

**Assessing the effects of local
hyperthermia on muscle function in
younger and older adults**

**A Thesis Submitted for the
Degree of Doctor of Philosophy**

By

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Thesis abstract

A preparatory process aimed at optimising skeletal muscle function prior to maximal muscular contraction is fundamental and commonly observed during physical exercise. Additionally, certain populations may benefit from such preparation before engaging in activities of daily living. A key strategy used to achieve optimal skeletal muscle function is to increase tissue temperature above resting baseline. Skeletal muscle tissue temperature is usually increased prior to exercise requiring high force or high velocity production through an active warm up, consisting of moderate intensity exercise and dynamic contractions. For some populations, for example those with limited physical capacity such as the frail and elderly, an active warm up is not always possible. Increasing muscle temperature passively before exercise is an emerging field that may be an appropriate strategy to prepare those populations for dynamic contractions that are otherwise limiting factors in daily life. The central aim of this thesis was to investigate the effects of localised passive heating during maximal knee extensor exercise to determine the efficacy of passive heating as an ergogenic aid in younger and older adults.

The first experimental study (Chapter 4) investigated the effects of heating across a variety of dynamic contractile speeds and aimed to quantify the inter and intraday reliability of isokinetic and isotonic force measurements in younger healthy adults. The reliability across all isokinetic contractile speeds were measured to be “excellent” in the heated limb ($>.9$ ICC and Cronbach’s alpha) and displayed low variance ($<10\%$ Coefficient of variation). In response to the heating intervention, skeletal muscle temperature was increased to levels associated with active warm ups. Heating increased peak torque by 8% during moderate and by 10 % during fast contractions from 30 min onwards relative to the unchanged control leg. Rate of force production at 50ms and early force production was increased during the slow contraction by 14% and 15% respectively from 30 min in the heated leg whilst the control leg was unchanged.

Within the second experimental study (Chapter 5) the findings of chapter 4 were expanded upon and included healthy older adults within a comparable testing protocol. It is shown when comparing passive thigh heating responses between younger and older adults it was revealed that the increases in peak force that were observed in younger adults did not increase significantly in older adults. Older adults did however report increases in early force production that were even more pronounced when compared against improvements in younger adults. Both Chapters 4 and 5 report decreases in perceived exertion and improved perceptions in readiness for exercise in response to the passive thigh heating intervention, but the older adults also rated this effect to be more pronounced.

The final experimental study (Chapter 6) sought to further investigate how passive thigh heating may improve muscle function beyond maximal force production considerations by investigating the effect upon fatigue resistance during a maximal effort 30 moderate speed isokinetic knee extension task. Whilst peak torque was increased, reproducing the outcomes of chapter 4 and 5, the heating intervention appeared to have no effect on any fatigue related measure with no change in total work, average torque or peak EMG amplitude.

This thesis therefore identifies that localised passive heating has the potential to be an effective ergogenic aid, especially for younger adults, by improving muscle function during maximal contractions. The studies reveal that thigh heating increases peak isokinetic force during moderate and fast contractions and enhances early force production and rate of force development in both younger and older adults during slow contractions. These effects are likely due to direct changes in muscle contractile properties in response to localised increases in tissue temperature, not systemic physiological factors. While older adults did not improve peak force, they did show better early force production, indicating passive heating may enhance neuromuscular function with age and facilitate the completion of tasks of daily living that are modulated more by power than absolute strength. Additionally, participants reported feeling more ready to exercise and experienced less perceived exertion, suggesting passive heating could encourage physical activity, particularly among those reluctant to exercise due to discomfort.

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- **Denny D.C.A.**, Low D.C., Gibson O.R. (2025) Passive thigh heating does not augment fatiguability during prolonged, repeated knee extensor contractions in older adults (Manuscript in preparation, Applied Physiology, Nutrition, and Metabolism)
- **Denny D.C.A.**, Low D.C., Gibson O.R. (2025) No sex differences in response to passive thigh heating in younger and older adults (Manuscript in preparation, Experimental Physiology)

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- **Denny D.C.A.**, Low D.C., Gibson O.R. (2025) Passive thigh heating improves peak and early isokinetic force production in younger and older adults. *The Physiological Society, Thermal Physiology in Health and Disease: Mechanisms and Therapeutic Applications, Brunel University of London, Uxbridge, UK* (Oral Presentation). 3rd - 4th June 2025
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List of abbreviations

ANOVA	Analysis of variance
Ca ²⁺	Calcium ion
CONT	Control condition
DBP	Diastolic blood pressure
EFP	Early force production
EMG	Electromyography
EVP	Early velocity production
HEAT	Heated condition
HR	Heart rate
ICC	Intraclass correlation coefficient
MAP	Mean arterial pressure
MDC ₉₅	Minimal detectable change
MVIC	Maximal voluntary isometric contraction
RFD	Rate of force development
SBP	Systolic blood pressure
SEM	Standard error of measurement
SERCA	Sarcoplasmic reticulum Ca ATPase
T _{mu}	Muscle temperature
T _{skin}	Skin temperature
T _{tymp}	Tympanic temperature

Chapter 1 Introduction

Muscle function refers to the ability of muscles to generate force, produce movement, and maintain posture. It is regulated by complex interactions among multiple physiological systems, including neural control, energy availability, and biochemical signalling. Key factors such as motor neuron activity, ATP supply, calcium ion handling, hormonal regulation, oxygen availability, and temperature all play critical roles in influencing muscle performance. Muscle temperature is a key modulator of contractile performance given that specific tissue temperature modulates calcium handling, muscle stiffness, and nerve conduction velocity (Kobayashi et al., 2005; Eng et al., 2018; Gray et al., 2006). Optimal muscle function occurs at temperatures 38 to 40°C above the physiological resting range of 32–35°C, with these elevations typically attained prior to exercise through active warm-up protocols (Bishop, 2003). The majority of the benefits from active warmups are attributed to temperature regulated mechanisms (e.g. decreased stiffness, increased nerve-conduction rate, altered force-velocity relationship and enhanced chemical reaction rates) (Bishop, 2003); however, non-temperature related mechanisms (e.g. increased $\dot{V}O_2$ consumption) and an increase in sport specific skill proficiency are also benefits observed following an active warm up (Bishop, 2003).

In recent years, passive heating has emerged as a promising alternative or adjunct to active warm-ups, offering a means to elevate muscle temperature and potentially enhance muscle function without the need or risk of physical exertion (McGorm et al., 2018). Passive heating may also help those who are unable to complete a traditional active warm up before physical activity, such as older adults or those undergoing physiotherapy. Passive heating, which involves the application of an external heating source to skeletal muscle, has been shown to increase muscle temperature by up to 6.5°C, leading to improvements in peak torque, rate of force development (RFD), and early force production (EFP) in younger adults (Rodrigues et al., 2021; Mornas et al., 2022). Early applications of passive heating, such as hot springs and saunas, date back to ancient civilisations, where thermal therapies were primarily used for relaxation and recovery. However, it was not until the 20th century that scientific research began to systematically explore its physiological effects on muscle function (Rodrigues et al., 2022; Treigyte et al., 2024). In the 1980s, seminal studies emerged, investigating the impact of elevated muscle temperature on contractile properties. For instance, research by Davies et al. (1982) demonstrated that passive heating could improve muscle relaxation and contractile speed, laying the groundwork for its application in sports science. Sprint cycling also provided

an interesting situation in which to explore how passive heating affects short term power output during maximal exercise at a range of contractile speeds (Sargeant, 1987). This research demonstrated a contractile speed-related increase of power output with increased muscle temperature, cementing the idea that passive heating was a viable tool to improve muscle function. Research then progressed to focus on lower repetition resistance-based exercise and others highlighted that while passive heating could improve muscle contractile properties, it did not significantly enhance maximal voluntary contraction (MVC) torque during isometric contractions (Morrison et al., 2004; Thornley et al., 2003), suggesting a nuanced relationship between passive heat exposure and muscle performance.

The ability of skeletal muscle to operate with sufficient force and control is a critical determinant of physical performance, mobility, and quality of life, across the lifespan (Reid & Fielding, 2012). A minimum threshold of muscle function, encompassing strength, endurance, coordination, and flexibility, is essential for performing daily tasks (e.g., walking, lifting) and engaging in physical exercise safely (Hasegawa et al., 2008). Whilst a decline in muscle function is somewhat inevitable with ageing, exercise and physical activity can attenuate the reductions in muscle function, as can be seen in Figure 2.5 (Booth & Zwetsloot, 2010). Falling below a threshold of frailty can lead to reduced independence, increased injury risk, and difficulty maintaining overall health, highlighting the importance of regular physical activity, strength training, and balanced nutrition to preserve muscle function. Ageing is associated with sarcopenia, a condition characterised by significant declines in muscle function including reductions in muscle mass and strength (Santilli et al., 2014). Common function tests such as the timed up and go and sit to stand tests have been used to assess sarcopenia and frailty and correlate highly to a higher quality of life (Stegemöller et al., 2014). These function tests primarily quantify muscular power instead of peak strength. Whilst reductions in peak muscular strength have been the focus of sarcopenia research, newer work suggests that larger reductions in the ability to generate muscular power are observed with ageing, consequently highlighting the need to distinguish between reductions in strength and power production via use of the terms sarcopenia/dynapenia and powerpenia respectively (Freitas et al., 2024; Metter et al., 1997). Powerpenia refers to the more rapid decline in an aging muscle's ability to generate power (muscular power is defined as the work done, measured in joules, divided by the time the muscle was contracting, power movements are typically considered explosive type exercises) which is typically quantified by a reduction in lower body power production capability. Power has been suggested to be more influential than muscle strength and mass in enhancing physical function and reducing falls in older adults and is a more sensitive marker of disease presence and/or physical inactivity (Radaelli et al., 2023; Reid & Fielding, 2012). Together sarcopenia and powerpenia contribute to mobility impairments, frailty, increased fall

risk, and diminished quality of life (Cruz-Jentoft et al., 2010), this thesis seeks to investigate how passive heating can improve muscular strength and power within a population that suffers from sarcopenia and powerpenia.

Age-related changes of muscle mass and strength are driven by a complex interplay of factors; the reduction in muscle mass is mainly driven by a reduction in type II muscle fibres – driven by a decrease in muscle synthesis whilst muscle catabolism remains largely unchanged through age (Horwath et al., 2024). Sarcopenia is further associated with a loss and dysfunction of muscle mitochondria impacting myofibril function and muscular repair (Tournadre et al., 2019). Aged muscle also experiences indirect impairments to muscle function, reduced calcium release, re-uptake, and increased leakage within the sarcoplasmic reticulum, along with decreased nerve conduction velocity reduce both reaction time and the rate of muscle contractions (Hunter et al., 1999; Rivner et al., 2001). Given the importance of muscle function for maintaining independence and preventing disability in older adults, interventions that can acutely enhance muscle force production or positively alter muscle contractile properties by enhancing one or more pathways regulating contraction are of significant interest. Passively heating the muscle before exercise may increase muscle function, potentially enabling a more active lifestyle, slowing or reversing the decline of muscular strength and power associated with ageing. Muscle function is commonly assessed using isokinetic dynamometry, a method that enables precise measurement of force production and strict control over the type of muscle contraction (i.e., isometric, isokinetic, or isotonic). Examining different contraction types is important, as various activities rely on different forms of muscle action, for example, isokinetic contractions often more closely resemble those used in daily living compared to isometric contractions.

The reliability of any passive heating measure or related performance enhancement appears unstudied, posing a variety of issues from an unknown repeatability, such as unknown levels of natural variance and unknown minimal detectable change, which limits confidence of significant findings within the field of research. In the absence of this knowledge, research in the field of passive heating relies on assumed or unquantified levels of individual variability, which increases the likelihood of Type I error, incorrectly rejecting a true null hypothesis. This reliance on assumptions rather than empirical data may undermine the reliability and validity of findings in this area when looking at both acute (intraday) and chronic (inter-day) testing of muscle function following passive heating. The impact of passive heating on acute muscular force production remains unclear, particularly in older adults whose altered physiology of increased intramuscular fat (Addison et al., 2014) and decreased calcium handling capabilities (Dong & Maturana, 2025) may affect the response to heating, when compared to younger

adults. Should these physiological factors not interfere with muscle enhancement following passive heating, older adults may benefit from acute functional improvements to offset age-related declines in muscle strength, and function (Lexell et al., 1988; Cruz-Jentoft et al., 2010). For younger adults, muscle function may be adequate for most daily living tasks since the demands fall well below their maximal muscle force capacity. However, for older adults these daily tasks may be near to their maximal functional capacity (Hortobágyi et al., 2003). Older adults may therefore benefit from a preparatory strategy i.e., passive that enhances muscle function before undertaking maximal/near-maximal physical tasks. At the current time the effects of passive heating of skeletal muscle on maximal contractions have not been comprehensively examined. Given the importance of fatigue resistance for activities of daily living in older adults (Mänty et al., 2012), in addition to understanding whether passive heating can improve maximal force production, the ability for increased temperature to attenuate fatigue and improve exercise capacity is therefore of applied significance. There has been no assessment of how passive heating affects perceptions of exertion or readiness for exercise, understanding how participants perceive the effects of a tool are essential for programming and implementation.

1.1 Thesis aims

Within this thesis, the effects of passive thigh heating on peak force and early force production during isokinetic and isotonic knee extension exercise are investigated. Furthermore, the effect of passive thigh heating on fatigue resistance, and perceptual responses are studied for the first time, in both younger and older adults.

Specifically, the primary aims of this thesis are to:

- Quantify the inter- and intra-day reliability of isokinetic and isotonic muscle function assessments following passive heating
- Examine the acute effects of passive thigh heating on peak torque, rate of force development and early force development in isokinetic and isotonic muscle function, across a variety of contractile speeds in younger and older adults.
- Compare the systemic physiological and muscle force responses to passive thigh heating between younger and older adults.

By addressing these aims, this research contributes to a deeper understanding of how passive heat exposure influences muscle contractile function in younger and older adults. Section 2.3 Aim of Thesis describes the overarching aims and objectives whilst section 2.5 Proposed Research studies and Hypotheses describes the research aims and hypotheses for each experimental chapter.

Chapter 2 Literature Review

This literature review is divided into three sections. The first explores the structure and function of muscle in healthy adults, along with the changes that occur naturally with ageing (2.1.1 Anatomy of Skeletal Muscle - 2.1.10 Force velocity relationship). The second examines existing research on the effects of passive heating on maximal muscle force production (2.2.1 Foundational insights into the role of temperature on muscle function - 2.2.10 Decay of the heating effect). The literature review concludes by identifying gaps in the existing research and outlining the objectives of this thesis to advance understanding within the field of passive heating (2.2.11 Advancing knowledge and application of passive heating to improve muscle function - 2.3 Aim of Thesis).

2.1.1 Anatomy of Skeletal Muscle

Muscle structure groupings can be broken down from largest to smallest as follows: muscle, muscle fascicles, fibres, myofibrils, and sarcomeres as shown in Figure 2. 1. (Hou, 2018). Sarcomeres are the smallest functional muscle unit and are where contractile force in the muscle is produced, myofibrils are formed of chains of sarcomeres. Myofibrils are grouped into fibres by the endomysium, a thin layer of connective tissue that separates individual muscle fibres and allows fibres to glide over one another during muscle contraction as well as facilitating the metabolic exchange between blood and muscle (Stecco et al., 2015). Fibres are bundled together to form fibre bundles or fascicle and are surrounded by the perimysium, a thick layer of connective tissue that has been suggested to facilitate “slip planes”, allowing for muscle deformation during contraction (Purslow, 2010). Finally, fascicles are grouped together to form the muscle, covered by the epimysium, a thick layer of connective tissue covering the entire muscle and connecting to the tendon. The epimysium protects the muscle from friction against bones and other muscles and has been shown to assist in myofascial force transmission (Huijing, 2009).

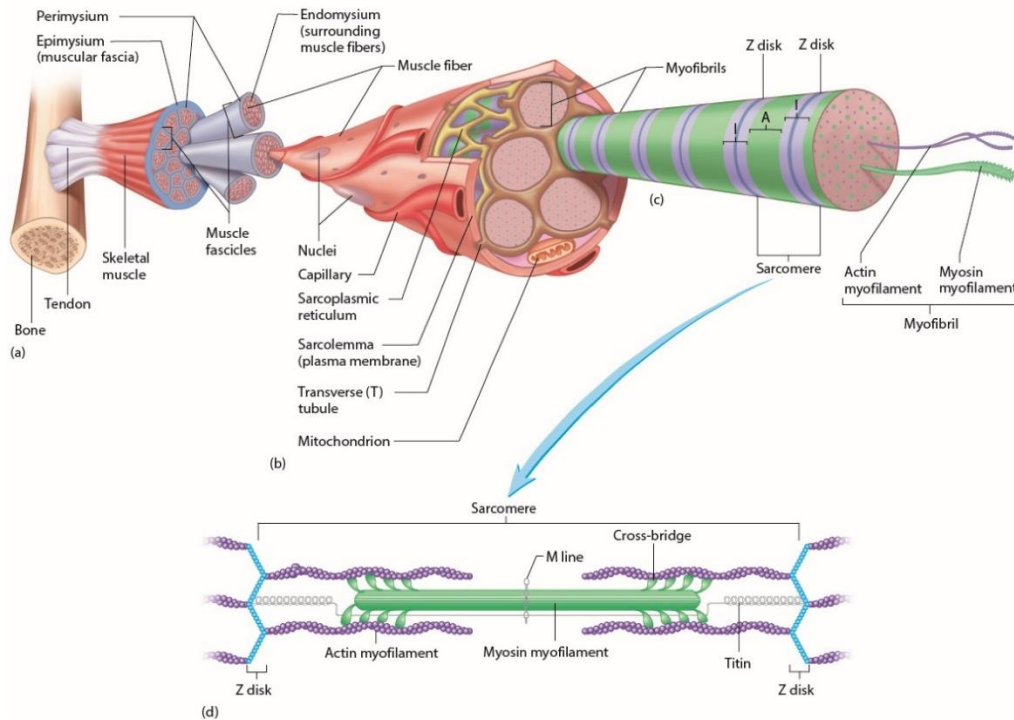


Figure 2. 1. An overview of the functional muscle units that comprise a skeletal muscle's structure, concluding with a depiction of a single sarcomere (Adapted from VanPutte, 2020).

In long muscles such as the *rectus femoris*, the peri and epimysium are composed of two sets of wavy collagens in a cross-ply arrangement however, in shorter muscles e.g., the *biceps brachji*, the connective tissue forms a dense outer layer, acting as a surface tendon allowing for greater force transmission (Purslow, 2010). Collagen represents 1-10% of dry muscle weight in cattle and has been suggested to be similar in humans (Bendall, 1967). Collagen fibres form covalent crosslinks which are essential for mechanical strength and stiffness, these crosslinks are formed both during childhood development and maturation though an enzymatic process and though the non-enzymatic process of glucose oxidation; primarily seen later in the life cycle (Bailey et al., 1998). Ageing causes collagen fibres to become increasingly rigid, inflexible, and brittle (Bailey et al., 1998), which results in fibres being more susceptible to tears and injury.

