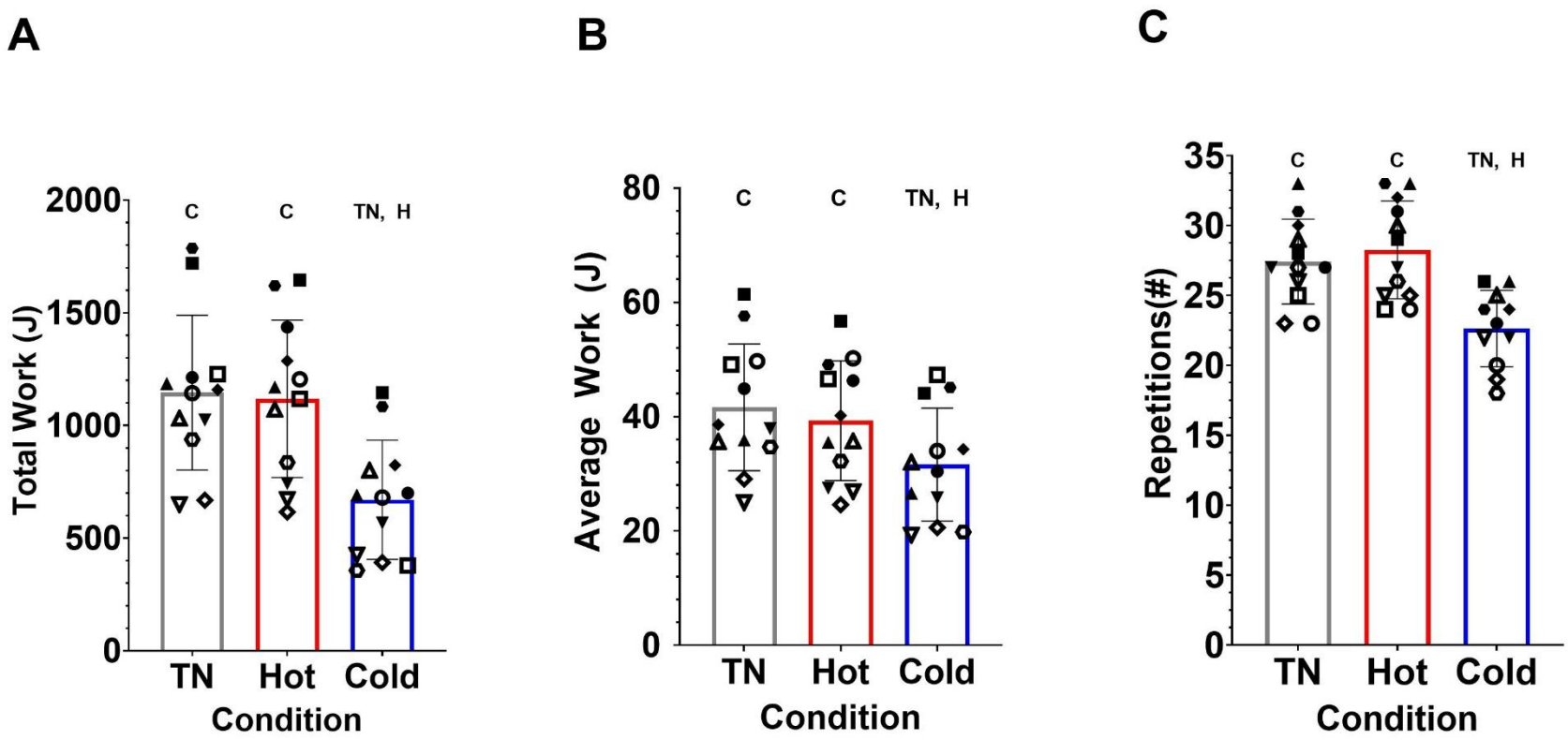

Figure 5.4: A) Total work, B) Average work and C) Repetitions completed during Session 2. If significant, pairwise comparisons can be interpreted as: TN different from TN, H different from Hot, C different from Cold. Significance set at $p \leq 0.05$

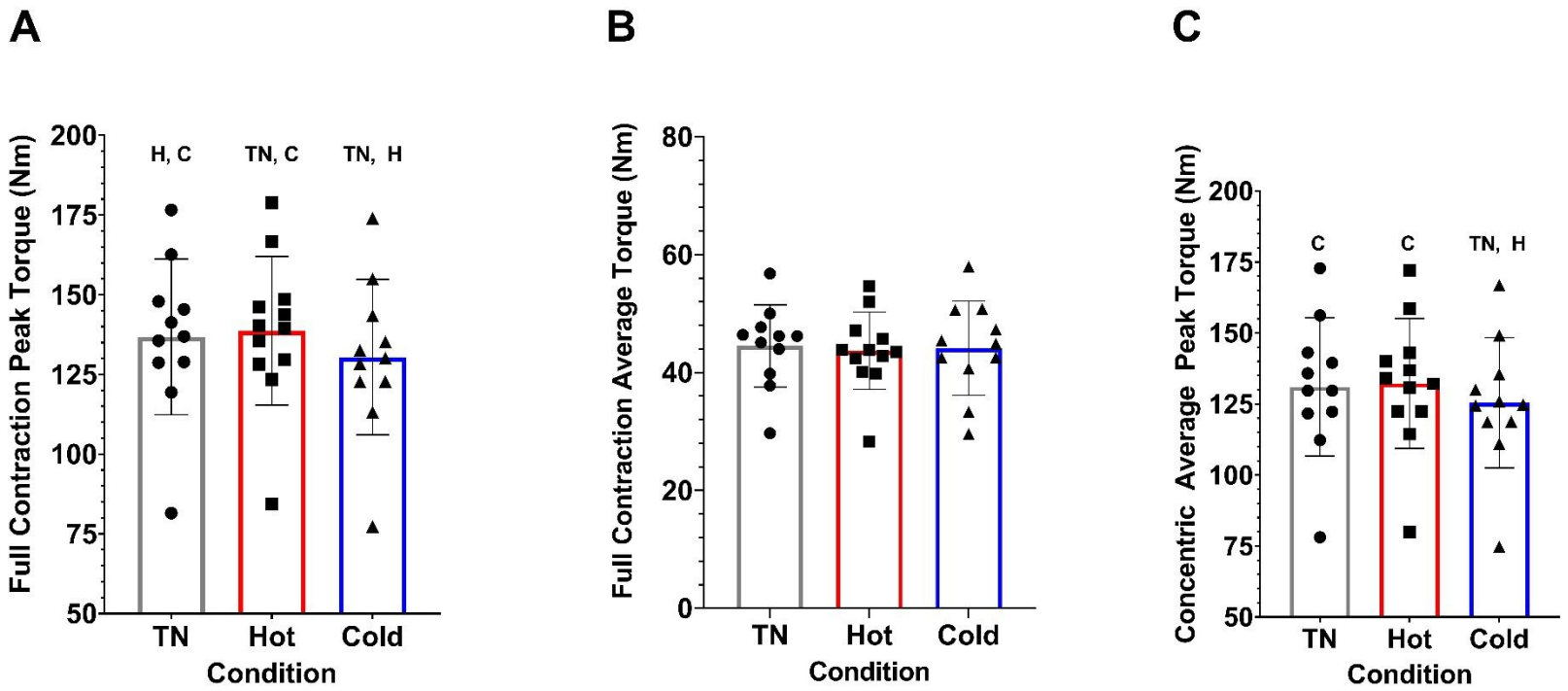

Figure 5.5: Peak torque (A), Average peak torque (B) and Concentric average peak torque (C) during session 2. If significant, pairwise comparisons can be interpreted as: TN different from TN, H different from Hot, C different from Cold. Significance set at $p \leq 0.05$