Lifestyle factors such as smoking, heat exposure and certain foods can facilitate the formation of additional crosslinks, creating similar collagen rigidity as seen in ageing, through the formation of advanced glycation end products (AGEs) (Purslow, 2010). Nerves and blood vessels permeate every muscle through the peri and endomysium, specialised nerve cells called motor neurons are responsible for initiating muscle contractions while the arteries supply oxygenated blood and substrates to the inner muscle through a network of capillaries

surrounding every muscle fibre, while blood and metabolites are carried away from the capillary bed by veins.

Muscle composition changes throughout the life course. Adipose tissue redistributes with ageing, moving from subcutaneous fat to intramuscular, increased levels of intramuscular fat (IMF) have been associated with lower muscle strength, power, chronic inflammation, impaired glucose tolerance, and elevated total cholesterol in older adults (Waters, 2019). The increase in IMF is partly associated with a decrease in physical activity levels however, another factor involved in increasing IMF during aging is the formation of small fat deposits, adipocytes, created when skeletal muscle satellite cells differentiate into a calorie storage form following injury, glucocorticoid treatment, or inactivity (Waters, 2019). Muscle composition can be changed through the reduction of myogenic (muscle regenerative) activity and an increase in fibrogenic (thickening or increase of fibrous tissue) activity, this decrease in muscle tissue repair and increase in connective fibre production has been shown to double a muscle's stiffness in aged mice (Wood et al., 2014).

2.1.2 Loss of Skeletal Muscle Function Across the Lifespan

The age-related decline of strength and muscle mass is referred to as sarcopenia. Sarcopenia is a multifactorial process involving decreased physical activity, deficient nutritional intake, mitochondrial dysfunction, neurodegenerative disease, and hormonal changes. The European Working Group on Sarcopenia in Older People (EWGSOP) states that at least two of the following criteria must be met to diagnose an individual with sarcopenia (Cruz-Jentoft et al., 2010):

- Low muscle mass
- Low muscle strength
- Low physical performance

The criteria differentiate between a loss of mass and a loss of strength as the relationship between muscle mass and strength is not linear, with muscle strength lost more quickly than mass (Goodpaster et al., 2006). This indicates a decrease in muscle quality associated with ageing.

Sarcopenia is influenced by multiple factors beyond physical inactivity (Figure 2. 2.), and those affected are not merely inactive. Exercise can mitigate its effects, but immobilization due to hospital stays or injuries accelerates neuromuscular decline. Sarcopenia results from social and physical mechanisms, leading to frailty, limited mobility, and reduced quality of life (Kara

et al., 2021). Essential daily tasks become difficult as muscle function declines, with knee extensor force decreasing significantly with age. This project focuses on muscle contraction mechanisms and ways to enhance force production.

Sarcopenia is linked to cognitive decline, depression, and loss of independence (Gariballa & Alessa, 2018). Strength training improves muscle function, reduces impairment, and lowers fall risk (Gillespie et al., 2003; Liu et al., 2009). Exercise enhances mitochondrial function and protein turnover (Coen et al., 2018). The UK Chief Medical Officers recommend strength training twice a week and 150 mins of moderate aerobic exercise for older adults, yet only 13% meet these guidelines (Gov.UK, 2020; Rawson, 2010). Muscle loss primarily affects type II fibers due to reduced innervation and capillarization (Andersen, 2003; Morley et al., 2001). Fear of injury further reduces strength training participation, worsening atrophy and degeneration (Skelton & Beyer, 2003). Aging also reduces muscle force due to declines in actin and myosin accumulation (Narici & Maganaris, 2006). Figure 2. 3. depicts the importance of maintaining muscle strength function is to maintain independent living and avoid frailty. Physical activity and exercise have been identified as the most effective tool at reversing and/or slowing the progression of sarcopenia (Marzetti et al., 2017), this thesis hopes to explore the foundations of how passive heating may benefit those partaking in physical activity.

Ageing disrupts the homeostasis of skeletal muscle with a chronic increase in the catabolic pathways and a decrease in the muscle protein anabolic pathways, resulting in decreased muscle mass through a reduction of myofibers (Cruz-Jentoft & Sayer, 2019), disproportionately affecting type II fibres (discussed further in 2.1.7). Dysfunctional and damaged mitochondria have been speculated to underly and regulate a significant portion of the molecular pathways that contribute to sarcopenia (Marzetti et al., 2013). Malfunctioning mitochondria increase the production of reactive oxygen species, a reactive and unstable molecule responsible for oxidative damage to DNA and bodily proteins (Alway, 2021). Young bodies can easily remove dysfunctional mitochondria through mitochondrial autophagy; however, the pathway responsible for clearing damaged cells, the ubiquitin-proteasome system, is weakened with ageing (Löw, 2011). Classifying the level of sarcopenia within an individual is a difficult task, it is not possible to accurately map out declines in muscular mass or strength across a lifespan nor is it feasible to assess all performance measures. It has been suggested that muscle mass and muscle function should be assessed within the classifications of “presarcopenia”, ‘sarcopenia’ and ‘severe sarcopenia’ as assessed by measurements of gait speed, grip strength and muscle mass (Cruz-Jentoft et al., 2010).

Research into the hormonal influences of sarcopenia identifies declines of androgenic hormones namely dehydroepiandrosterone sulphate (DHEAS) and testosterone in older men (Vitale et al., 2016). DHEAS is responsible for converting active androgens and oestrogens and stimulating insulin like growth factor-1 (IGF-1). These compounds are important in muscle growth and maintenance (Maggio et al., 2013). Testosterone is the primary sex hormone of males and a powerful anabolic steroid. Bioavailable testosterone decreases at a rate of 2% every year after 30 years for males and testosterone levels drop rapidly from 20 to 45 years of age for females (Maggio et al., 2013). While the menopause is associated with a decline in sex hormone production, the role that the oestrogen plays in maintaining muscle mass, strength or function is still unclear. For example, a 64 week trial of oestrogen replacement therapy on postmenopausal women did not display any significant changes in muscle mass (Hansen et al., 2003).

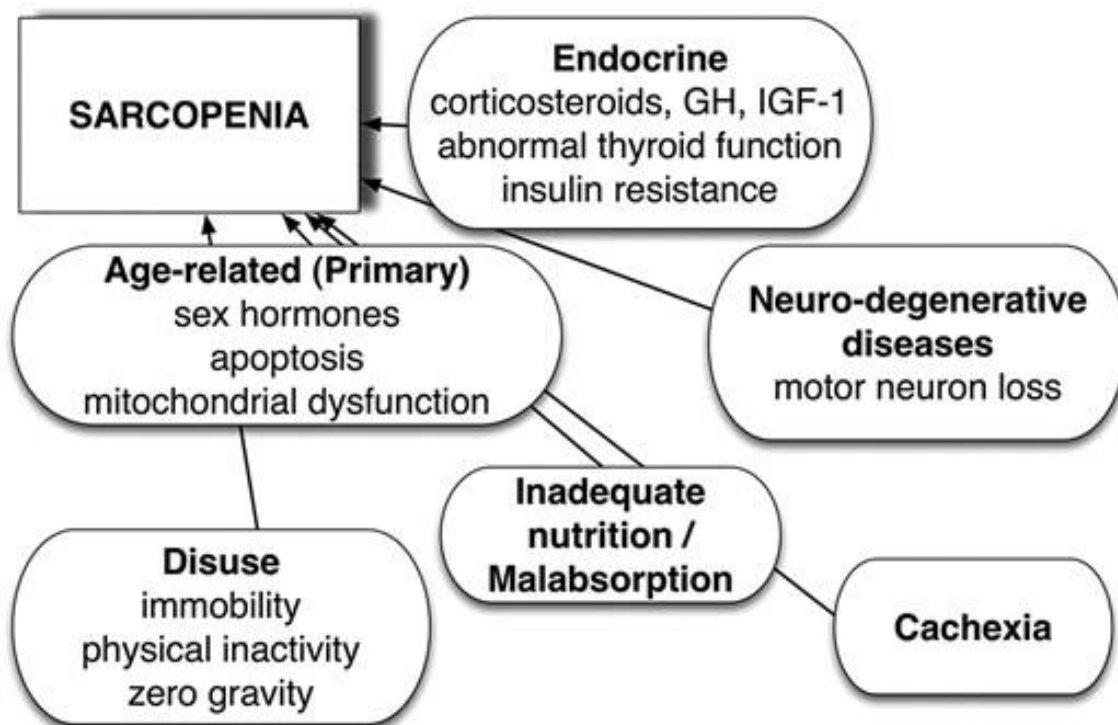


Figure 2. 2. The mechanisms involved in the onset and progression of sarcopenia, adapted from Cruz-Jentoft et al., 2010

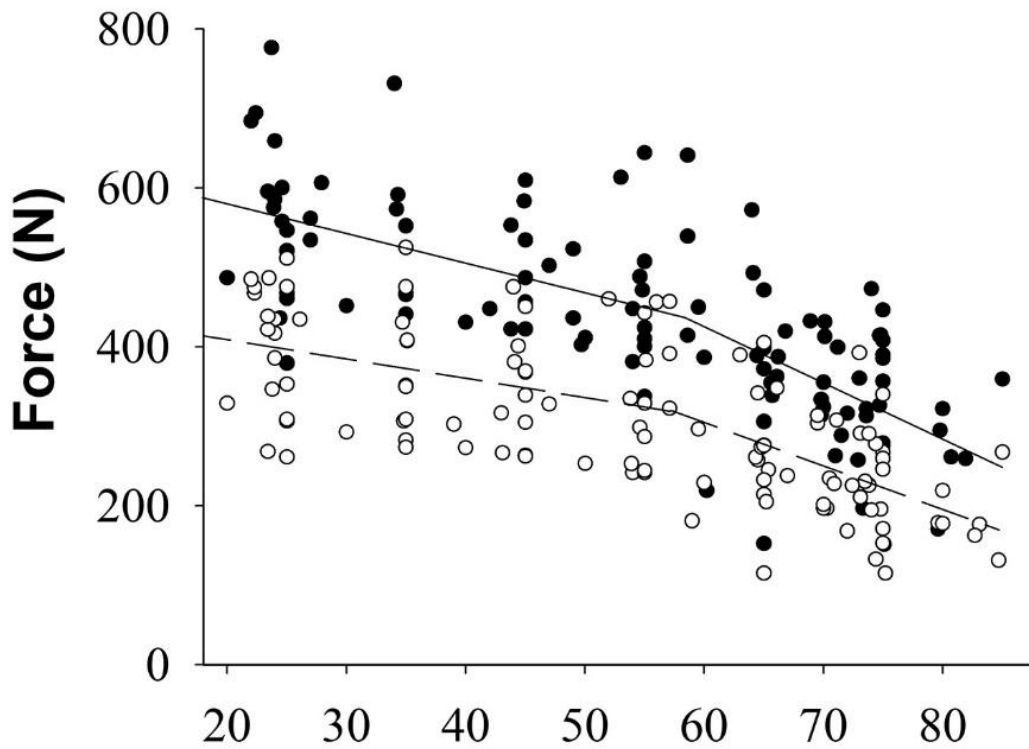


Figure 2. 3. Mean isometric force produced by the knee extensors across the lifespan (X axis, age in years) (Adapted from Haynes et al., 2020).

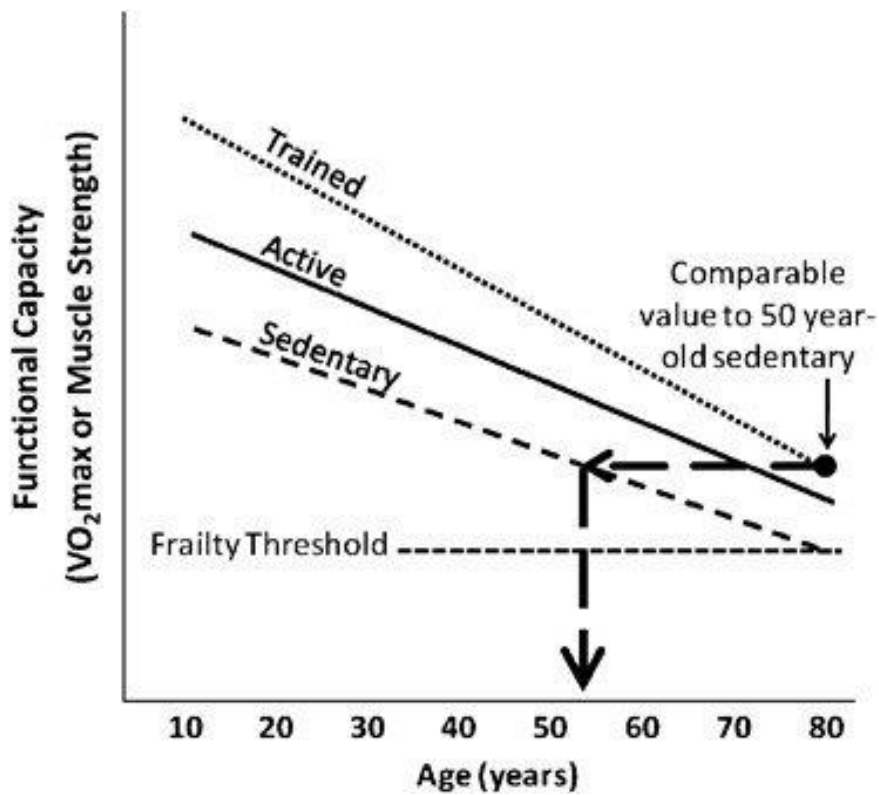


Figure 2. 4. Schematic depicting that physical activity delays the decline in physical function with aging (Adapted from Booth & Zwetsloot, 2010).

2.1.3 Muscle force production

Each stimulus received by the muscle causes a muscle twitch. If stimuli are delivered slowly the muscle can relax between twitches, however, if the stimuli are delivered quickly, the twitches begin to overlap, causing either unfused or fused tetany. Unfused tetany occurs when the muscle fibres only partially relax, between contractions while, fused tetany results in no relaxation for the muscle fibre due to the highest rate of stimulation, this results in the strongest form of steady state muscle contraction. The force of contraction is voluntarily regulated through the frequency of stimulation provided to the muscle from the nervous system. Assuming the length and force are equivalent, higher frequency stimulations result in greater force production (Rongsawad & Ratanapinunchai, 2018). Optimal stimulation frequency is dependent on fibre type and temperature. When observing *in vivo* muscle fibres between temperatures of 30-35°C, which is a suboptimal temperature for peak muscle function, a supraphysiological frequency between 100-200 Hz is required for peak contractile force (Fitts et al., 1991). At higher stimulus frequencies, an increase in intracellular Ca²⁺ is present in the contracting muscle due to the inability of the body to reabsorb the ions back into the sarcoplasmic reticulum before the beginning of the next contraction. An increase in Ca²⁺ improves muscle contractile efficiency, resulting in a greater formation of cross bridges (Sale, 2004). Muscular strength does not simply scale with an increase in muscle fibre diameter (Reggiani & Schiaffino, 2020), however, an increase in fibre diameter does though facilitate a greater number of cross-bridge formations due to an increase in myofibrils.

Older adults (65+ years old) have a reduced adenosine triphosphate (ATP, cellular energy source) cost for muscle tetany compared to a younger population (25-30 years old), this may be partially explained by a reduced type II muscle mass and a decrease in nonoxidative energy producing pathways (Tevald et al., 2010). The reduction in ATP cost to achieve muscle tetany may contribute to observations that older populations outperform younger populations in muscular endurance task. This phenomenon was shown in Tevald et al., (2010) where 9 young and 9 old (26 ± 4 and 72 ± 5 years old respectively) participants performed an invoked fatigue task at 25 Hz and at 50% of maximum twitch. The energy cost of a single twitch at 25 Hz was 27% lower in older individuals (0.13 ± 0.04 mM ATP/twitch, 0.18 ± 0.06 mM) while during a 90 sec fatigue test at 50% of maximal invoked frequency the older group displayed a 49% reduction in ATP cost (1.0 ± 0.2 mM ATP/s, 1.9 ± 0.2 mM ATP/s.)

In an otherwise equal state, to further increase force production during a muscle contraction, additional motor recruitment is required. By increasing the number of motor units stimulated, additional groups of muscle fibres are recruited into contracting (Clamann, 1993). During motor unit recruitment, smaller motor units, typically composed of type I (slow-twitch) muscle

fibres, are activated first. As the intensity of the stimulus increases, larger motor units, which generally consist of type II (fast twitch) fibres, are progressively recruited to generate greater force. In older adults, the loss or atrophy of type II fibres may limit this capacity for high-force production, potentially restricting them to relying predominantly on smaller motor units and thereby reducing overall muscle strength. The minimum required force to stand up from being seated is between 35 – 50 N/kg (Yoshioka et al., 2012) and maximal knee extension force in adult males has been reported as 2.9–3.5 N.m/kg while elderly males typically have a lower range of 1.7–2.2 N.m/kg (Šarabon et al., 2021). While knee extensors are not solely responsible for a standing from seated movement, they play a large role in force production along with the gluteal muscles (Millington et al., 1992).

2.1.4 Types of contraction

There are three general classifications of muscle contractions; isometric contractions, where the muscle does not shorten during contraction and no movement occurs, eccentric contractions, where the muscle activates but lengthens, this is often a 'braking force' and concentric contractions, whereby the muscle shortens, and movement is generated. Isometric contractions often occur when maintaining posture and when applying force to an object that you cannot move. Isometric contractions can be used to observe a muscle independent from the rest of the body as the high pressure generated from a maximal isometric contraction occludes blood flow (Edwards et al., 1972). Isokinetic contractions occur when the velocity of the muscle contraction remains constant while the length of the muscle changes, while isotonic contractions maintain constant tension within the muscle whilst muscle length changes; this is usually achieved through fixed resistance exercise.

Concentric contractions are a common form of muscle contraction during everyday living, as the muscle shortens force is generated and applied, seen in locomotion, and almost all daily and sporting activities such as kicking a ball or standing out of a chair. Eccentric muscle contraction occurs when the muscle lengthens as contraction occurs, often referred to as a 'braking force', slowing down and smoothing movement (Reed & Bowen, 2008). Eccentric contractions can generate the highest force of any contraction, as the elastic properties of the muscle and connective tissue aid in force production (Radák, 2018). For more explanation of how aging affects eccentric contractions, see 2.1.2.

Isokinetic contractions can be defined as a contraction where the velocity of the muscle contraction remains constant while the length of the muscle changes. Isokinetic training requires specialised equipment that allows the individual to exert a maximum force throughout the full range of motion, the machine provides a resistance to maintain the velocity of

contraction. Isokinetic dynamometry is considered the gold standard of muscle function testing (Kambič et al., 2020) and is often used when assessing dynamic strength and muscle function in professional athletes (Julia et al., 2021), elderly individuals (Bohannon, 2020) and research (Rawson et al., 2011). Isokinetic dynamometry requires that a speed of contraction be set, by manipulating the speed different contractile functions can be measured, speed of contraction has long been established to be influential in muscle force production after heating, with higher speeds of contraction incurring higher benefits (Sargeant, 1987). The slower contraction speeds (60 degrees per sec) generate the highest forces (as discussed in 2.1.8) and are often used to measure maximal voluntary dynamic contraction (Almosnino et al., 2014).