6. Discussion

In most cases, sport performance is based on repeated submaximal contraction-relaxation cycles (e.g., running) rather than maximal or sustained single contractions, which have been traditionally used to study mechanisms of neuromuscular capacity. Therefore, we sought to establish the novel IPFT as a single-joint dynamic exercise test to assess neuromuscular capacity/function during submaximal repeated contractions. It was important for the IPFT to be a single-joint test, as whole-body models are confounded by various psychophysiological factors and do not isolate the function of skeletal muscle. In Session 1, we found no differences in performance between the 4 IPFT bouts indicating minimal familiarization effects. Additionally, there were no differences in MVC performance pre and post the 4 IPFTs, indicating minimal cumulative fatigue. Importantly, the IPFT was repeatable across 72 h, where there was no difference in IPFT performance or neuromuscular capacity between IPFT-4 and TN in Session 2. In Session 2, we tested the sensitivity of the IPFT to changes in local muscle temperature in order to validate the IPFT against stressors that typically affect neuromuscular capacity and exercise performance. Contrary to our hypothesis, heating T_{mu} to ~38.3°C led to no change in performance or neuromuscular capacity compared to TN. Cooling T_{mu} to ~33.3°C decreased IPFT performance through decreasing work performed, average power, peak torque, average rate of torque development, and the number of repetitions completed while increasing time-to-peak torque and average contraction time. Contrary to our hypothesis, despite impairments in Cold there were no differences in total work compared to TN. In addition to developing the IPFT, these findings highlight that local passive cooling can negatively impact 30 s submaximal repeated single-joint dynamic performance.

6.1 Session 1 – Validity of the IPFT

We aimed to develop a single-joint dynamic test to isolate the plantar flexors while mimicking the muscular demands exhibited in sporting situations, which is more often reliant on rapid and repeated dynamic force production and relaxation, rather than maximal or sustained isometric contractions. Existing isometric single-joint models which yield highly joint angle-specific results [8–12] are not a suitable tool for assessing repeated dynamic submaximal contractions as they have poor correlation relative to dynamic movements like sprinting [232,233], change of direction [234], or jumping [235–237]. While isokinetic models are dynamic in nature, and in turn correlate to dynamic movements such as jumping [235], the fixed contraction velocity limits its ability to truly replicate the muscular demands of movements like sprinting or repeated jumps. Instead, the IPFT affords full control of contraction characteristics such as movement speed and force applied by the participants to better resemble repeated dynamic movements, while isolating single-joint muscle groups to allow for interventions to study peripheral neuromuscular function.

The IPFT was designed to isolate the plantar flexor movement, and specifically the soleus muscle. To bias the soleus, the knee was positioned at 90° of flexion to reduce contribution of the gastrocnemius [238–240], while the upper-body and thigh were secured to the chair of the dynamometer to minimize the contribution of other muscles. While participants actively performed repeated plantar-and-dorsi-flexions during the IPFT, only the plantar flexion movement phase (~70% MVC) was loaded, and the dorsiflexion phase was unloaded (0.5 Nm) to further bias the soleus and minimize fatigue. In TN in Session 2, the average relative EMG of the soleus was 71.0 ± 17.5%, while the tibialis anterior was 44.8 ± 32.8% during plantar

flexion. Other actions of the tibialis anterior include foot inversion and stabilization of the foot arch which may have contributed to EMG activation. While the greater average soleus EMG activity was not statistically significant between muscles, this is likely due to a small sample size for EMG data (N = 6). The combination of i) the nature of the movements performed, ii) the controls implemented to bias the soleus during plantarflexion, iii) only loading the concentric phase of the plantarflexion, alongside the higher but non-significant average EMG of the soleus suggests that we were successful in loading the plantar flexors without significantly loading the tibialis anterior. Future research implementing the IPFT is required to further validate the EMG activity of each muscle throughout the phases of each repetition.

For a test to be effective so that it can be utilized across a diverse pool of participants, it should have a rapid familiarization curve to reduce the amount of time needed to learn and re-learn the protocol. We observed no difference in work performed, average power per contraction, torque produced, average rate of torque development or number of repetitions completed across the 4 IPFTs (see Table 5.1), suggesting that there was no familiarization effect. The familiarization curve of the IPFT is less than other widely adopted exercise physiology tests. For example, cycling-based time-to-exhaustion tests need 3-5 familiarization trials to fully learn the protocol depending on the fitness and experience of participants [241], while the Wingate requires at least 3 full bouts to fully learn [242,243]. Studies using isokinetic dynamometry has previously implemented 3 familiarization repetitions prior to starting data collection [244,245], but have been suggested to require 1 familiarization session to limit practice-based improvements and increase reliability [246,247]; subsequent familiarization sessions further increase the reliability during isokinetic knee flexion and extension [248]. The lack of a significant

familiarization curve relative to other adopted tests, along with our choice of recreationally active rather than highly trained individuals, and that workload is relative to an individual's MVC suggests that minimal skill is needed with the IPFT. Therefore, in comparison to whole-body models of repeated contractions like sprinting and jumping which have an underlying technical component that impacts performance, the IPFT may allow for the assessment of dynamic neuromuscular function across various populations. Ideally, future studies can use a single familiarization IPFT to yield stable and repeatable performance.