Research should include a variety of contractile speeds to adequately understand how an experimental variable affects muscle function and how it might be applied in a real-world setting. As previously mentioned in Section 2.1.2, force velocity relationship, slower speed contractions generate more force than quicker contractions, accurately assessing a muscle's full function, velocity specific measurements must be made (Pousson et al., 1999). Changes in muscle function with age vary depending on the type and speed of contraction, Pereira et al., (2019) compared maximal strength and rate of force production across; isometric, isokinetic and isotonic knee extensor contractions between a young population (20-29 y; n = 13) and an older population (+70 y; n = 9). Isokinetic testing occurred at a slow, medium, and fast contraction velocity (60, 180, 240°/s) with the older population showing a decrease in strength by 61%, 56% and a 55% respectively. Isotonic force production showed similar results with a 47% decrease in 1 MVC (92 kg compared to 49 kg). During the isometric task the elderly group displayed a 60% decrease in force produced (340 kg/s compared to 135 kg/s). These data highlight the age-related loss of specifically type II fibres (discussed in 2.1.10) and how this disproportionately affects slower maximal contractions and explosive strength as weakened type II fibres struggle to produce rapidly output force.

Paturel, (2014) further demonstrates the decrease in explosive force production an older (69 - 81 y) and younger group (19 - 30 y) participated in an isotonic knee extension velocity task, kicking against 10% of their isometric MVC. The older group had a 23% reduced contractile velocity ($448 \pm 33^\circ/\text{s}$, $346 \pm 24^\circ/\text{s}$ respectively). This pattern of reduced explosive power was also observed in the isokinetic test where participants completed knee extensions at $400^\circ/\text{s}$; the younger group generating 60% more torque (Younger 96 ± 16 Nm, Older 38 ± 14 Nm). This study clearly demonstrates an age-related decrease in fast contractile ability, being 36% slower than the younger cohort and generating 25% less force at a fast contractile velocity. The reduced isotonic velocity is suspected to be a failure to generate enough force to achieve a high velocity against resistance, whereas the isokinetic tests assist in velocity of the limb

demonstrating a failure to generate force at high velocity suggesting a physiological limitation of the aged muscle fibre to form and cycle cross bridges at high velocities.

Age-related declines in muscle function, particularly in concentric and eccentric contractions, are closely linked to reduced mobility and increased fall risk in older adults. Concentric contractions, essential for initiating movement (e.g., rising from a chair), and eccentric contractions, for controlled descent and balance recovery (e.g., stair descent), both deteriorate with age due to loss of type II fibres, reduced neuromuscular activation, and impaired coordination (Reid & Fielding, 2012). While isometric strength, important for postural stability, is relatively preserved, deficits still contribute to instability (Hunter et al., 2016). These impairments hinder functional tasks and increase susceptibility to falls, highlighting the need for targeted interventions that address contraction-specific weaknesses in the aging population.

2.1.5 Muscle activation

Muscle activation originates in the cognitive cortical areas of the frontal lobe and proceeds through the nervous system. The central nervous system (CNS), consisting of the brain and spinal cord, acts as the body's central processor, making executive decisions and coordinating bodily functions. The peripheral nervous system (PNS) includes all neural elements outside the CNS, acting as the messenger by transmitting sensory data to the CNS and relaying motor commands to muscles and glands. Within the PNS, afferent pathways carry sensory information from receptors detecting external (e.g., temperature, light, touch) and internal (e.g., muscle temperature, thirst, blood pressure) stimuli to the CNS, where it is processed for an appropriate response (Hug et al., 2023). Efferent pathways transmit signals from the CNS to the body via two divisions: the somatic nervous system (SNS), which controls voluntary movements by directing signals to muscles (Carp & Wolpaw, 2010), and the autonomic nervous system, which regulates involuntary functions such as heart rate, gland secretion, and smooth muscle contraction. Motor neurons extend from the CNS to muscles, facilitating activation through a network of nerve cells. Afferent neurons, with short axons and long dendrons, detect stimuli and report to the CNS, while efferent neurons, with long axons and short dendrons, transmit CNS signals to effector organs, muscles, and glands. In addition to voluntary muscle activation, proprioceptors play a crucial role in movement regulation. These sensory receptors provide continuous feedback on muscle length, tension, and position, enabling the nervous system to adjust motor neuron activity and ensure smooth, coordinated movement (Keynes et al., 2011).

Each motor neuron controls several muscle fibres with individual tendrils of the motor neuron (axons) innervating a single muscle fibre, the number of muscle fibres innervated by a single motor neuron range from 95 in the first lumbrical to 1,096 in the platysma in healthy younger adults (Duchateau & Enoka, 2022). Ageing is accompanied by an increased innervation ratio whereby more muscle fibres are connected to each motor unit (Hunter et al., 2016). To contract a muscle, an action potential must be sent from the brain or spinal cord along the motor neurons to the muscles, for the action potential to cross from the motor neurons axon branch to the muscle fibre it must pass through a synapse. As the action potential reaches the presynaptic terminal, voltage gated Ca^{2+} channels open which commence a release of acetylcholine (ACh); a neurotransmitter that facilitates the communication between nerves and target cells. ACh diffuses into the synapse, binding with the Na^+ ligand gated channels, opening the channels allowing the Na^+ to enter the muscle fibre depolarising the motor end plate and continuing the action potential across the sarcolemma. A conductive plasma membrane surrounds the muscle fibre and facilitates the transmission of action potentials across a muscle fibre.

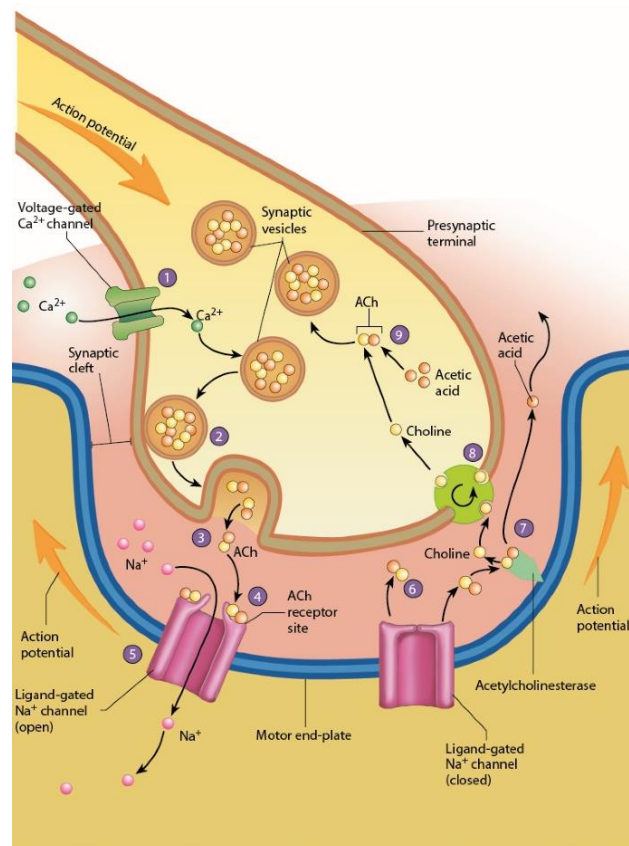


Figure 2. 5. A depiction of an action potential traveling to a muscle fibre and stimulating contraction. (Adapted from VanPutte, 2020).

The ACh then unbinds from the receptor and is broken down by the enzyme acetylcholinesterase into choline which is reabsorbed into the presynaptic terminal and acetic acid which is diffused away from the synapse. Voluntary activation has been found to decrease with age; impaired function of the CNS and the decreased involuntary activation of the motor cortex and motor units seem to mainly affect the larger muscle groups such as the quadriceps (Clark & Taylor, 2011). An age-related decrease in neuromuscular junction stability, caused by the gradual decrease in the nervous signalling network as motor neuron death and axon degeneration occur, has also been described as responsible for a reduction in muscle force production (Hepple & Rice, 2016). However, the effects of a decreased neural drive on muscle force production are considered slight, most reductions are due to physiological factors within the muscle.

2.1.6 Force production (Sliding Filament Theory)

For the chemical reaction of the action potential to be turned into a mechanical contraction, the action potential travels along the sarcolemma until it reaches the T tubules. T tubules are tubes that wrap around the sarcomere, allowing the action potential to access the interior of the muscle fibre. When the action potential reaches the T tubules, it opens voltage gated Ca^{2+} channels, flooding the sarcoplasm around the myofibrils and inducing a contraction.

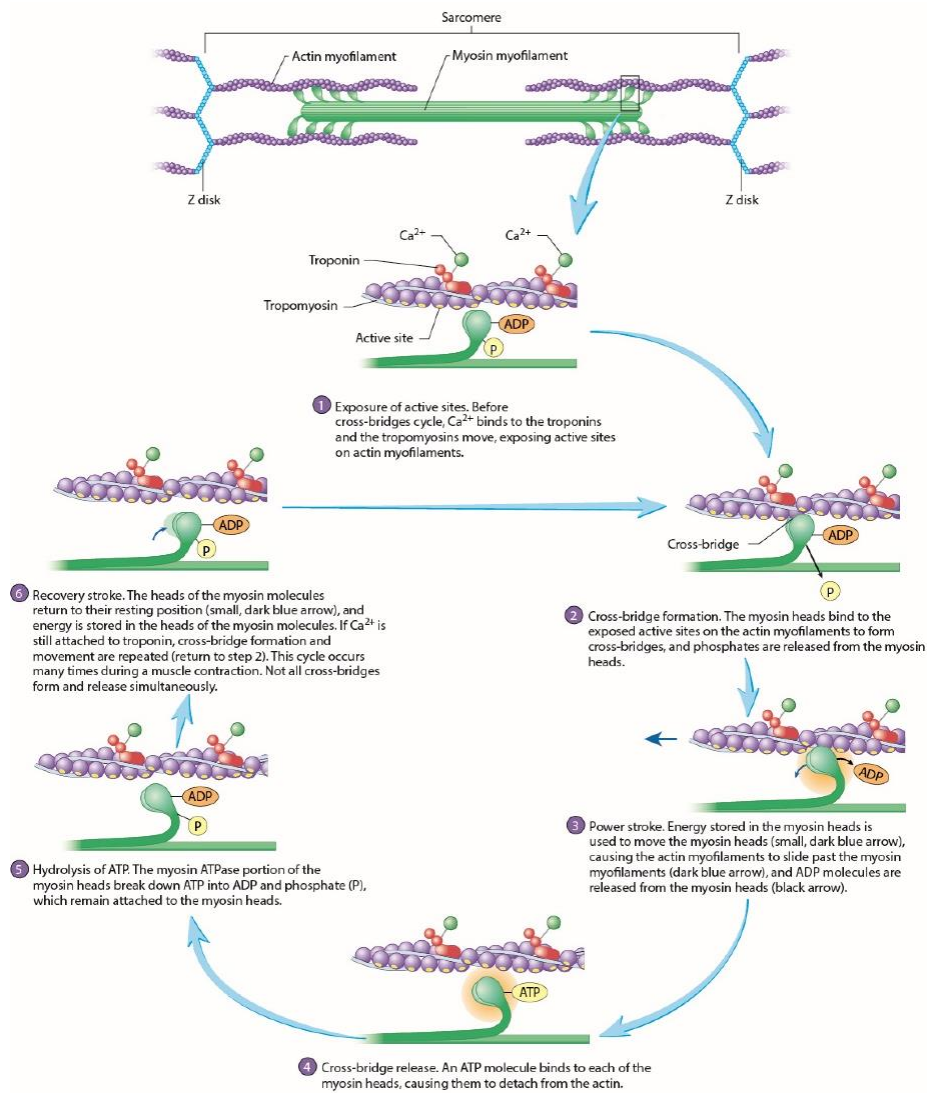


Figure 2. 6. A figure of the sliding filament theory, depicting one cross bridge cycle. (Adapted from VanPutte, 2020).

The mechanism of muscle contraction is currently best explained through the Sliding Filament Theory. Within the sarcomere exist two myofilaments, a thick myosin filament and a thin actin filament. When a muscle contracts, the myofilaments do not shorten, however, they slide past each other contracting either end of the sarcomere. After the voltage gates in the T tubules open, the Ca²⁺ binds to troponin molecules on the actin myofilament causing a barrier of tropomyosin to move, exposing the active sites allowing the heads of the myosin filament to bind forming a “cross bridge” between the two myofilaments. Each myosin head has an adenosine diphosphate (ADP), and phosphate molecule bound to it, after the cross bridge has been formed the phosphate molecule is released, the energy stored in the myosin head is used to create movement (power stroke) sliding the actin myofilament to slide towards the centre of the myosin, resulting in the ADP being released. For the cross bridge to be reset the myosin head binds to an adenosine triphosphate (ATP) molecule, which is then hydrolysed by

ATPase into ADP and a phosphate; the energy generated from this reaction is stored in the myosin head for the power stroke in the next cross bridge formation. If Ca^{2+} is still attached to the troponin, the cycle begins again. As action potentials cease, the muscle can relax. ACh is removed from the synapse, preventing the release of Ca^{2+} into the sarcoplasm and removing Ca^{2+} from the troponin molecules, moving the tropomyosin and closing the active sites. Current theories of muscle contraction focus solely on the force produced by the power stroke of the sliding filament theory, also referred to as a myosin motor. However, it has been also proposed that an elastic protein, titin found in the sarcomere that attaches the myosin filament to the z-disks, is responsible for producing force through a stepwise folding contraction (Rivas-Pardo et al., 2016). Titin was previously thought to provide only passive elasticity in striated muscle (Trombitás et al., 1995), however, additional functions include maintaining the structural integrity of the sarcomere (Horowitz & Podolsky, 1987), providing protection by limiting sarcomere lengths (Granzier et al., 1990) and providing passive stiffness within a muscle fibre (Brynnel et al., 2018).

Aged mice muscle has shown an increase in resting calcium concentration leading to disruption to the internal homeostasis, increased sodium concentrations and oxidative stress. (Mijares et al., 2021). While resting calcium levels in aged muscle have not been tested in humans, the transport of calcium from the sarcoplasmic reticulum to the muscle is known to be compromised through oxidative stress damaging the Serca isoform of PM-Ca-ATPase reducing calcium release and reuptake within the muscle fibre (Squier & Bigelow, 2000) (further discussed in section 2.2.5). Similarly resting ATP levels remain untested however an age-related decay in mitochondrial function results in diminished ATP generation within the muscle (Rooyackers et al., 1996) . Petersen et al., (2003) attributes this ATP reduction to a decrease in mitochondria number/function, discovering a ~40% reduction in mitochondrial oxidative and phosphorylation activity in 15 elderly (70 ± 2 y) muscle compared to a group of 13 younger (27 ± 2 y) participants. ATP production may also be linked to physical activity levels, as people adopt a more sedentary lifestyle the need for ATP generation drops. It is proposed that afferent sympathetic nerves co-ordinate with the hypothalamic centre in the brain to regulate ATP production (Nair, 2005). Along with these proposed mechanisms is the degradation of the myosin head reducing the possible actin-myosin interactions that generate force (Lowe et al., 2001).

The SERCA pump function is a key regulator of muscle health, and a decrease in function has been associated with pathological conditions such as aging, neurodegeneration, and muscular dystrophy (Xu & Van Remmen, 2021). The SERCA pump is especially vulnerable to increased oxidative stress caused by ageing. Oxidative stress is caused by an accumulation of oxygen

reactive species (ROS) in cells and tissue, which can damage cellular structures like proteins, lipids, and nucleic acids when they interact with ROS (Wu et al., 2013). Oxidative damage to the SERCA pump can reduce Ca^{2+} regulation, reducing force generation, rate of force development and twitch contraction half relaxation time (Xu & Van Remmen, 2021). This points to the potential for efficacy of passive heating in older individuals, who have a damaged SERCA pump, receiving a benefit from the increase in SERCA pump activation following heating.

2.1.7 Muscle fibre types

Muscle fibres can be divided into two main categories, fast and slow twitch fibres. Both slow and fast twitch fibres are present in human muscle tissue however the distribution varies depending on the needs of the muscle; postural muscles contain more slow twitch fibres while the arms and shoulders have a higher fast twitch count. Healthy individuals reach adult muscle fibre size between the ages of 12-15 and while the function of muscle fibres may suggest severe proportional differences, there is less than a 12% difference in mean fibre diameter is seen across all fibre types (Miljkovic et al., 2015).

2.1.7.1 Slow twitch fibres

Histological assessment of slow twitch fibres shows that they contain more mitochondria and are more densely populated with blood vessels. Slow twitch fibres are named as such due to the slow contraction velocity, they contain a slow form of the enzyme ATPase resulting in a slower cross bridge cycling rate. Aerobic respiration is the primary method of ATP synthesis, supported by a large blood supply, many mitochondria, and high concentrations of myoglobin; a protein that carries oxygen from red blood cells and stores the oxygen in the muscle fibre providing an oxygen supply further aiding in aerobic respiration. In addition to the slow contraction velocity slow twitch fibres respond more slowly to nervous stimulation (Baylor & Hollingworth, 2012). Slow twitch fibres account for between 17.6 - 65.6% of the fibres in the vastus lateralis for men and 16.5 - 97.4% of the fibres in women (Staron et al., 2000), individual differences play a large part in the muscle composition of an individual however, fibre type distribution is often associated with usage. As the vastus lateralis is mainly used in locomotion therefore the primary muscle being suited to low force, long duration repetitive exercise would be advantageous. Whilst type I fibres may be more populous in men, type II fibres are often bigger and constitute a larger cross-sectional area within the vastus lateralis, the inverse is true for women (Staron et al., 2000). With ageing, type I muscle fibres are relatively preserved in number and size compared to type II fibres, largely due to their frequent use in daily activities and lower activation thresholds. Older adults tend to have a higher proportion of type I muscle fibres compared to younger adults, this is primarily due to the selective loss and denervation

of type II fibres with age, rather than an actual increase in type I fibres. However, ageing still leads to reduced force production and slower contractile properties in type I fibres, due to excitation–contraction coupling changes and reduced calcium handling (Lamboley et al., 2015). Older adults often exhibit greater fatigue resistance than younger adults during low-intensity, low-force contractions, primarily due to a higher relative proportion of type I (fatigue-resistant) muscle fibres resulting from the age-related loss of type II fibres (Kent-Braun, 2009; Lanza et al., 2004). Conversely, the age-related reduction in type II fibres leads to decreased fatigue resistance during high-velocity and high-intensity tasks, where rapid force production and power are required (Avin & Frey Law, 2011).