An ideal neuromuscular test should allow for multiple test points in a single session without fatigue impacting performance. To this end, we compared the performance across the 4 IPFTs in Session 1 and IPFT-4 in Session 1 compared to TN in Session 2 and found no difference across all bouts in total work, average work, average power per contraction, number of repetitions completed, peak torque, average torque, time-to-peak-torque, average contraction time and average rate of torque development (Table 5.1, Table 5.2). There appeared to be minimal cumulative fatigue between bouts as there was no difference in peak torque from the 2 plantarflexion MVCs performed before and after the 4 IPFTs in Session 1 and the 3 IPFTs in Session 2 (Figure 5.1). However, RPE values in IPFT-3 and IPFT-4 were greater than in IPFT-1 and IPFT-2, which may indicate some development of perceptual fatigue. A good test should be repeatable with minimal variance across multiple bouts in a single session and/or day-to-day. In Session 1, the IPFT was repeatable with excellent reliability (all average measures ICC values ≥ 0.934, Table 5.6) with a maximum average variability of 3.94 % when comparing each IPFT in Session 1 with IPFT-1 (Table 5.3), and a maximum average variability of 11.38 % when comparing each IPFT in Session 1 with the previous IPFT (Table 5.4) across key performance

variables; the maximum average variability observed in the IPFT is similar to the expected variability found in isokinetic measurements [249]. Similarly, we observed no significant difference in performance when comparing IPFT-4 to TN in Session 2 (Table 5.2), suggesting that performance is stable across multiple sessions. The IPFT has exhibited similar intraclass correlations as the widely adopted isometric and isokinetic single-joint exercise models [250], further suggesting that it matches the needed reliability to be adopted. Collectively, these findings demonstrate that the novel IPFT falls in-line with other widely adopted models, and in turn may be a valid, reliable, and repeatable test of submaximal single-joint dynamic performance of the plantar flexors. Future research is needed to correlate IPFT performance to standard single joint models (e.g., isometric and isokinetic contractions). This approach would aid in validating the IPFT and understand the scope in which the IPFT can be effectively applied to study neuromuscular capacity and power.

6.2 Session 2 – Sensitivity to Local Thermal Manipulation

A neuromuscular test should ideally be sensitive to major stressors that are known to alter neuromuscular performance. Therefore, we examined the effects of passive heating and cooling on IPFT performance. Previous literature has demonstrated that passive heating may improve muscular strength, power, and contractility by 2-5% for every 1°C rise above resting muscle temperature (34-36°C) in isometric and dynamic contractions [4,5,25]. Contrary to our hypotheses (Chapter 3.2, hypotheses 2 and 3) only peak torque was significantly affected by passive heating when compared to TN in Session 2, indicating that the IPFT may not be sensitive to passive heating up to a muscle temperature of 38.3°C. Outside of peak torque, our null results when compared to other literature may be in part due to the magnitude of increase in muscle temperature, as the relationship established by Racinais & Oksa [5] was done with studies that