2.1.7.2 Fast twitch fibres

Fast twitch fibres can be divided into two categories, type IIa (fast twitch oxidative glycolytic, using both aerobic and anaerobic respiration) and type IIb (fast twitch glycolytic, using primarily anaerobic respiration) they both respond more quickly than slow twitch fibres to nervous stimulation, have fewer mitochondria, reduced blood supply, less myoglobin and a fast-acting ATPase on the myosin head allowing for quicker contractions. The aforementioned characteristics make this fibre type unsuited to aerobic respiration however, fast twitch fibres have high glycogen stores making them are well adapted to anaerobic respiration. Due to a higher dependency on anaerobic respiration fast twitch fibres have a shorter time to fatigue than slow twitch fibres. Men typically have larger type II fibres than type I and women typically have the opposite. The ratios of muscle fibre types remain similar between the sexes with the vastus lateralis reportedly containing 42% type I, 38% type IIa and 20% type IIx in both groups (Staron et al., 2000) however, while these percentages provide an average, individual differences provide the chance for certain individuals to possess fibre type ratios of >60% type I fibres or <25% type I fibres (Staron et al., 2000) these ratios will change with age as sarcopenia affect different muscle fibres at different rates (Deschenes, 2004). A reduction in lower limb muscle mass due to aging is in part caused by a 30-40% reduction in the number of muscle fibres between ages 20-80 years old (Evans & Lexell, 1995). Muscle fibre size decreases, but to a lesser extent, and is fibre type specific; with type IIa fibres reducing in size by 10-40% and becoming less innervated resulting in a further loss of function (Frontera et al., 2000). A reduction in type IIa fibres naturally shifts the ratio of fibre types towards a more type I focused muscle, this shift is further exaggerated by the fast to slow fibre shift, affecting type IIx associated with aging (Miljkovic et al., 2015).

2.1.8 Substrates for maximal force contractions

Substrates typically refer to the utilisation of lipids and carbohydrates as a fuel source, however as this review is interested in short, maximal contractions which require the usage of anaerobic and then glycolytic pathways those will be the focus.

As previously discussed, ATP is the essential energy currency for muscle contraction. To maintain a constant supply, the body can utilise a variety of ATP regenerating pathways. As exercise begins the small reserve of ATP stored in the muscle tissue is rapidly exhausted. The quadriceps contains only ~9 mM of ATP which can be used to sustain around six sec of maximal high intensity exercise (Sahlin, 2014) however, by utilising the ~32 mM of phosphocreatine (PCr) stores ADP can be resynthesised (Gerdle et al., 2013). This 'ATP-PCr system is responsible for maintaining ATP homogenesis and extending exercising capabilities for around fifteen sec. When exercise demand exceeds the capability of the stored ATP and PCr the body turns to anaerobic respiration, primarily type II fibres are responsible for the anaerobic generation of ATP. Anaerobic respiration occurs when no oxygen is present after glycolysis to continue the electron transport chain; the generated pyruvate is reduced to lactate and 2 ATP. Anaerobic respiration can provide ATP for up to 3 mins of high intensity exercise and is primarily used during strength and resistance exercise. Longer form exercise requires an energy source that can generate ATP for a prolonged duration, aerobic respiration begins similarly to anaerobic respiration, where glycolysis breaks down glucose into pyruvate however the pyruvate is processed in the mitochondria, where in the presence of oxygen the citric acid cycle and electron transport chain are able to generate 32 ATP for each glucose molecule. Aerobic respiration is responsible for most of the energy production at rest and can be sustained for hours during moderate intensity exercise. Whilst these energy systems appear to operate independently, each system works in tandem and often overlaps with multiple systems providing energy at the same time; while aerobic respiration provides many more ATP than the other systems, the rate of energy production is much slower. ATP synthesis rate has been shown to be a temperature dependant reaction, *in vitro* mitochondria have demonstrated a synthesis rate of 13.61 nmol ATP per min per mg mitochondrial protein at 20 °C, increasing to about 20.7 nmol ATP per min per mg protein at 30 °C. (Roussel & Voituron, 2020). The increased synthesis rate is linked to heat activation of the electron transport chain and a decline in ATP efficiency, requiring more oxygen per ATP produced. In human studies, the effect of temperature on ATP production during exercise has been explored. A study examining dynamic knee-extensor exercise found that both aerobic and anaerobic ATP production rates were similar under normal (~34°C) and elevated (~38°C) muscle temperatures, suggesting that moderate increases within this range may not significantly alter ATP synthesis during such activities (Ferguson et al., 2006).

Aged muscle has demonstrated distinct phosphocreatine profiles linked to the level of sarcopenia experienced within the muscle. (Hinkley et al., 2020) compared PCr levels between 17 non-sarcopenic older adults (71 ± 1 y) and 7 sarcopenic adults (71 ± 2 y). Sarcopenic adults had 17% lower levels of PCr (from 27.9 mM to 23.1 mM) in the quadriceps muscles, the reduction in PCr stores is related to the decrease in muscle mass. In addition to decreased PCr levels, increased levels of creatine were found in the muscle. This has been speculated to be due to the inability of the muscle to form PCr at rest, specifically mitochondrial dysfunction being unable to process the creatine into PCr. As previously mentioned in 2.1.9, ATP generation within the mitochondria is diminished in older adults as the sympathetic nervous system adapts to age related sedentary living in conjunction with continued and increased levels of oxidative stress through the generation of reactive oxygen species degrading the mitochondria in older adults however to what extent this system is responsible for mitochondrial dysfunction in aging remains unclear (Trifunovic & Larsson, 2008). However, it is worth noting that these detrimental changes with age can be slowed or reversed with physical activity, Berg et al., (2018) examined the intramuscular and metabolic adaptations of 8 weeks of maximal knee extension training within 10 older individuals (75 ± 9 y). After 8 weeks of maximal strength training ATP generation from anaerobic glycolysis was increased 40% (10 ± 7 mM \cdot min $^{-1}$; 14 ± 7 mM \cdot min $^{-1}$) and a 24% increase in the creatine kinase reaction within the quadricep muscle (31 ± 10 mM \cdot min $^{-1}$; 41 ± 10 mM \cdot min $^{-1}$), this increase in substrate management and production aided in increased dynamic (23 ± 6 kg to 30 ± 9 kg) and isometric contractile strength (133 ± 36 N.m to 147 ± 49 N.m). Expression of fast myosin heavy chain, quadriceps muscle volume, and submaximal cycling net efficiency were also increased with 8 weeks of maximal strength training. These results suggest that strength training may be an effective counter measure to preserve muscle metabolic function and substrate production within muscle during aging.

2.1.9 Force length relationship

The strength of a muscle contraction is partly limited by the initial length of the muscle fibre prior to contraction, a construct known as the force-length relationship, whereby the potential maximum cross bridge formation is determined by the amount of overlap between the myosin and actin filaments within the sarcomere (Gordon et al., 1966). For maximal force production actin and myosin must optimally overlap, occurring during a short window of sarcomere length believed to be between 80-120% of resting length (Figure 2. 7.). During sarcomere lengthening beyond >120% of resting length, the actin-myosin overlap is diminished, and fewer cross bridges form, resulting in a weaker contraction. Conversely, as the sarcomere contracts past optimal range i.e., <80% of resting length, the z disks at the end of the sarcomere restrict

further filament contraction. Non-uniform sarcomere lengths help maintain consistent muscle contraction compared to individual sarcomere or muscle fibres and allow for higher force production at the extremes of contractile length (Morgan et al., 2000). During knee extension tasks the leg may start with a 110° bend at the knee, the sarcomeres in the quadriceps would start elongated past their optimal: around 160-180% in figure 2. 7. As the leg extension task progressed the angle at the knee would decrease and the sarcomere length would begin to reach optimal range around 60° to 80° of bend at the knee. After maximal force had been produced, the leg would continue to extend to a 0° where it would reach the 60% of resting sarcomere length as seen in figure 2. 7. Older individuals have similar optimal sarcomere lengths to younger adults; however, they experience a greater decline in strength within the optimal range of sarcomere length. Thompson et al., (2018) had 21 young (22 ± 3 y) and 23 older (73 ± 6 y) individuals complete maximal isometric knee extensions at 20°, 60°, and 90° of joint angles. Older participants produced 32% less force within the optimum range (60-90°) (168 N.m vs. 113 N.m) however, force produced at the extremities of contraction (20° joint angle) was only 27% weaker in older adults (101 N.m vs 73 N.m). This suggests that older adults are maintaining force production capabilities outside optimal lengths. This phenomenon may be due to the knee extensor muscles working at extremes during regular locomotion, walking typically begins far outside of an optimal sarcomere length with a joint angle of between 5° to 20° and only moving through $60 \pm 3^\circ$ of movement during a normal stride (Wurdeman et al., 2017) if walking is the main form of lower body exercise this may explain how force production outside of optimal ranges is preserved while function within optimal ranges is lost.

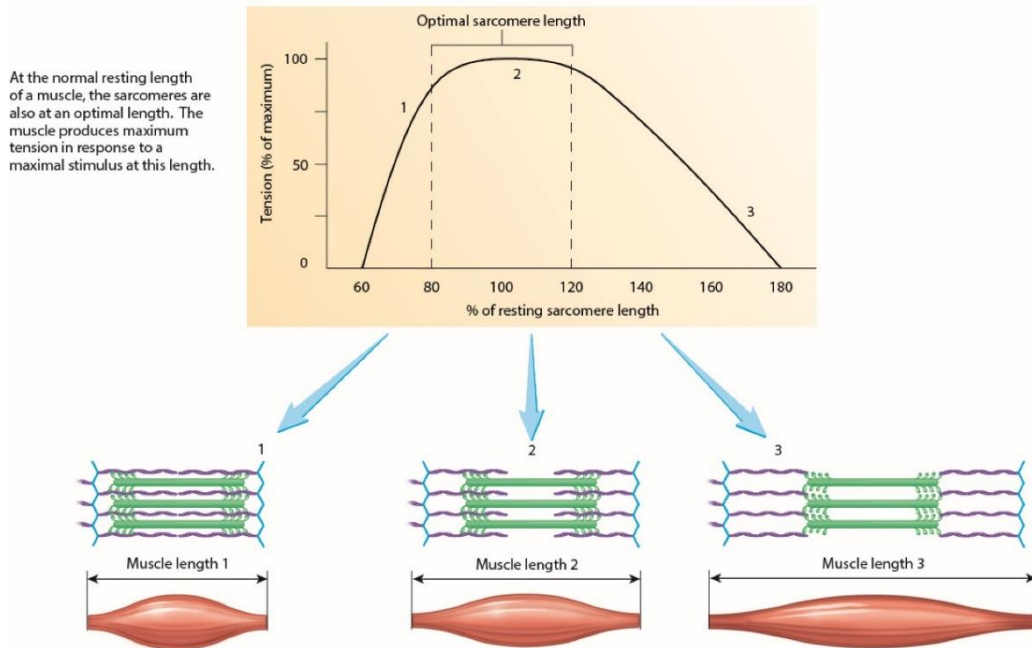


Figure 2. 7. Force length relationship of the sarcomere length. Demonstrating actin and myosin overlap during differing contractile length. (Adapted with permission from VanPutte, 2020)

The force produced by a muscle or total muscle tension is the resultant force of all actions within the muscle, there are two key forces: active tension, the force produced by the muscle during contraction and passive tension, the elastic force of the muscle and connective tissue. Force produced by the muscle is transferred through the tendon unit to the bone, resulting in movement. The tendon unit is often represented as a spring (Kubo et al., 2000), lengthening and shortening with the muscle, storing, and releasing elastic energy (Ishikawa et al., 2007). A stiffer tendon allows for more rapid and efficient transfer of force from muscle to bone (Urlando & Hawkins, 2007).

2.1.10 Force velocity relationship

The velocity of a muscle's contraction influences the capacity of force production, best described as an inverse hyperbolic relationship between muscular tension and shortening velocity (Newhard et al., 2019). The force-velocity relationship in dynamic muscle contractions describes the inverse correlation between the force a muscle produces and the speed at which it changes length. During concentric contractions (muscle shortening), as contraction velocity increases, the force generated decreases. Originally the increased velocity of a muscle's contraction leading to a reduced force output was believed to be caused by a viscous resistance inside the muscle or inhibitory effect of the nervous system (Gasser & Ill, 1924). However, it has since been discovered as an inherent property of muscle: at higher contraction

velocities fewer actin and myosin cross bridges can form in a given time, resulting in decreased force production. Maximum shortening velocity results in minimum force production and vice versa. While isometric contraction results in higher force production than concentric contraction, under negative contraction velocity, the rule also holds true; eccentric contraction can generate the highest force of any muscle contraction. Eccentric contractions utilise the elastic countermovement of the muscle to aid in force production, it has also been theorised that during maximal eccentric contraction myosin cross bridges are stretched to their limit, broken and then reattach further down the filament (Gulch, 1994). While the relationship is clear in a single isolated muscle or during simple exercise such as cycling, during more complex movements, such as a countermovement jump, the relationship can be obscured, with multiple joints and muscle groups acting at one determining overall force output.

Aging typically results in reductions of force generating capacity right across the force velocity spectrum due to sarcopenia, with the greatest decline occurring in concentric contractions. Hortobágyi et al., (1995) report that during concentric contractions older individuals produced only 58% of the force of their younger counterparts however eccentrically they produced 80% of the younger populations force. This phenomenon was first observed in women (Vandervoort et al., 1990) and later in men (Poulin et al., 1992). The preservation of eccentric strength with age was found to be independent of muscle mass, muscle fibre type, or size. While it remains unclear as to the underlying mechanisms preserving eccentric strength it is speculated that the greater quantity of intramuscular connective tissue found in older adults provides a relative increase in passive resistance within the muscle during lengthening (Hortobágyi et al., 1995). This hypothesis was tested by Porter et al., (1997) where no increase in passive tension was discovered in older individuals however, a slower rate of torque development suggested that a slower rate of cross cycling was occurring within the muscle while this resulted in slower force development it was speculated to be advantageous to eccentric contractions.

Estimated maximal contraction velocity has been found to be lower in older individuals compared to the young. Older individuals achieved 83% of the younger cohort's contractile velocity (Toji & Kaneko, 2007). While the mechanisms underpinning slower maximal contractile velocity remain unclear, *in vitro* studies support that ageing has a detrimental effect on contractile function. Larsson et al, (1997) took skinned muscle fibres from the *vastus lateralis* of 4 young (25-31 years old) and 4 old (73-81 years old) participants and observed that the maximum unloaded shortening velocity was significantly lower in the older group whereby type I fibres achieved 57% of the velocity of the younger group and type IIa fibres achieved 72% of the younger group's velocity. The maximal shortening velocity of a muscle provides a link between its macroscopic properties and the biochemical reactions

underpinning movement aged muscle demonstrates a decreased ability to generate force and contract rapidly, likely due to the aforementioned reduced cross cycling rate within the myofilament (further explained in 2.1.6 Force production (Sliding Filament Theory)).

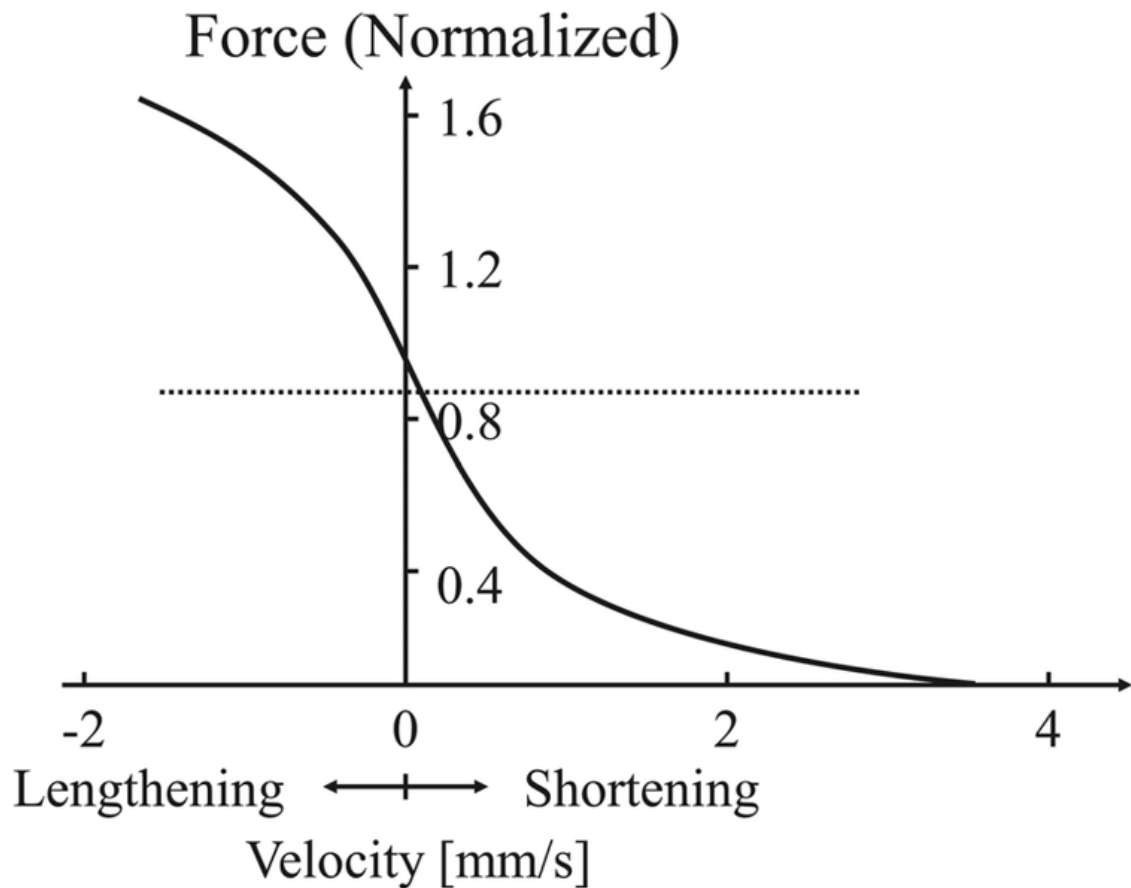


Figure 2. 8. Theoretical force velocity relationship curve of a single muscle, depicting eccentric, isometric and eccentric contraction. Adapted from (Tanaka et al., 2019)