increased muscle temperature ≥ 39°C. Additionally, those studies occurred with active warming of the whole body rather than isolated passive heating of an isolated muscle [5], which introduces potentially confounding central and non-temperature mediated mechanisms [251–254]. Notably, other literature implementing acute and chronic passive heating of muscle between 37.3-38.6 °C have also demonstrated no improvements in maximal torque or rate of torque development during evoked and voluntary knee extensions during both isometric and isokinetic knee extensions [255–257]. This suggest that the null performance results in Hot are in line with other studies only heating T_{mu} within the ~38.3°C range used in the current study, demonstrating the IPFT is not sensitive to the same heating stimulus. Additionally, the IPFT is a 30-second submaximal test and as such may have influenced its sensitivity to passive heating; this may be the case because most dynamic efforts determined by muscular power in sporting situations are < 30-seconds (e.g., vertical jumps are performed in >1-second, average adult 40-meter sprint times are < 8-seconds). This is further exemplified by Bergh and Ekblom [25] who identified peak torques occurring for 0.1 - 0.2 ms during dynamic efforts and in 2 - 4 seconds during isometric efforts. Therefore, to test the sensitivity of the IPFT to heat, future studies are needed to determine i) if there is a passive heating T_{mu} threshold (i.e., $T_{mu} \geq 39.0$°C), ii) if the combination of passive increases in muscle and core temperature is required, and/or iii) the effects of active warm-up to improve IPFT performance. This approach would be able to isolate the separate and combined heat-related central and peripheral temperature-mediated changes in IPFT performance.

Passively cooling muscle has been demonstrated to reduce muscular power [4,25,168,169,258] and dynamic torque production during single-joint isokinetic contractions at velocities ranging from

30 to 240°·s^{-1} [25,141,255,259]. Similarly, passively reducing muscle temperature to 33.3 ± 1.2°C (range: 30.1 - 34.4°C) significantly impaired IPFT performance where there were decreases in total work, average work, average power per contraction, average rate of torque development, number of contractions completed, alongside increases in both time-to-peak torque and average contraction time. These findings not only extend the findings of previous literature by demonstrating that passive cooling inhibits repeated submaximal dynamic performance but also demonstrates the sensitivity of the IPFT to passive cooling. The decreases in work and power are likely due to fewer but longer contractions occurring with cooling. When comparing the change in performance between TN and Cold to the average variability in performance in Session 1 we consistently observed a significantly greater % change with passive cooling than occurred in Session 1 (see Table 5.5), further supporting that the IPFT is sensitive to cold manipulation. Although not statistically significant likely due to a small sample size (N = 6), we observed greater sEMG activity in Cold (Table 5.7); these findings are in line with literature which has demonstrated that higher threshold motor units are recruited earlier to compensate for the reduction in neuromuscular capacity with cooling [260], meaning that for any given work load a greater number of motor units are recruited when compared the thermoneutral performance [151,225,261–263]. Collectively, muscle cooling appears to make repeated dynamic submaximal (~70% MVC) contractions more challenging compared to TN and impairs performance. Future studies may want to increase the total duration of the IPFT (e.g. 45-60 seconds) to determine if changes in total work occur beyond the current 30-second time limit used in the current study, or if fatigue becomes a greater determinant of performance with the increased time.

6.3 Strengths, Limitations and Methodological Considerations

We performed the 2 pre-and-post experimental plantarflexion MVCs at 20° of

plantarflexion, whereas future implementations of the IPFT protocol should perform the plantarflexion MVCs in 5-15° of dorsiflexion instead. This is because the optimal joint angle for torque production during plantarflexion is within this range [229,230,264], while we artificially reduce the torque producing capabilities when placing the angle at 20° of plantarflexion. Similarly, we only performed 2 pre-experimental plantarflexion MVCs without the use of the twitch interpolation technique. Previous literature has demonstrated that the plantarflexors, specifically the soleus, are difficult for full activation to occur during voluntary contractions [265,266]. Future applications of the IPFT should use a combination of more pre-experimental plantarflexion MVCs as well as the interpolated twitch technique in order to ensure that the MVCs used to calculate the isotonic load are truly maximal efforts. In turn while we assumed that plantarflexion was performed against 70% of the mean peak torque performed during the pre-experimental MVCs the load may have been much lower due to a combination of the methodological errors outlined above. Finally, while we selected a 70% resistance for the IPFT to ensure that the 30-second bouts were high intensity but submaximal, this choice may have inhibited the ability of muscle to produce maximal power given that optimal resistance for power production occurs at ~20-30% maximal strength. As a result, future implementations of the IPFT that are chiefly concerned with muscular power should set isotonic load during plantarflexion at a lower percentage of pre-experimental average peak torque.