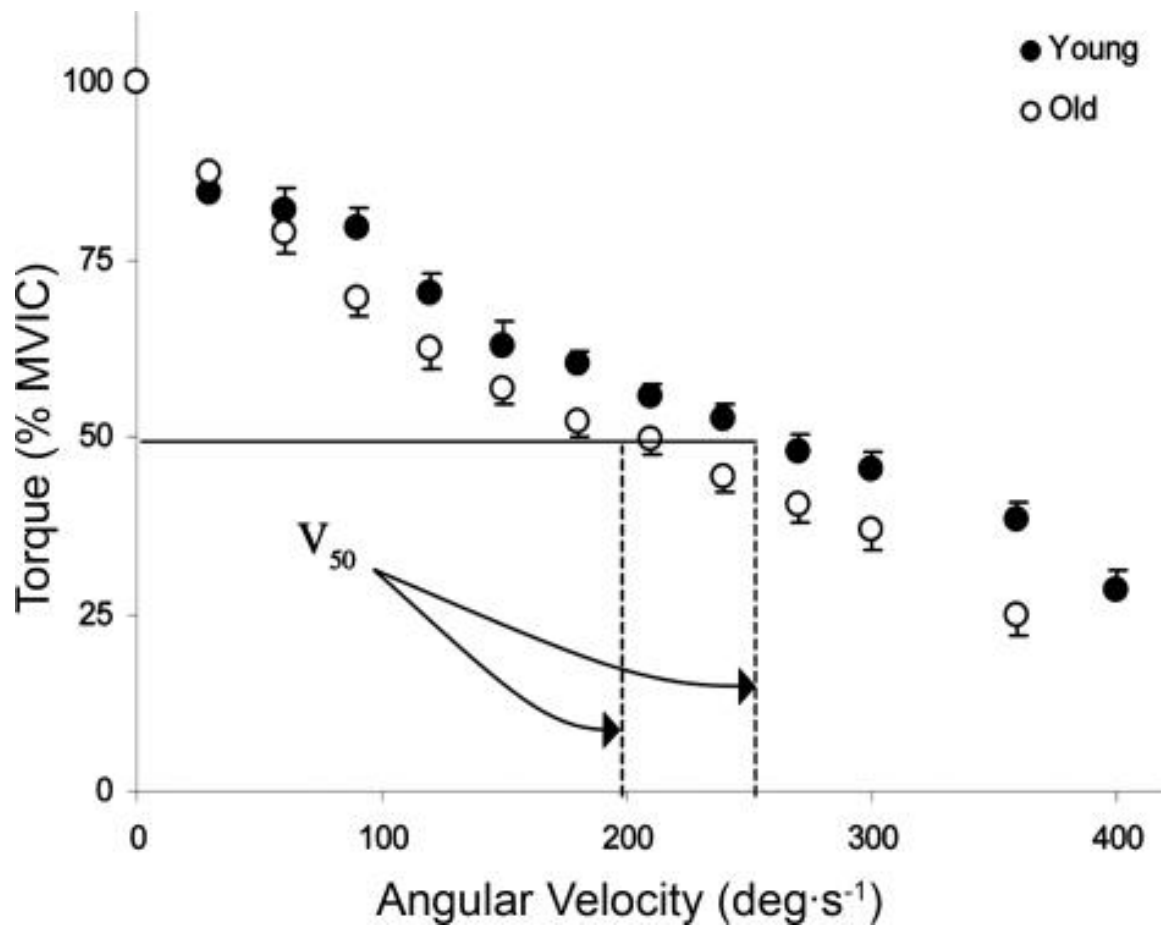


Figure 2. 9. A comparison of force-velocity relationships in young and older groups. Shown is mean \pm SE torque produced across the range of angular velocities for young and older groups, expressed as a percentage of peak isometric torque [maximal voluntary isometric contractions (MVIC)]. Adapted from Callahan & Kent-Braun, (2011)

2.2 A review of published literature examining passive heating to improve muscle function and the reliability of muscle function assessment

The use of passive heating for therapeutic or ergogenic means can be traced back to ancient Roman hot baths for curing oedema and continued in popularity for psychiatric treatments well into the 19th century (Papaioannou et al., 2016). Passive heating's usage in antiquity has since evolved, *in vitro* animal muscle tissue testing began investigating the role of heating on muscle function, early work by Truong et al. (1964) investigated the role of temperature on 30 skinned rat muscle fibres. The authors discovered a 50% reduction in contraction time and 60% reduction in half relaxation time (the duration it takes for a muscle to relax halfway after it has contracted) in the skinned fibres at the hotter temperature 41°C as opposed to 30°C. During half relaxation time, the muscle is still partially contracted, which means that it cannot fully contract again until complete relaxation has occurred. Truong et al. (1964) suggest that hyperthermia results in increased calcium release and reuptake from the sarcoplasmic reticulum which is responsible for decreasing half relaxation time. After a proof of concept that heating influenced muscle function human trials initially began focusing endurance tasks such as cycling (Sargeant, 1987) while strength-based research focused on isometric contractions (Thornley et al., 2003).

2.2.1 Foundational insights into the role of temperature on muscle function

Initial research investigating the role of temperature on the contractile properties of muscle (Bennett, 1984) provided a comprehensive analysis of how temperature influences vertebrate skeletal muscle performance. Bennett (1984) observed that rates of force development, contraction, and relaxation exhibit Q_{10} values (the degree of temperature dependence a muscle exhibits) of approximately 2, indicating that rates of force development, contraction, and relaxation double in rate with every 10°C increase in temperature. Conversely, maximal isometric force generation during both twitch and tetanic contractions showed minimal thermal dependence, with Q_{10} values close to 1.0, suggesting that peak isometric force output remains relatively stable across a range of temperatures. Bennett (1984) also highlighted that muscle performance generally lacks acclimation to varying thermal environments indicating a limited capacity for physiological adaptation to varying thermal conditions. Despite a lack of adaptations to chronic heat exposure, rate processes in muscle function remain strongly temperature dependent. This intrinsic thermal dependence observed *in vitro* is mirrored *in vivo*, where time-dependent activities, such as rapid movements, are enhanced at higher muscle temperatures, while maximal force generation remains largely unaffected by temperature changes.

Continuing this line of research Ranatunga et al. (1987) investigated the effects of temperature on the contractile properties of human skeletal muscle. Using the adductor pollicis muscle, they conducted isometric contractions at temperatures ranging from 25°C to 37°C. The researchers observed that the maximal isometric force remained relatively stable across this temperature range. However, they noted significant temperature dependence in contraction speed parameters: both the time to peak tension and half-relaxation time decreased with increasing temperature, exhibiting Q_{10} values of approximately 2. This again indicated that rate processes doubled in speed with every 10°C rise in temperature. As seen in figure 2. 10, increased temperature improves the rate of muscle function in human muscle fibre.

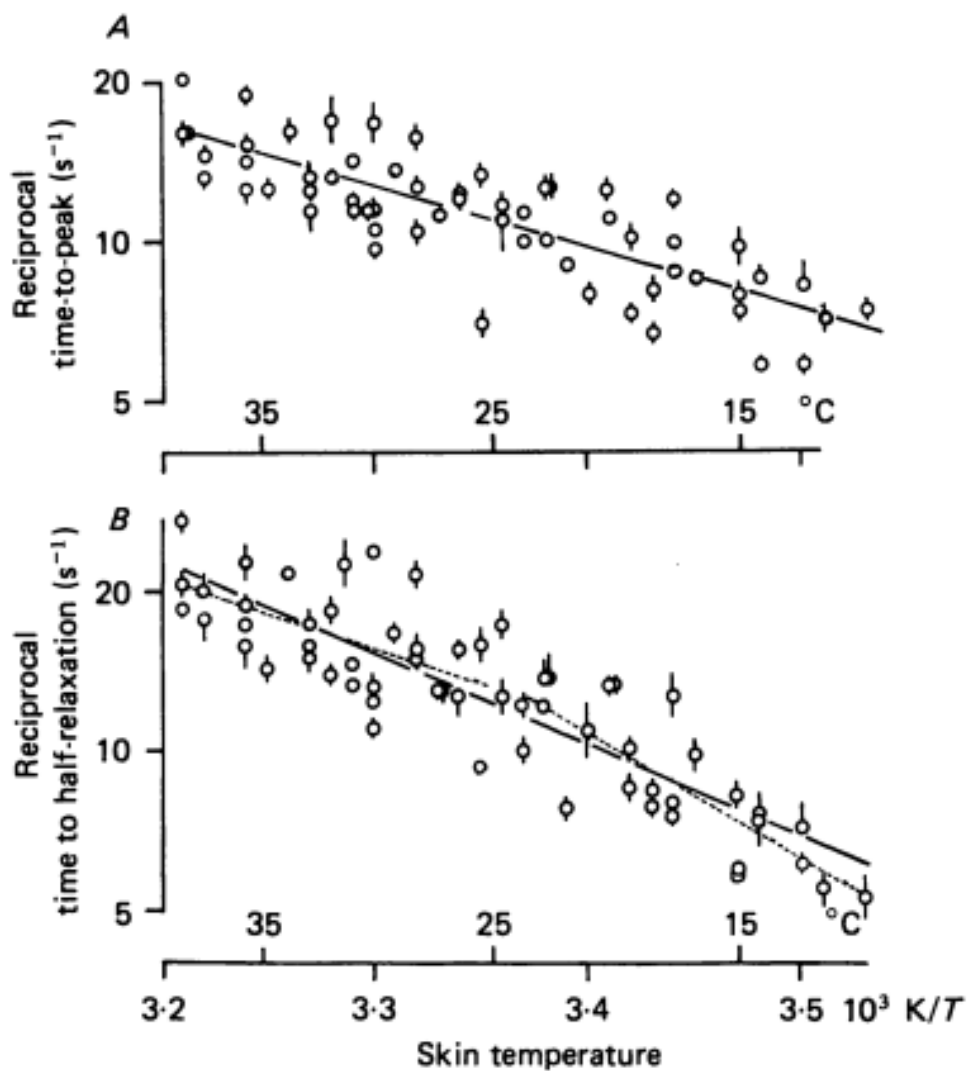


Figure 2. 10. Time to peak (A) and time to half relaxation (B) measured from maximal twitch contractions taken across a series of skin temperatures. Adapted from (Ranatunga et al., 1987)

Further investigating the mechanisms within animal fibres, Ranatunga. (1994) described how, after passive heating, skeletal muscle fibres exhibit increased tension due to enhanced cross-bridge cycling and calcium-independent activation of thin filaments. As temperature rises above 30°C, the troponin-tropomyosin regulatory system becomes inactivated, leading to an increased sensitivity of muscle fibres to stretch. This results in a heightened rate of cross-bridge cycling, even in the absence of calcium, which contributes to greater muscle tension. Additionally, the titin-myosin composite filament system becomes more sensitive to stretch under elevated temperatures, further influencing muscle contractility. These changes enhance muscle performance, particularly in rapid movements, by making the contractile process more efficient at higher temperatures.

These findings (Ranatunga, 1994; Ranatunga et al., 1987) align with the previous research on vertebrate skeletal muscle (figure 2. 10.), which demonstrated that while maximal force generation is relatively temperature-independent, contraction velocity and relaxation are strongly affected by temperature changes. This thermal sensitivity of rate processes has significant implications for muscle function during rapid movements and activities requiring quick muscle responses, as higher muscle temperatures facilitate faster contraction and relaxation cycles.

The work of Sargeant. (1987) examined how variations in muscle temperature affect short-term dynamic exercise performance. Four male subjects performed 20-sec maximal sprint efforts on an isokinetic cycle ergometer at a constant pedalling rate of 95 revolutions per min under four different temperature conditions: resting at room temperature (22°C), and after 45 min of leg immersion in water baths at 44°C, 18°C, and 12°C. The corresponding muscle temperatures at a depth of 3 cm were approximately 36.6°C, 39.3°C, 31.9°C, and 29.0°C, respectively.

The findings revealed that higher muscle temperatures enhanced both peak torque and power output during the sprint efforts (figure 2. 11.), with moderate contractile speeds (140 rev.min⁻¹) receiving greater enhancement than slower contractions (54 rev.min⁻¹). Specifically, at a muscle temperature of 39.3°C, subjects exhibited the highest peak power, while lower muscle temperatures (31.9°C and 29.0°C) resulted in diminished force and power output. This study underscored the importance of elevated muscle temperature in optimizing dynamic exercise performance, suggesting that increasing muscle temperature prior to high-intensity activities can significantly improve outcomes. This study was foundational in demonstrating how passive heating can positively influence human muscle function during exercise.

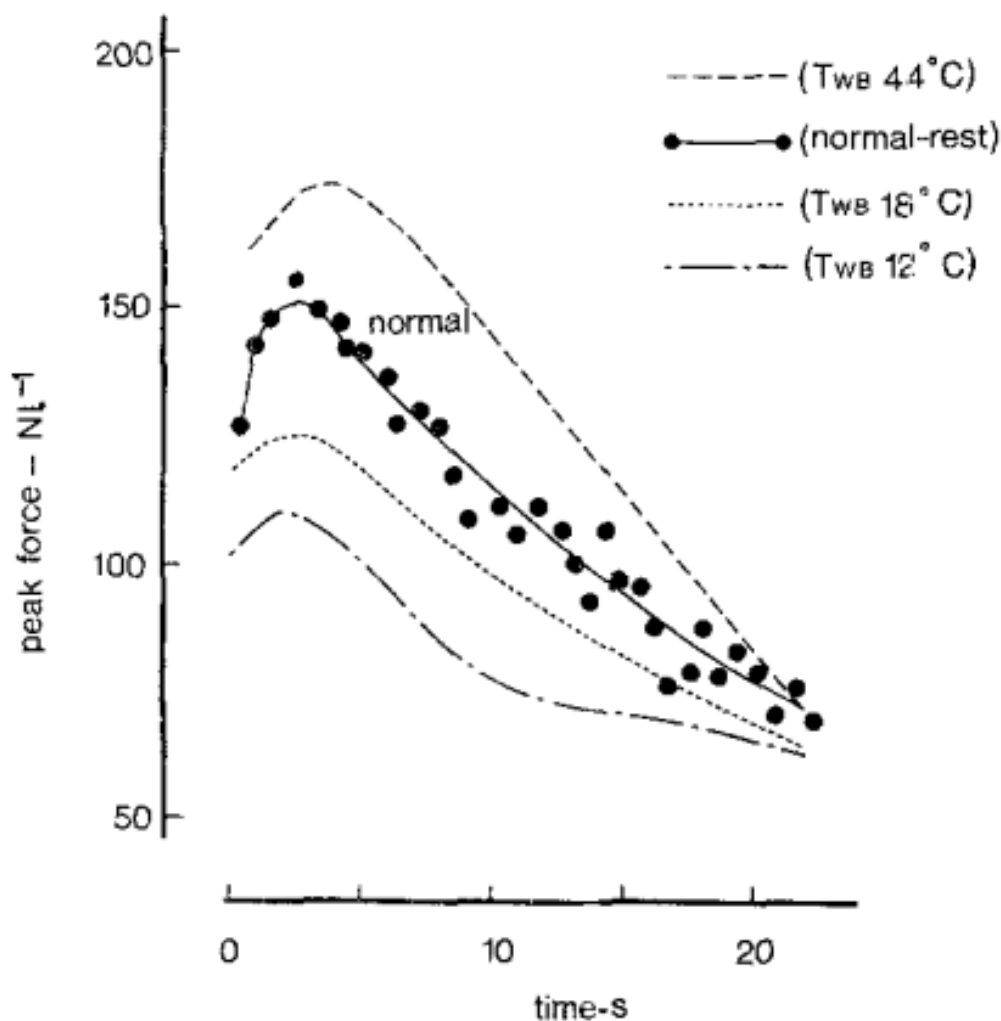


Figure 2. 11. Peak force production during maximal cycling at 95 rev^{-1} following 44°C water immersion (T_{WB} , Water bath temperature) (dashed line), normothermic (solid line), 18°C water immersion (dotted line) and 12°C water immersion (dashed and dotted line). Adapted from Sargeant, (1987a)

2.2.2 Contemporary research into the role of muscle temperature on muscle function

Research into physiological responses to acute hyperthermia of the whole body and/or isolated limbs, and chronic heating therapy interventions have become a contemporary area of study. Some authors have even claimed that passive heating can serve as an exercise mimetic with the potential to elicit similar positive health benefits to those of physical activity (Cullen et al., 2020). Acute passive heating, which leads to increases in muscle temperature, has been shown to promote positive changes in skeletal muscle structure and function (Kim, Monroe, et al., 2020; Rodrigues et al., 2021) however, at the current time, responses to acute skeletal muscle heating remain incompletely understood, particularly regarding the influence

of incremental temperature on muscle force production across a range of contraction types, and whilst benefits of heating have been described in young individuals, the efficacy in clinical, vulnerable, or older individuals has been seldom reported.

Recent commentary on the topic has stated that inducing local skeletal muscle hyperthermia via passive heating presents an opportunity to improve strength and power in populations who require or would benefit from enhanced muscle function during physical activity and daily living tasks (Rodrigues et al., 2022). This includes those individuals who suffer from low contractile velocity and function, such as the elderly and populations suffering from temporary and permanent muscular injury. This literature review will examine these claims against the existing evidence base and identify the gaps in the research with an emphasis on muscle responses, force production and fatigability, in young and older populations following localised passive heating.

2.2.3 Heating methodologies and their effect on muscle temperature

In our laboratory, normal resting muscle (*vastus lateralis*) temperature has been measured to be between $34.7 \pm 0.9^{\circ}\text{C}$, $33.8 \pm 1.5^{\circ}\text{C}$ (Gibson et al., 2023), $34.8 \pm 0.8^{\circ}\text{C}$ (Gibson et al., 2014), 34.9°C (Pearson, 2010). Muscle temperature has been measured to be depth dependant between 1.0 cm and 3.5 cm ($36.18 \pm 0.18^{\circ}\text{C}$ and $34.92 \pm 0.17^{\circ}\text{C}$, respectively) (Rodrigues, Trajano, et al., 2020). There is a multiplicity of methods to increase muscle temperature within the current published literature including microwave diathermy, environmental chambers, heat- and steam-generating sheets, ultrasound stimulation, heat pads, thermal blankets, water perfused garments, and warm/hot water immersion (McGorm et al., 2018). Muscle temperature is usually increased prior to exercise through an active warm up, and usually this warm up consists of low to moderate intensity exercise, that sometimes reflects the activity that is being prepared for. To demonstrate the thermic effect of an active warm up, 15 min of a modified FIFA 11+ soccer specific warm-up raised muscle temperature by 3.2°C (Marshall et al., 2015). Different heating methodologies have varying heating rates, and hot water immersion has shown increases in muscle temperature by 2.8°C after 90 mins (Rodrigues, Trajano, et al., 2020) whereas microwave diathermy has been shown to increase temperature by 7.1°C in 20 mins (Nosaka et al., 2007). Table 2.2 presents a full overview of heating rates across a range of heating methodologies and timings. Despite differences in heating methodology, the application of passive heating produces a similar rate of temperature change, with a rapid increase in the initial 30 min which is followed by smaller increases over time before reaching a plateau, this effect is demonstrated in figure 2. 12.