The ankle range of motion during the IPFT was set to a total of 35° (5° dorsiflexion through 30° plantarflexion) rather than performing the test within each participant's full volitional range of motion. We selectively limited ankle range of motion to i) standardize the range of motion so that our results were not confounded by functional differences between participants, and ii) to

reduce the development of fatigue, specifically in the dorsiflexors as the test was primarily concerned with plantarflexion performance. However, we were unable to fully eliminate heel lift from occurring during the plantarflexion despite strapping the foot to the dynamometer foot plate with both forefoot and midfoot straps. As a result each contraction may not have been occurring within our defined range of motion, and likely impacted the performance of the plantarflexors during the IPFT given the effect of joint angle on torque production [229,230,264]. Future applications of the IPFT may want to use stiffer strapping or additional straps to secure the foot in order to prevent heel lift to ensure the ankle is moving within the defined range of motion. While we instructed participants to complete each contraction through the full range of motion and provided practice repetitions prior to each IPFT bout where participants went through the full range of motion to ensure they knew the feeling of a full contraction, a main parameter of the IPFT was to provide full volitional control of contraction characteristics to the participants. As a result of this, participants may not have completed the full 35° range of motion during each contraction which would in turn impact outcomes relative to measures of torque, rate of torque development, time to peak torque and power. Future applications of the IPFT may want to consider methods that would either ensure participants complete the full range of motion, or a completed range of motion cut off to include and exclude partial repetitions from being counted. While this is a major methodological consideration, partial range of motion contractions often occur during in-vivo/sporting situations which is why we afforded full volitional control of each contraction to the participants. Due to this design, our findings may not be generalizable to performance where full plantarflexion is achieved such as during calf raises, jumping or sprinting. Furthermore, we did not utilize any isometric or evoked

contractions with temperature manipulations and therefore cannot determine changes in muscle contractility (e.g. motor unit recruitment patterns, electromechanical delay, or half-relaxation time). Additionally, the vinyl tubing was used to manipulate temperature which may have compressed the muscle, where compression has been demonstrated to increase both muscle blood blow and repeated sprint cycling performance [267]. However, the vinyl tubing was not adjusted throughout the protocol and therefore compression would be the same throughout each session allowing comparison within our design.

Surface heating and cooling can lead to a wide variability in deep muscle temperature, which is due to differences in muscle morphology (i.e., shape and size) and subcutaneous adiposity [268]. For example, 20 minutes of cooling the rectus femoris with icepacks only yielded a significant change in muscle temperature 1 cm into the muscle but not at depths of 2 or 3 cm [269]. Both our TN and Hot manipulations had a consistent and narrow T_{mu} range between participants of 35.8 - 36.8°C (~1.0°C) and 38.0 - 38.7°C (~0.7°C), respectively, whereas Cold had a greater T_{mu} range between participants (30.1 -34.4°C, ~4.3°C). This supports the inter-individual variability in cooling rates of both local muscle temperature [270–272] as well as whole body cooling [273–275]. Future studies may benefit from the use of continuous intramuscular muscle temperature monitoring so that muscle temperature can be manipulated to specific thresholds (e.g., T_{mu} = 39.0 °C, 30 °C etc.) rather than controlled by time to better understand the effects of muscle temperature on IPFT performance and neuromuscular capacity.

6.4 Future Directions

The present study has laid the foundation for future research to adopt the IPFT model, as we have tested the familiarization effect, variability, fatiguability, repeatability, along with its sensitivity to temperature. Future research is needed to compare IPFT performance against other

dynamic single-joint exercise models such as isokinetic models or dynamic power tests such as jumping to validate the novel test against already adopted methods. We anticipate that the IPFT will better correlate to tests that are based on the faster contraction velocities compared to slower velocity contractions [235].

While the current study concluded that Hot had no effect on IPFT performance when compared to TN, future research is needed to test muscle temperatures ≥ 39°C to determine if there is a muscle temperature threshold before passive heating impacts IPFT performance. Additionally, future research is needed implementing passive heating protocols that increase muscle and core temperature concurrently (e.g., waist-high water immersion) or with active warm-ups to determine whether IPFT performance exhibits the same relationship with temperature as demonstrated in literature [5].