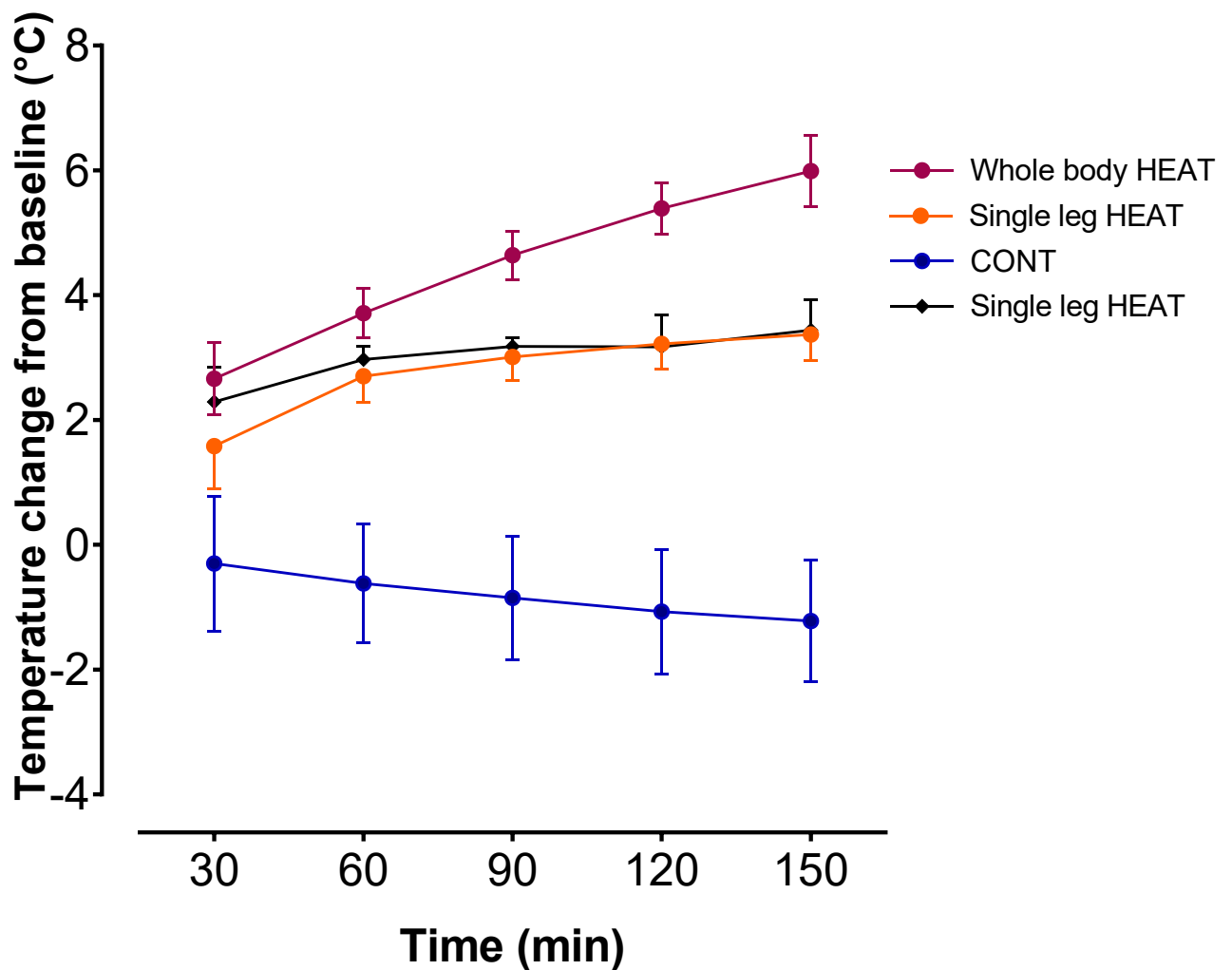


Figure 2. 12. Local temperature change from baseline of the *vastus lateralis* in response to single leg, double leg and whole body passive heating over time. Figure displays data from prior work, red circles whole body heating (Watanabe et al., 2024), orange circles single leg heating (Watanabe et al., 2024), blue circle thermoneutral control (Watanabe et al., 2024), black diamond single leg heating (Gibson et al., 2023).

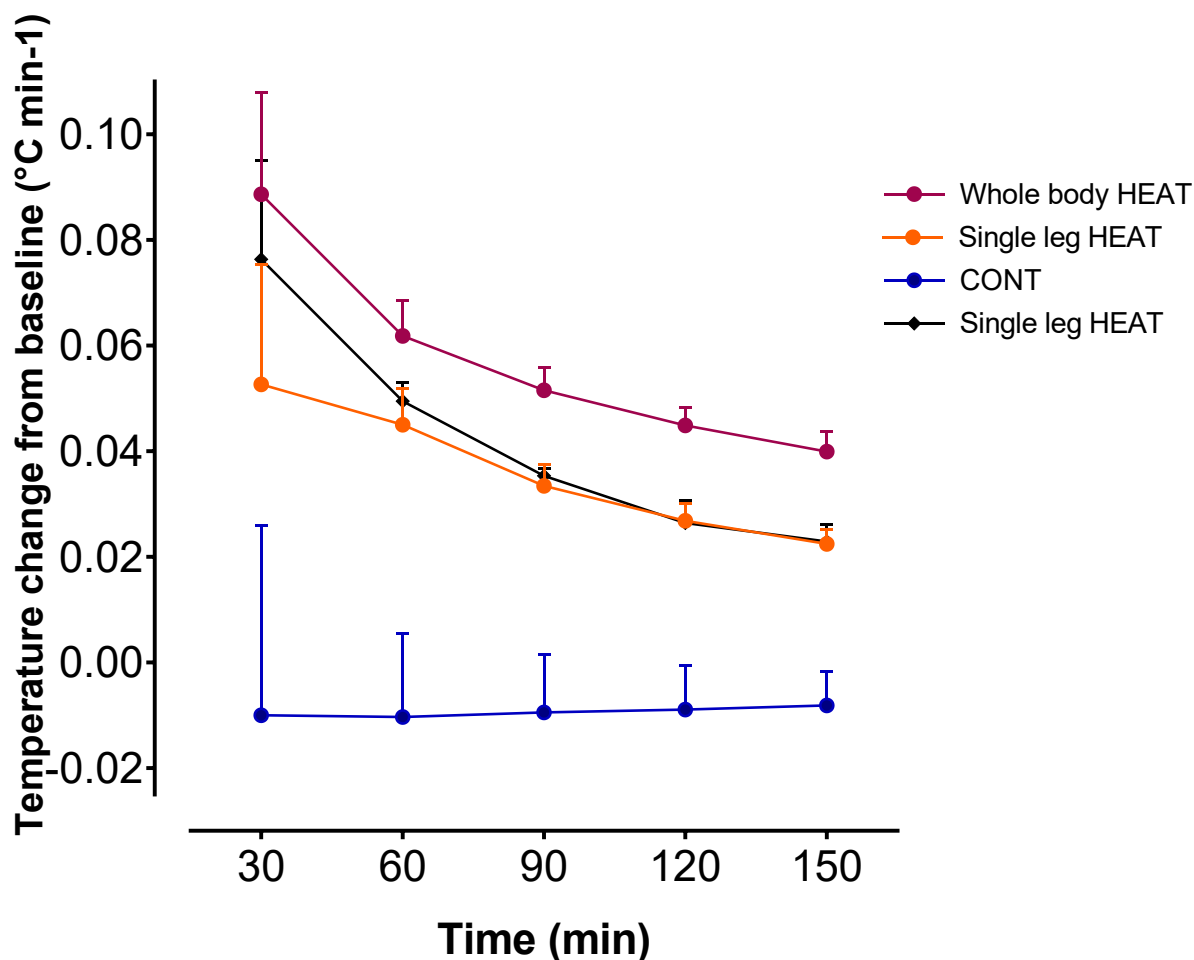


Figure 2. 13. Local temperature change in °C min⁻¹ from baseline of the *vastus lateralis* in response to single leg, double leg and whole body passive heating over time. Figure displays data from prior work, red circles whole body heating (Watanabe et al., 2024), orange circles single leg heating (Watanabe et al., 2024), blue circle thermoneutral control (Watanabe et al., 2024), black diamond single leg heating (Gibson et al., 2023).

Heating methodologies usually either produce whole body heating effects (water immersion and environmental chambers) whereby increases in both muscle and core temperature are observed, or localised heating effects (i.e. water perfused garments, heat pads, and diathermy) whereby core temperature remains unaffected but muscle temperature increases. Local heating methods may provide a weaker thermal stimulus and struggle to raise muscle temperature significantly above core levels due to the body's natural heat dissipation mechanisms. Increases in core temperature have been shown to decrease voluntary and evoked maximal isometric muscular function (Morrison et al., 2004) due to a decrease in neural activation and central drive. Therefore, when seeking to increase muscular contractile function, it is important to maximise the local heating effect at the muscle and minimise the

increases in core temperature. Whole body heating has previously demonstrated chronic benefits to passive heating, such as increased mitochondrial function and the expression of certain beneficial genes (Ihsan et al., 2020). It had been assumed that localised passive heating lacked the thermal effect to drive changes in gene activation; however, recent research demonstrated that localised heating creates a demand to produce heat stress proteins and increases mitochondrial biogenesis signalling (Gibson et al., 2023). This thesis will focus on water perfused garments as they have been identified to be a safe and effective method of passively increasing local muscle temperature of the lower limbs (Chiesa et al., 2015; Heinonen et al., 2011; Koch Esteves et al., 2021; Watanabe et al., 2024).

Table 2 1.

Rates of muscle temperature changes reported across a variety of passive heating modalities

Study	Increase in muscle temperature (°C)	Increase in muscle temperature (°C.min ⁻¹ .min)	Method of heating	Duration of Heating
(Koch Esteves et al., 2021)	3.6 ± 0.3°C	0.02°C.min	50°C Water perfused garment	180 min
(Rodrigues, Trajano, et al., 2020)	2.8 ± 0.1 °C	0.03°C/min	Lower body submersion in 42°C water	120 min
(Gibson et al., 2023)	3.5 ± 1.5°C	0.04°C/min	50°C water perfused garment	90 min
(Morton et al., 2007)	3.6 ± 0.5°C	0.06°C/min	Lower body submersion in 45°C water	60 min
(Morton et al., 2007)	3.6 ± 0.5°C	0.06°C/min	Single leg immersion in 45°C water	60 min
(Ihsan et al., 2020)	4.2°C ± 1.3°C	0.07°C/min	50°C water perfused garment	60 min
(Kuhlenhoelter et al., 2016)	6.5 ± 0.1°C	0.07°C/min	48°C water perfused garment	90 min
(Ihsan et al., 2020)	5.0°C ± 0.5°C	0.08°C/min	Chamber 44–50°C, 50% humidity	60 min
(Mitchell et al., 2008)	1.8 ± 0.4°C	0.09°C/min	40 W microwave diathermy	20 min
(Draper et al., 1999)	3.8 ± 1.2°C	0.25°C/min	48 W microwave diathermy	15 min

2.2.4 Muscle fluid and blood flow

A central outcome of muscle heating is an increased in blood flow to the heated tissue, (Koch Esteves et al., 2021) investigated how local heating affects blood flow and tissue oxygenation in different regions of the leg, aiming to determine whether temperature-sensitive mechanisms in small blood vessels regulate circulation during heat exposure. The study included three heating protocols: heating the entire leg for three hours, heating only the upper leg for one hour with a cooling break, and heating only the lower leg for one hour. Measurements of temperature, blood flow in major leg arteries, and tissue oxygenation showed that heating the whole leg increased leg temperature by an average of 4.2°C and tripled blood flow ($1122 \pm 250 \text{ ml}^{-1}$ compared to a normothermic control of $360 \pm 110 \text{ ml}^{-1}$) without affecting core body temperature. Heating either the upper or lower leg also significantly increased temperature, blood flow, and oxygenation in the heated regions, but did not affect adjacent areas, confirming a localized response. The increases in blood flow and oxygenation correlated with the rise in local temperature, and the lack of changes in overall blood pressure or large artery diameter suggested that small blood vessels were responsible for these effects rather than larger arteries expanding. The study concludes that local heating can substantially and sustainably improve blood flow and oxygen supply through temperature-sensitive mechanisms in small blood vessels, with potential therapeutic applications for improving circulation in specific limb regions.

The increase in blood flow following passive heating to the muscle promotes the movement of additional blood into the muscle tissue as vasodilation reduces vascular resistance allowing additional blood plasma to fill interstitial spaces surrounding muscle fibres. This increase in intramuscular fluid, in tandem with localised vasodilation, increases peripheral tissue perfusion rates facilitating a greater movement of fluid to the muscle tissue (Sugi et al., 2015). Intramuscular fluid is incompressible during muscle contraction reducing muscle deformation and acting as a spring within the muscle (Eng et al., 2018), additional intramuscular fluid increases internal pressure in the muscle, increasing stiffness of the muscle tendon unit, and a stiffer medium then allows a more efficient and rapid transfer of force. Passive tension, the stored elastic potential force of the muscle, also increases within the muscle as intramuscular fluid increases (Eng et al., 2018). Overall, the incompressibility of intramuscular fluid allows muscles to function as more effective springs, optimising force transfer, reducing energy waste, and enhancing both strength and movement efficiency.

2.2.5 Ca²⁺ handling and kinetics

Muscle contraction is regulated by calcium (see 2.1.9). Local hyperthermia does not change total muscle Ca²⁺ concentration, however, it modifies a muscle's handling of Ca²⁺, bolstering contractile function by upping the rate of cross bridge formation CITE. Heat stress triggers the transient receptor potential vanilloid 1 (Trpv1) channel which releases Ca²⁺ from the sarcoplasmic reticulum into the myoplasm at a higher rate (Obi et al., 2019). The heat induced increase in myoplasmic Ca²⁺ uncovers the active site between the actin and myosin heads at a greater rate, therefore allowing for cross bridges to be formed and muscle contraction to occur. The observed decreases in twitch contraction half relaxation time following acute passive heating (Davies & Young, 1983; Rodrigues et al., 2021) suggest that muscle hyperthermia may stimulate the sarcoplasmic reticulum Ca ATPase (SERCA) pump to increase the rate of Ca²⁺ reabsorption into the sarcoplasmic reticulum. This works in conjunction with the previously discussed increased rate of Ca²⁺ deployment to decrease half relaxation time.

The effect of improved Ca²⁺ handling and kinetics after local hyperthermia can be collectively observed in the 32% (25 ms to 17 ms) decrease in time to peak force during isometric stimulated contraction of an isolated muscle fibre warmed by submerging in a water tissue bath (Segal & Faulkner, 1985). Increased maximal single evoked contractile function has been observed in thigh muscle heated by a submersion in 46°C water (22% reduction in half-relaxation time) (Davies & Young, 1983) (Racinais et al., 2017). Furthermore, a 15% reduction in electromechanical delay after a maximal evoked twitch in thigh muscle was observed after 90 mins of immersion in 42°C water (Rodrigues et al., 2021). Ca²⁺ handling is a key component in muscle contraction; the aging process diminishes a muscle's natural capability to properly release and reuptake Ca²⁺. Passive heating may be a useful tool to aid in transiently restoring Ca²⁺ handling in older adults. Whilst there has been some promising evidence displaying an interaction between heating and Ca²⁺ handling more research is needed into exactly how this ageing interacts with heating in relation to Ca²⁺ production, release, and reuptake.

2.2.6 Reliability of dynamometer muscle function assessment

Dynamometry is a widely used method for the objective assessment of muscle strength and function, allowing for the quantification of force, torque, power, and work during voluntary muscle contractions. In sport and health science, isokinetic dynamometers are considered a gold-standard tool (Van Driessche et al., 2018; Wilk et al., 2024) for evaluating neuromuscular performance due to their ability to precisely control movement parameters and provide highly

reproducible measurements in both younger and older cohorts (Parraca et al., 2022; Van Driessche et al., 2018). These devices enable the assessment of muscle function under controlled conditions across different contraction modes, including isokinetic, isotonic, and isometric actions. As such, dynamometry is commonly employed in both research and clinical settings to monitor training adaptations, assess injury risk, evaluate rehabilitation progress, and examine age- or disease-related changes in muscle function (Bond et al., 1986; Chang et al., 2023; Gomes et al., 2021; Gordon et al., 2013; Kambič et al., 2020; Rosene et al., 2001). The versatility of isokinetic dynamometry allows for the standardisation of joint angle, contraction velocity, range of motion, and external load, thereby minimising extraneous variability and enhancing measurement accuracy. This level of control is particularly valuable when investigating subtle changes in muscle performance, such as those induced by acute interventions. Consequently, the reliability of measurements obtained from isokinetic dynamometers is a critical consideration when interpreting changes in muscle function.

Isokinetic testing, which involves muscle contractions performed at a constant angular velocity, has consistently demonstrated high reliability across a range of muscle groups, velocities, and populations. Early work by Feiring et al. (1990) showed very high test–retest reliability for knee extension peak torque across multiple velocities (ICC values 0.95–0.97 at 60–300°/s) and similarly high reliability for single repetition work (ICCs 0.95–0.97) in healthy adults, indicating excellent consistency of repeated measures when protocol and positioning are standardised. Subsequent studies have reinforced these findings across different populations and outcome measures. Sole et al. (2007) reported “very high” relative reliability of knee extension and flexion peak torque and work, with ICCs >0.90, standard error of measurement (SEM) between 5% and 10%. In older adult cohorts, reliability remains strong: intra-session ICCs for peak torque and work often exceed 0.94–0.98, with SEM percentages typically ≤10% indicating both excellent reliability and acceptable measurement error (Parraca et al., 2022). The controlled velocity and accommodating resistance of isokinetic contractions reduce the influence of acceleration and deceleration phases, contributing to the strong reproducibility observed in both athletic and clinical populations. As a result, isokinetic testing is frequently used as a reference standard for assessing dynamic muscle strength and detecting meaningful performance changes.

Isotonic testing performed on an isokinetic dynamometer also demonstrates good reliability when external loads, movement velocities, and range of motion are carefully controlled. Unlike traditional free-weight isotonic assessments, dynamometer-based isotonic testing enables precise regulation of resistance and continuous measurement of torque and power

throughout the movement cycle. Webber & Porter (2010) reported good-to-excellent test–retest reliability for isotonic knee extension peak torque and mean torque across multiple contraction velocities, with ICC values ranging from 0.86 to 0.96, and coefficients of variation typically below 10% when consistent loading parameters were used. Power output measures demonstrated slightly greater variability, particularly at higher velocities, but remained within acceptable reliability thresholds (ICC ≥ 0.80). More recently, Van Driessche et al. (2018) examined isotonic strength and power outcomes using a dynamometer-based protocol and found excellent relative reliability for peak torque and mean power (ICC 0.90–0.97), alongside moderate-to-low absolute measurement error (SEM approximately 5–9%). The authors noted that reliability was highest when contraction velocity and range of motion were strictly standardised and when participants completed prior familiarisation sessions. Across studies, reliability appears sensitive to task novelty and movement speed, with lower reliability observed during faster or ballistic contractions and during initial testing sessions.

Isometric testing on an isokinetic dynamometer is widely regarded as highly reliable due to the absence of joint movement, which reduces mechanical variability and coordination demands. Peak torque measurements obtained during isometric contractions consistently demonstrate excellent relative reliability across a range of muscle groups and populations. For example, Toonstra & Mattacola (2013) reported intraclass correlation coefficients (ICC) ranging from 0.95 to 0.99 for isometric knee extension peak torque across testing sessions, with standard error of measurement (SEM) values generally below 5%, indicating minimal absolute measurement error. Similarly, Webber & Porter, (2010) observed good-to-excellent reliability for isometric knee extensor strength (ICC 0.90–0.97) when joint angle and participant positioning were standardised. In clinical and older adult populations, reliability remains high. de Araujo Ribeiro Alvares et al. (2015) demonstrated excellent test–retest reliability for isometric peak torque (ICC ≥ 0.93) across repeated sessions, with coefficients of variation typically under 10% when familiarisation procedures were implemented. More recently, Croci et al. (2023) reported excellent within-session and between-day reliability for isometric dynamometry measures in clinical cohorts, with ICC values frequently exceeding 0.90, and acceptable SEM and minimal detectable change (MDC) values, supporting the sensitivity of isometric assessments for detecting meaningful strength changes. The fixed joint angle and controlled testing environment allow for consistent muscle length and lever arm positioning, making isometric dynamometry particularly useful for assessing maximal voluntary strength and detecting small changes over time. However, reliability remains dependent on participant effort, stabilisation, and verbal encouragement, highlighting the importance of standardised protocols.

2.2.7 Changes in isometric muscle function after whole body and localised heating

Early *in vivo* study of human contractile performance during isometric muscle action, following passive heating can be observed in the landmark study by Thornley et al, (2003) who reported the relationship between human leg muscle temperature and isometric peak torque and isometric strength endurance during knee extension. The study involved 9 non-trained males, aged 22 ± 3 years, who were affixed with gel pads to create four different temperature conditions across four separate, randomised visits (55°C (hot), 34°C (warm), 22°C (temperate) and -17°C (cold)). Following the 30 min intervention, skin temperature was increased in Hot ($40.1 \pm 0.6^{\circ}\text{C}$) and Warm ($35.7 \pm 1.3^{\circ}\text{C}$) and decreased in cold ($12.4 \pm 2.8^{\circ}\text{C}$) relative to the temperate trial ($29.5 \pm 1.4^{\circ}\text{C}$). Peak isometric torque was measured at 90° across 3×5 sec maximal contractions with 30 sec of rest between repetitions. This was followed by an isometric strength endurance test at 70% of MVC where the objective for participants was to produce the required force for as long as possible. In contrast to the experimental hypothesis, Thornley et al. (2003) found that 1) the applied temperature had no significant effect on isometric peak force production however, 2) the time to fatigue on the isometric strength endurance task was strongly negatively correlated with temperature. This demonstrates that increased muscle temperature is manipulating a factor that is increasing the fatigue rate of the muscle, the mechanisms for this are unclear but are well documented (Morrison et al., 2004; Todd et al., 2005). Peak torque was not different between conditions though cold demonstrated a tendency to reduce (207 ± 40 N.m to 188 ± 56 N.m; $P = 0.06$). Torque impulse, a marker of muscular strength was lower in Hot ($-17 \pm 10\%$ vs Temperate) than Cold ($-7 \pm 26\%$ vs Temperate). Time to fatigue was shorter in Hot (47 ± 15 s; -36%) and Warm (54 ± 28 s; -26%) than Cold (73 ± 40 s), with time to fatigue demonstrating a strong correlation with skin temperature ($r^2 = 0.98$).

Thornley et al. (2003)'s observed decreased muscular time to fatigue in the cold trial was suggested to be controlled by local factors, increased skin temperature may have increased pain sensitivity and inhibitory feedback, or the cold condition may have blunted the feedback loop. Cheung & Sleivert. (2004) investigated the effects of skin and core temperatures on maximal force production during isokinetic knee extensions. Twenty healthy male participants were passively heated in a 42°C water bath until their rectal temperature (T_{re}) reached 39.5°C . Subsequently, they were cooled back to a T_{re} of 38.0°C . At 0.5°C intervals during both heating and cooling phases, participants performed two maximal voluntary knee extensions at angular velocities of 60, 120, and 240 degrees per sec. The results showed that peak torque did not significantly differ across the core temperature range of 37.5 – 39.5°C . However, when skin temperature was reduced, even with a maintained elevated core temperature of 39.5°C , peak torque decreased significantly across all

contraction speeds. The primary outcomes of the study conducted by Cheung & Sleivert. (2004) indicate that local heating has a null effect on MVC force production and is detrimental to isometric endurance, conversely cooling appears to provide a benefit through a decrease in the stimulation frequency required for the same force production. The temperature of the muscle during recovery between the first and sec maximal isometric peak torque test may have been a confounding factor within this study, as the cold condition may have resulted in localised vasoconstriction within the muscle, restricting the rate of removal of metabolites (such as hydrogen ions and lactate) from the muscle tissue. Other limitations of this study include a limited cohort of only young and healthy participants tested and a lack of muscle temperature measurements which make it impossible to know if muscle temperature or skin temperature is the mediating factor or if these results are applicable to older adults. Recent work (Thomas et al., 2006) discussed in sections 2.2.3, has since found that increases in core temperature can reduce central drive to produce force, and therefore undermine potential local benefits occurring following skeletal muscle heating.

Further investigation into passive heating on isometric muscle function can be seen in, Morrison et al. (2004) whereby relationship between maximal isometric force production, voluntary activation, skin and core temperature was tested. A key difference from Thornley et al. (2003) was the use of a whole-body heating method. It was hypothesised that an increase in both skin and core temperature would result in a decrease in neuromuscular function as the body seeks to prevent cell damage from overheating. Twenty-two young males were heated via a water perfused suit, circulating 52°C water until core temperature reached $39.0 \pm 0.1^\circ\text{C}$. Participants completed a 10 sec maximal isometric knee extension contraction with a supramaximal interpolated twitch imposed to examine changes in voluntary activation of the right quadriceps muscle group. These measurements were taken at every 0.5°C increase in core temperature until 39°C and every 0.5°C decrease of core temperature during the cooling phase until participants had returned to baseline core temperature. After core temperature returned to baseline (37.4°C) maximal isometric contraction force had decreased from 322 ± 66 N to 281 ± 85 N (-12%). Peak twitch force did not change across the experiment however, half relaxation time (84.79 ms to 65.10 ms), time to peak force (117.83 ms to 110.54 ms), and voluntary activation (83% to 71%) all significantly decreased with heating. From this dataset it is clear that whole body heating does not increase isometric knee extensor force production, rather elevations in whole body temperature impair muscle function. Morrison et al. (2004) suggest that, as improvements in maximal isometric force and voluntary activation were not seen early in the cooling protocol, where skin temperature decreased but core temperature remained high, this indicates that central factors rather than peripheral ones were contributing to the attenuation of muscle function. These results have been suggested to be caused by

hyperthermia impaired central nervous system function through reduced cortical excitability, in addition, elevated muscle temperatures can accelerate metabolic reactions, potentially leading to faster depletion of energy substrates like adenosine triphosphate (ATP) and phosphocreatine (Racinais et al., 2019). This rapid depletion may impair muscle contractility and reduce force production.

Along with physiological explanations, psychological causation may include an increased perception of exhaustion derived from the elevated core temperature, as the authors link an increased core temperature to an increase in perceived fatigue and suggest that the anticipation of fatigue in hyperthermic conditions are a physiological response, designed to prevent dangerous hyperthermia and encourage an individual to rest. A lack of muscle temperature measurement is also present in this study leading to the speculation that peripheral factors are obsolete whereas, muscle temperatures would have provided better understanding of the thermal conditions in the muscle during different timepoints of the study.

The main studies in this section (Cheung & Sleivert, 2004; Morrison et al., 2004; Thornley et al., 2003) found that neither localised nor whole-body passive heating improved isometric or isokinetic contractions. These studies isolated active muscles during multi-sec static contractions, limiting the observation of benefits like increased muscle blood flow and contraction velocity. Additionally, the lack of direct muscle temperature measurements in these studies hinders confirmation of heating-induced temperature changes. Nonetheless, they offer valuable insights into passive heating methodologies. Therefore, this work formed the foundation for passive heating not improving peak force output in isometric contractions; this provided a baseline to compare how heating affected dynamic contractions and aspects of contraction aside from peak force.

Developing upon prior work, Thomas et al. (2006) sought to investigate how local and central feedback modulate voluntary muscle activation, focusing on maximal isometric voluntary contraction torque. Young healthy subjects were passively heated using a liquid-conditioning garment to elevate their core temperature from approximately 37.0°C to 39.5°C. One leg (the heated leg) followed the changes in core temperature, while the other leg (the thermoneutral leg) remained at a constant temperature with the use of an ice pack-filled sleeve. Results showed that maximal isometric voluntary contraction torque in the heated leg decreased from a baseline mean of 172 N.m to 160 N.m during heating, while voluntary activation decreased from 96% to 91%. In the thermoneutral leg, torque decreased from 178 N.m to 165 N.m, and voluntary activation dropped from 97% to 94%. During the cooling phase, heart rate reserve decreased from 58% to 31%, indicating a reduction in cardiovascular strain, but torque and

voluntary activation did not immediately return to baseline values. Full restoration occurred only when core temperature was lowered back to normal levels. The study concluded that impairments in voluntary muscle activation during brief isometric contractions were primarily due to central factors related to increases in core temperature, rather than changes in local muscle temperature or skin afferent input, highlighting the significant role of core temperature in modulating neuromuscular function under hyperthermic conditions. This finding is central to understanding passive heating research and its application as an ergogenic aid, as increases in core temperature are detrimental to performance and heating protocols need to be targeted and localised to muscles that are going to be engaged in physical activity.

Thomas et al. (2006) suggest that a decrease in central neuromuscular activation led to the outcome of their study as a small, but significant decrease in interpolated twitch contraction time (131 ms to 106 ms) was observed, which indicates that there was a decreased ability of the central nervous system to fully activate the muscle. Furthermore, the impairment of neuromuscular engagement is believed to be centrally controlled and not influenced by local muscle temperature (Nybo, 2008). Neuromuscular fatigue is attributed to a failure of the nervous system to recruit and activate muscle units, a change in muscle characteristics, or a combined effect of both. However, Thomas et al. (2006) suggest that a decrease in central drive that affected both limbs independently of local muscle temperature is the most likely physiological factor underpinning their results. The instantaneous decrease in skin temperature at the beginning further supports this theory, as skin temperature drastically changed (40.56°C after heating, 34.49 immediately following cooling) while core temperature took longer to decrease (39.66°C at the end of heating, 36.96°C immediately following cooling). Skin temperature can decrease very quickly, however the skeletal muscle underneath, and certainly core temperature, can lag, suggesting a central control.

To conclude, the work of Thomas et al. (2006) agrees with prior work (Morrison et al., 2004) that proposes central hyperthermia as decreasing voluntary maximal force through a decrease in central neural drive and that this occurs independently of local muscle temperature. The decrease in neuromuscular function also seems to be progressive with higher impairments at higher core temperatures. The authors also conclude that there is not a cut-off point, or critical temperature threshold where muscle function is drastically impaired as historically theorised (González-Alonso et al., 1999; Nielsen et al., 1993). This lack of critical threshold aligns with more recent observations (Nybo & González-Alonso, 2015). This study demonstrates the methodological considerations that passive heating studies must parse before planning a study that induces hyperthermia intending to observe muscle function changes.

2.2.8 Changes in rate of force development during local heating

Recently Rodrigues et al. (2021) sought to study the impact of local hyperthermia on muscle function. The authors endeavoured to use isometric contractions to observe the influence of local hyperthermia on early aspects of the contraction such as rate of force development that are more likely influenced by passive heating. Rodrigues et al. (2021) developed a protocol around localised passive heating of the leg while seeking to maintain core temperature and hypothesised that an increase in muscle temperature would increase rapid force production and neuromuscular function. Fifteen healthy young adults (9 male) were either submersed to the waist in water for 90 mins to create a heated (water temperature = 42°C) or a control (water temperature = 36°C) condition. Superficial (+3.5°C) and deep muscle (+2.3°C) temperature was significantly increased in the heated condition with no change in the control limb. Knee extension torque was measured with an isokinetic dynamometer with knee angle fixed at 70°. Muscle function assessments were conducted through supramaximal femoral nerve stimulation resulting in knee extension, and peak torque and torque produced at 50 and 150 ms following stimulation were recorded. Force production was measured at baseline before whole body heating and immediately post heating. EMG measurements and muscle temperature were taken at the same time as rapid force production assessment. Rate of torque development increased from 1.4 to 1.9 MVC/s (+26%) at 50 ms and force produced via electronically evoked contractions increased after whole body heating compared to the control after the heating protocol (Figure 2. 12.) (1061 N.m/s, 1106 N.m/s, +4%).

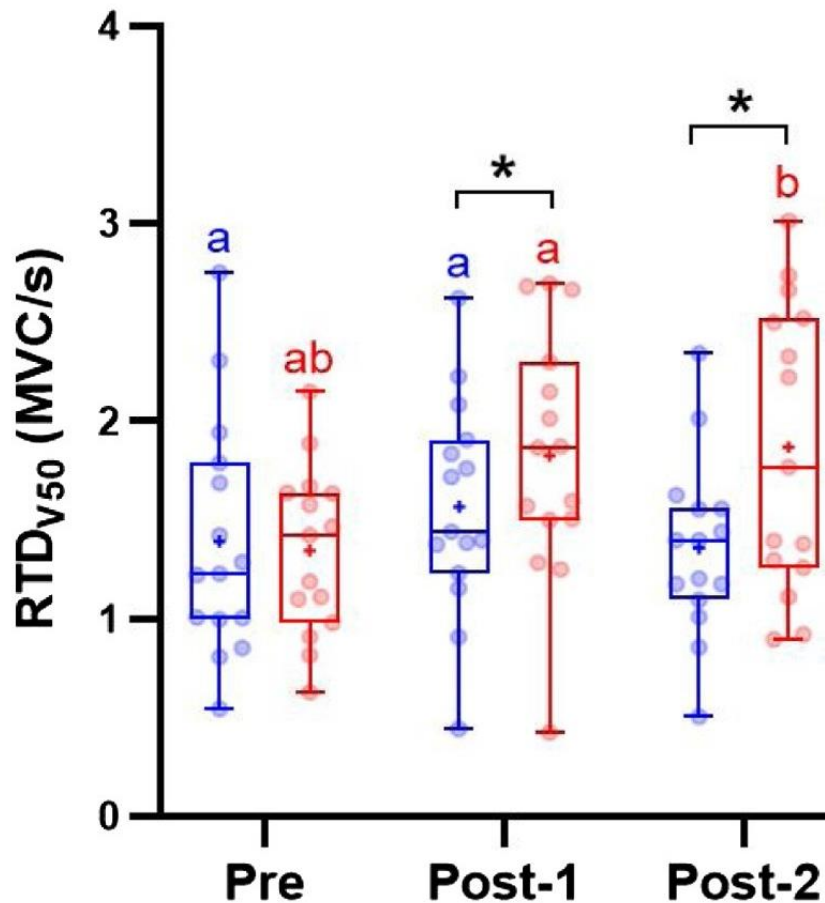


Figure 2. 14. Voluntary rate of torque development (RTD) at 50 ms (RTD_{v50}). Same letter means significant difference between time-points within water immersion treatments (42 °C or 36 °C). * denotes statistical difference between water immersion treatments (42 °C vs. 36 °C). $p < 0.05$. Adapted from Rodrigues et al. (2021)

Rodrigues et al. (2021) note that their findings of an increased core temperature of 0.9°C (37.34°C to 38.28°C) does not impair force production or muscle function is in contrast with prior research. They suggest this may be due to a lower core temperature increase i.e., $g < 1.0^{\circ}\text{C}$ compared to the +2.3°C increase (37.2°C to 39.5°C) reported by other e.g., Thomas et al. (2006). Rodrigues et al. (2021) suggest that neuromuscular enhancements are a result of peripheral heating, independent of central heating as their study identified that an increased rate of torque development was expected to be driven by neural contribution, however the EMG results did not show increased activity between conditions. This indicates that a change in the muscle's contractile properties was responsible for the increase in force production rather than a change in central factors such as an increased neural drive.

Increased muscle temperature can also impact the rate of force development in younger adults (Mornas et al., 2022), During the control condition, conducted in a thermoneutral environment

($25.8 \pm 1.8^\circ\text{C}$), core and muscle temperatures were recorded as being $37.0 \pm 0.3^\circ\text{C}$ and $34.0 \pm 1.1^\circ\text{C}$, respectively. In the hot condition, following immersion in hot water ($47.4 \pm 1.8^\circ\text{C}$), core and muscle temperatures increased to $38.4 \pm 0.3^\circ\text{C}$ and $37.0 \pm 0.8^\circ\text{C}$, respectively. Maximal voluntary force decreased by $5.0 \pm 11.3\%$ ($P = 0.052$) and voluntary activation decreased by $4.6 \pm 8.7\%$ ($P = 0.038$) in the HOT condition; early rate of force development (0–50 ms) also increased by $48.2 \pm 62.7\%$ ($P = 0.013$) compared to the CON condition. However, no significant differences were observed in fascicle dynamics or muscle-tendon unit properties between the two conditions. Therefore, whilst passive heating enhances the early rate of force development, it does so without altering muscle architecture or tendon stiffness, likely due to intrinsic contractile property enhancements resulting from increased muscle temperature.

Rodrigues et al. (2021) conclude that passive heating increases force production in the early phase of voluntary explosive contraction as well as decreases half relaxation time due to an increase in muscle tendon stiffness and Ca^{2+} kinetics. The study does not include a control for different age groups, training status or measurement of the decay of the heating effect, however, it is a key paper displaying the positive effects of passive heating, focusing on early-stage isometric muscle contraction. The difference in this study compared with others (Morrison et al., 2004; Thomas et al., 2006) examining temperature dependent changes in isometric contraction may be due to not increasing core temperature which impairs voluntary activation (Thomas et al., 2006).

2.2.9 Changes in isokinetic muscle function during whole body heating

Despite theoretical (Ranatunga, 1984; Ranatunga et al., 1987) and some experimental support (Rodrigues et al., 2021) for the use of heating to increase muscle function during isometric activity there are very few studies investigating the effect of passive heating of muscle tissue on isokinetic knee extensor function. Early isokinetic work was conducted by Stanley et al. (1994) where physically active young males performed three maximal knee extensor contractions at 30, 180, 300 and $400^\circ/\text{s}$ as well as a 5 sec isometric contraction after 45 mins of either hot water immersion (44°C water up to the gluteal fold), thermally neutral water immersion (35°C) or a control (23°C) ambient air temperature trial. This study did not find any significant differences between the conditions at any speed or with either contraction type. A non-significant increase in peak torque of 1, 4 and 3% at 180, 300 and $400^\circ/\text{s}$ was seen respectively. However, this study did not take any temperature measurements of the muscle or the skin and did not control or measure core temperature making it impossible to know if central hyperthermia was depressing force production or if muscle temperature was

adequately changed, as heating duration was only half that as contemporary work that found heating to be beneficial (Rodrigues et al., 2021). Additionally, the use of hot water immersion necessitates that the participant is not being heated during exercise and in the transitional period from heating to the dynamometer a loss of heat from the muscle was likely. Future research should investigate how muscle temperature and performance benefits are affected by a period of time between heating and muscle function assessment. It can be estimated that this heating protocol led to a muscle temperature increase of $\sim 2^{\circ}\text{C}$, which is lower than what was observed in Rodrigues et al. (2021). This does, however, raise an important future direction which is to examine changes in muscle function in response to incremental increases in muscle temperature.