With local changes in muscle temperature there are also changes in muscle blood flow and oxygenation, for example heating increases vasodilation to increase muscle blood flow, oxygen saturation, and oxygen unloaded [166,200]. Inversely, cooling causes vasoconstriction which decreases muscle blood flow, muscle oxygenation, and decreases oxygen unloading [151,202]. Previous evidence has shown that the use of hyperoxic gas can improve thermoneutral performance [3] and 20 km time-trial performance in the cold with a core temperature decrease of Δ0.5°C [129]. Therefore, a logical next step would be to test the separate and combined effects of passive muscle cooling and hyperoxia to determine if increased oxygen availability can mitigate the cold-related decrements in IPFT performance. Additionally, to our knowledge there is no literature directly investigating the separate effects of passive heating and hyperoxia during single-joint dynamic performance which could better test the separate and combined effects

increasing oxygen delivery (i.e., increased muscle blood flow from heating) and/or oxygen availability (i.e., oxygen saturation from hyperoxia) on performance. Therefore, future studies can use the IPFT to investigate the roles of temperature, muscle blood flow and oxygen availability on performance.

6.5 Conclusions

In summary, we implemented a novel IPFT to study dynamic neuromuscular capacity and power and its sensitivity to thermal manipulation. In Session 1, we determined that there was no familiarization effect or fatigability in performing 4 IPFT bouts in a single session. We also determined that the average variability when comparing each IPFT in Session 1 to IPFT-1, which ranged from 0.44 to 11.38% depending on the variable, and that the IPFT is repeatable across multiple days. Furthermore, we determined the differential effect of temperature on IPFT performance, where heating muscle to ~38.3°C did not change performance and cooling muscle to ~33.3°C led to reductions in muscular power, contractility, and performance. Future work is needed to relate IPFT performance to traditional methods of assessing neuromuscular capacity (e.g. isometric and isokinetic models), whole body exercise (e.g. jumping, sprinting), and environmental stressors (e.g. temperature and oxygen).

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8. Appendix A

Brock University
Research Ethics Office
Tel: 905-688-5550 ext. 3035
Email: reb@brocku.ca

Health Science Research Ethics Board

Certificate of Ethics Clearance for Human Participant Research

DATE:	6/26/2024		
PRINCIPAL INVESTIGATOR:	CHEUNG, Stephen - Kinesiology		
FILE:	23-325 - CHEUNG		
TYPE:	Masters Thesis/Project	STUDENT: SUPERVISOR:	Johnathan Ljubanovich

TITLE: The Separate and Combined Effects of Passive Heating and Hyperoxia on Local Muscle Exercise Capacity (EEL-155).

ETHICS CLEARANCE GRANTED

Type of Clearance: NEW Expiry Date: 6/1/2025

The Brock University Health Science Research Ethics Board has reviewed the above named research proposal and considers the procedures, as described by the applicant, to conform to the University's ethical standards and the Tri-Council Policy Statement. Clearance granted from **6/26/2024** to **6/1/2025**.

The Tri-Council Policy Statement requires that ongoing research be monitored by, at a minimum, an annual report. Should your project extend beyond the expiry date, you are required to submit a Renewal form before 6/1/2025. Continued clearance is contingent on timely submission of reports.

To comply with the Tri-Council Policy Statement, you must also submit a final report upon completion of your project. All report forms can be found on the Research Ethics web page at https://brocku.ca/research-at-brock/office-of-research-services/research-ethics-office/#application-forms.

In addition, throughout your research, you must report promptly to the REB:

a) Changes increasing the risk to the participant(s) and/or affecting significantly the conduct of the study;
b) All adverse and/or unanticipated experiences or events that may have real or potential unfavourable implications for participants;
c) New information that may adversely affect the safety of the participants or the conduct of the study;
d) Any changes in your source of funding or new funding to a previously unfunded project.

We wish you success with your research.

Approved: [redacted]

Kimberley Gammage, Acting Chair
Health Science Research Ethics Board

Note: Brock University is accountable for the research carried out in its own jurisdiction or under its auspices and may refuse certain research even though the REB has found it ethically acceptable.

If research participants are in the care of a health facility, at a school, or other institution or community organization, it is the responsibility of the Principal Investigator to ensure that the ethical guidelines and clearance of those facilities or institutions are obtained and filed with the REB prior to the initiation of research at that site.