Cheung & Sleivert. (2004) continued the research into whole body heating and skin temperature affecting knee extensor function as seen in Morrison et al. (2004) however, using dynamic contractions on an isokinetic dynamometer rather than isometric contractions. Twenty healthy young males began at a core temperature of 37.5°C and were heated by sitting in a pool of 42°C water up to the neck, with muscle function assessed every 0.5°C of core temperature increase until 39.5°C was attained. At each interval the participants completed two maximal contractions at 60, 120 and $240^{\circ}/\text{s}$ with 10 sec of recovery between reps and 1 min between sets. After a 39.5°C core temperature was reached participants were passively cooled back to 38°C with isokinetic measurements taken every 0.5°C decrease. Peak torque remained consistent across the core temperature range of $37.5\text{--}39.5^{\circ}\text{C}$. It was hypothesised that highest torque would be seen at highest core and skin temperature. Specifically, at a core temperature of 37.5°C , peak torque was 168 ± 34 N.m, 145 ± 29 N.m, and 112 ± 17 N m for 60, 120, and $240^{\circ}/\text{s}$, respectively. At a core temperature of 39.5°C , peak torque was 166 ± 38 N.m, 150 ± 31 N.m, and 119 ± 17 N.m for the same respective speeds. However, when skin temperature was reduced, peak torque decreased immediately by 5%, 11%, and 11% (159 ± 42 N.m, 133 ± 34 N.m, and 107 ± 22 N.m) at the respective contraction speeds. While core temperature increases did not significantly alter peak torque, reductions in skin temperature led to immediate decreases in peak torque, suggesting that skin temperature may play a crucial role in muscle performance. The authors suggest that skin temperature appears to regulate force production by affecting peripheral nerve conduction and muscle fibre contractility. Cooling the skin can slow neural signals, reduce motor unit activation, and impair muscle contraction speed, leading to decreased maximal force output. It is worth noting that this study had participants complete only two maximal contractions at each speed, which may not have provided enough of an opportunity to capture a true maximal effort; especially as the first repetition was from a static start. The paucity of research on isokinetic muscle function following passive heating necessitates further research into the topic; the next steps should

seek to combine localised passive heating, minimising core temperature increases, whilst additionally observing changes in early rate in force production as well as peak torque.

2.2.10 Decay of the heating effect

The length of time that the demonstrated positive effects on muscle contractile function following local hyperthermia persist after heating has ceased exercise begins has so far not been fully investigated. Preliminary work (Faulkner et al., 2013) has demonstrated the importance of this research. Eleven young healthy males completed a 15-min warm up and then during a 30-min rest period before a 30 sec sprint test participants either wore standard tracksuits, insulated tracksuits or heated tracksuits warming the lower limbs. Muscle temperature was increased via an active warm up to 38.1°C (+2.8°C from baseline), in the control condition of standard tracksuits muscle temperature reduced to 36.9°C, muscle temperature remained at 37.1°C (+1.7°C from baseline) in the insulated condition and 37.5°C (+2.1°C from baseline) in the heated condition after a 30 min rest.

Peak power output was 9.6% greater in the heated condition where heat was preserved from the warm up, in comparison to the regular tracksuits which did not maintain muscle temperature (1609 ± 270 W, 1468 ± 260 W), mean power output, time to peak force and peak cadence did not change; these factors likely did not change as there is no evidence that heating increases either total exercise capacity or cadence. The authors claim a relationship between muscle temperature and cycling sprint performance of 4-10% per degree increase of muscle temperature based off their results in conjunction with Sargeant. (1987). The increase in power is attributed to a higher ATP turnover rate (see 2.1.11) in warmer muscle facilitating the increase in peak power output. This study demonstrates that after increasing muscle temperature if that temperature is not maintained and is allowed to cool, performance gains are also lost.

A similar study investigating the importance of heat maintenance after a warmup within a simulated rugby game was demonstrated by Fairbank et al. (2021) whereby thirteen young males completed a warmup before a 15 min rest period where they either wore their usual kit or a "Passive Heat Maintenance Garment". After the warmup tympanic temperature was raised to $36.0 \pm 1.1^\circ\text{C}$, and the garment attenuated the decline in tympanic temperature to $35.3 \pm 1.9^\circ\text{C}$ compared to the control at $34.6 \pm 1.2^\circ\text{C}$. The study reported a 4.7% increase in high intensity distance covered by the group in the garment condition (648.4 ± 88.6 m, 618.1 ± 72.8 m), all other distances and RPE showed no difference. While this study seems to contradict prior examples of increased core temperature reducing performance however, the core temperature increased displayed here are modest and do not approach the 39°C seen

in Thomas et al. (2006), and the garment likely maintained muscle temperature. This study does not expand into the mechanics behind performance improvement however speculates that maintaining muscle temperature following a warmup should be a consideration for rugby players. While this study did not measure muscle temperature directly and did not have clearly defined metrics of performance it still adds credence to the effect that increasing muscle temperature provides performance benefits and after muscle heat has been achieved without intervention the heat will dissipate and the performance benefits will be lost without the preservation of intramuscular heat, however more research is needed in this area before conclusions can be definitively drawn. The current body of research also completely neglects how heating may dissipate differently in older individuals who often have different muscle composition, including more intramuscular fat which may influence the thermodynamics of the thigh. The decay of the heating effect has been previously measured to be an 8.8% decrease in cycle ergometer peak power output after 30 mins after heating (Faulkner et al., 2013) however, there are currently no studies investigating how passive heating affects low repetition muscular strength, as may be seen in traditional strength-based resistance training.

2.2.11 Advancing knowledge and application of passive heating to improve muscle function

Current research contains a plethora of heating methodologies (see table 2.2) however to this author's knowledge there has yet to be a study examining the intra-day reliability of passive heating protocol's ability to augment physiological responses and muscle function. Without this understanding, current and future research into the area of passive heating are working with estimations of reliability. While previous work often assesses a baseline and then post heating intervention (Mornas et al., 2022; Morrison et al., 2004; Rodrigues et al., 2021; Thornley et al., 2003) the relationship between incremental magnitudes of heating and the response of the muscle has been overlooked in isokinetic and isotonic contractions. While it may be assumed that there is a dose response relationship between localised hyperthermia and the physiological responses this has yet to be demonstrated. Without test-retest reliability within and between sessions type 1 error becomes more likely, and a calculation of the minimal detectable change (MDC) is not possible; minimal detectable change is the change needed between two scores to be sure that the change reflects a true improvement or decline, rather than random noise or inconsistency in the measurement process. For research purposes the MDC provides a threshold above which you can be confident the change reflects a genuine improvement or decline especially in individual participants, this allows for a comparison between responders and non-responders and is essential especially within the early stages of this research.

Future research that explores the dose response (to establish a minimal effective dose or observe if more heating returns more benefits) of heating in conjunction with observing contractions throughout a variety of contractile speeds and types will aid in understanding the underlying balance of the physical and chemical mechanisms that drive the performance increases previously seen through highlighting where performance changes occur. Evidence supporting a chemical/calcium handling mechanism being more prevalent than a mechanical mechanism after passive heating can be seen in cycling studies, where higher speeds (140 RPM) were seen to benefit 10% while slower cycling speeds (54 RPM) only benefited 2% (Sargeant, 1987). While current research appears to suggest that a chemical/calcium handling mechanism is dominant in providing passive heating benefits it is important to note that strength-based tasks such as isokinetic work done at low velocities have currently only been conducted after whole body heating (Cheung & Sleivert, 2004). Whole body heating has been identified as detrimental to maximal voluntary muscle activation (Thomas et al., 2006) and therefore must be considered when reviewing prior work in the field of muscle force production following passive heating. Currently there is limited work documenting the rate at which the physiological responses to local hyperthermia decay and how this affects muscle function once heating has ceased, and preserving intramuscular heat has been identified as an important factor following a passive or active warm up, as rugby players who maintained muscle temperature using a thermal garment had a 5% increase in high intensity distance travelled in a simulated game (Fairbank et al., 2021). Similarly, Faulkner et al. (2013) describes the need to maintain muscle temperature following a warmup to preserve the performance benefits that were achieved through the prior heating. These studies highlight the phenomenon of heat decay and its unmeasured effect on performance and understanding the timeframe in which performance benefits remain post heating will be essential for the implementation of passive heating as a tool.

Given the combined effects of aging on skeletal muscle composition and neuromuscular function which result in reduced muscle function and subsequently lead to reduced capacity for physical activity and daily living tasks, an investigation into the possible positive impact of passive heating on dynamic muscle performance in older adults is warranted. By examining isokinetic and isotonic contractions across multiple velocities, this thesis aims to provide a detailed assessment of muscle function that reflects both strength and functional performance.

Current research has predominantly focused on a young and healthy population however, it is understood that older individuals have a different heating response, typically having a delayed response to heating, with higher levels of intramuscular fat insulating the muscle and with reduced sweat rates and diminished cardiovascular capacity restricting the older

individual's ability to dissipate heat (Kenny et al., 2010). Whilst this is known, exposure to a potent thermal stimulus is likely to produce expected increases in muscle temperature. Passive heating has demonstrated positive changes in muscular function in older adults, and older peripheral arterial disease patients have demonstrated positive acute responses to heating, such as a 10% increase in 6-min walk distance was seen after 15 mins of lower limb heating (Pellinger et al., 2019), potentially demonstrating that older adult exercise capacity is enhanced by passive heating. Older adults suffering from sarcopenia causing diminished muscle function which negatively impacts their ability to partake in physical activity or daily living tasks may be the greatest beneficiaries of a tool that is able to increase muscle function. Those who live just under the threshold of ability to conduct independent physical activity may feel more capable of partaking in strengthening exercise with an effective preparatory tool.

This thesis will therefore investigate whether passive heating confers benefits to maximal dynamic muscle function in both younger and older adults. While potential improvements in dynamic muscle function have been reported in younger populations, it is important to extend this research to older adults, who may respond differently to heating and therefore require alternative or population-specific heating strategies.

To build upon the prior research dynamic contractions need to be studied, and isokinetic and isotonic testing across a variety of speeds is essential to comprehensively assess muscle function, as different contraction velocities may reveal distinct aspects of strength, power, and endurance relevant to daily activities and athletic performance. Testing at multiple speeds allows for a more detailed understanding of underlying mechanisms. Additionally, establishing the reliability of these tests both between days and within sessions is crucial to ensure that measurements are reproducible and knowledge of natural variance is considered when analysing results.

2.3 Aim of Thesis

This thesis aims to investigate the effects of muscle temperature on isokinetic and isotonic muscle function, examining various contraction speeds and types before and after heating. The research aims to identify a successful heating protocol and quantify intra- and interday reliability of isokinetic and isotonic knee extensor exercises following passive thigh heating. Additionally, this thesis will aim to establish validity of the changes in muscle force production and demonstrate how these changes are meaningful. Finally, this thesis, will explore the effects of muscle temperature on isokinetic and isotonic muscle function in healthy younger and older adults how aging influences muscle function following passive heating.

2.4 Research questions explored within this thesis

This thesis reviews evidence showing how muscle structure and function decline with age and explores the growing interest in passive heating as a method to enhance maximal force during low-repetition exercise. While passive heating has been shown to improve muscle contractile function through chemical, mechanical, and neural pathways, most research focuses on static or prolonged exercise. There is a lack of detailed investigation into how passive heating affects dynamic muscle function, particularly at different levels of heat stress, contraction speeds, and during the early phase of force generation. The duration of these benefits after heating ends remains unclear, especially for peak and early dynamic force. Additionally, little is known about the reliability of performance changes following heating, and most studies involve only young, healthy individuals, overlooking older adults who may benefit most. To address these gaps, this thesis will assess the effects and lasting impact of passive heating on dynamic muscle function in both young and older adults, aiming to evaluate its potential as a practical performance-enhancing strategy.

2.5 Proposed Research studies and Hypotheses

The following specific research questions and associated hypotheses are proposed for this thesis

2.5.1 Inter and intraday reliability of muscle force production and systemic physiological responses to localised passive heating

To observe and quantify both between-day and within-session reliability of an isokinetic and isotonic knee extension protocol, in which one limb is subjected to passive heating while the contralateral limb remains unheated.

- It is hypothesised that the protocol will be highly reliable both between days and within the same session, as measured by high ICC and low coefficient of variation.

2.5.2 Muscle force responses to localised passive heating

Determine the effect of prolonged localised passive heating on knee extensor peak and early force production in young, healthy adults during slow, medium and fast isokinetic contractions and during maximal isotonic contractions. Muscle function was measured at 30 min intervals to monitor potential changes over time and to determine whether these changes were temperature dependant. At each measurement point systemic physiological measures were taken before isokinetic and isotonic muscle function assessments were conducted.

- It is hypothesised that localised passive heating will enhance peak and early force production across all contractile velocities from 30 mins post-intervention onward, with incremental improvements corresponding to increases in muscle temperature

2.5.3 Differences in muscle force responses to localised passive heating in younger and older adults

To determine whether localised passive heating elicits differences in peak or early-phase force production during slow, moderate, or fast isokinetic contractions, as well as maximal isotonic velocity efforts, between younger and older adults.

- It is hypothesised that both younger and older adults will exhibit comparable improvements in dynamic muscle function following localised passive heating.

2.5.4 Changes in muscular fatigue resistance in older adults following localised passive heating.

To investigate whether localised passive heating can enhance fatigue resistance in aged muscle during a 30-repetition isokinetic fatigue task, by comparing the performance of a heated limb to that of an unheated limb.

- It is hypothesised that localised passive heating will attenuate the decline in peak torque of the knee extensors throughout the fatigue task.

Chapter 3 General methods

3.1 Health and safety

All research was conducted within the HNZW 038 Biodex Laboratory located within the Heinz Wolff building at Brunel University of London.

All experimentation and handling of biological materials and waste, such as used needles and contaminated dressings, were conducted and disposed of in accordance with Brunel University of London's Standard Operating Procedures for research within the Department of Sport, Health and Exercise Sciences. During each data collection session at least two experimenters were present throughout, at least one of whom was qualified at first aid. Contact information for additional first aiders or security was always kept within the Biodex laboratory. Risk assessments were also completed for use of the laboratory, exercise, and invasive techniques including but not limited to isokinetic dynamometry, passive heating and intramuscular temperature measurement. All apparatus was cleaned before and after use.

Electrical equipment contacting the body such as heart rate monitors, skin temperature thermistors, EMG probes and saturation probes were cleaned using warm water and soap, followed by an alcohol cleaning wipe after testing had concluded.

3.2 Ethical approval

Brunel Research Ethics Online (BREO) approved the protocols and procedures of all experimental studies presented in this thesis. Ethical approval codes are provided in the 'ethical approval' sections of each experimental chapter. Letters of ethical approval from BREO are presented in Appendix B and C.

3.2 Participants

Healthy adults (male and female) aged between 18 and 30 years were recruited through convenience sampling for the first experimental study. The age range was capped at 30 years for the younger group and the older group recruited those over 55 years old and who averaged an age of 68 years old. This age group separation was to avoid confounding the physiology of younger and older adult groups that were used in this thesis. Participants were deemed healthy to take part prior to participation following completion and passing of a health questionnaire. Individuals that reported adverse health issues or were taking blood thinning medication were excluded from study participation. Smokers were not permitted to take part in the experiments due to the impact of smoking potentially impacting oxygen transport

through increased levels of carbon monoxide and decreased lung function which could increase fatigability through a reduction in available oxygen to the muscles (Wiener et al., 2020). The stimulating effect of nicotine would also be a confounding factor in measuring muscle function (Wiener et al., 2020). Individuals with illness, disease or a history of heat intolerance or any other chronic illness or injury that may have impacted participation, were also excluded. A detailed description of the participants recruited to each study is provided in the method section of relevant experimental chapters.

3.2.1 Participant recruitment

Participants were recruited via posters placed around Brunel University London and the surrounding areas. Announcements to students were made in lectures advertising the opportunity to participate.

3.2.2 Pre-participation

Before agreeing to participate, volunteers were provided with a detailed participant information sheet, which described, the requirements of the study, precise experimental procedures and the benefits and risks associated with participation. Willing volunteers were encouraged to ask any questions regarding the studies before deciding whether to take part and were informed that they had the right to withdraw from the study at any point for any reason without the need to provide a justification or explanation and that doing so would not result in any penalty. All participants signed, written informed consent prior to taking part.

3.3 Participant characteristics

3.3.1 Anthropometry: stature

Participants' standing stature (cm) was recorded at the first experimental visit of each study using a stadiometer (SECA model 213, Hamburg, Germany), with shoes and socks removed. Participants were required to stand with their heels, buttocks and upper back aligned vertically, facing away from the stadiometer their head tilted to align the superior helix of the ear with the eyes. The stadiometer measurement arm was lowered until it rested upon the top of the head, participants were instructed to take one deep inspiration, followed by a relaxed expiration to ensure correct posture. Stature was recorded to the nearest 0.1 cm.

3.3.2 Anthropometry: body mass

Participants body mass was measured (to the nearest 0.1 kg) using electronic scales (SECA model 875, Hamburg, Germany) at each experimental visit (specific time points of measurement are detailed in the relevant experimental chapters). Participants were required to be dry and stepped onto the zeroed scales until the digital display was stable.

3.3.3 Body composition

Body composition was assessed using the Durnin & Womersley four-site skinfold method (Womersley & Durnin, 1977), the four-site measurement and formula have been shown to be accurate (Sardinha et al., 1998) and reliable (Durnin & Womersley, 1974). Measurements were recorded to the nearest millimetre after two sec of calliper contact with the skinfold and taken in a circuit allowing fat tissue to redisperse between measurements. Skinfold calliper measurements were taken from the triceps (located halfway between the olecranon process and the acromion process when the hand is supinated), biceps (on the same horizontal level as the triceps), subscapular (1 cm below the inferior angle of the scapular following the natural cleavage lines of the individual) and iliac crest (diagonal fold taken immediately above the ilium in the mid auxiliary line). These skinfolds were then used to calculate the participants body density.

Equation 3. 1. Calculation of body density for adult males (Durnin and Womersley 1974)

$$\text{Body density} = 1.1610 - 0.0632 \text{ Log } \sum \text{Iliac Crest, Subscapular, Triceps, Biceps}$$

Equation 3. 2. Calculation of body density for adult females (Durnin and Womersley 1974)

$$\text{Body density} = 1.1599 - 0.0717 \text{ Log } \sum \text{Iliac Crest, Subscapular, Triceps, Biceps}$$

Equation 3. 3. Calculation of percentage body fat for human populations (Siri 1956)

$$\% \text{ Body Fat} = (4.95 - 4.5) \times 100$$

Given the experimental interest in the lower limbs, additional skinfold assessments were made for this region. The British Olympic Association recommends that anterior thigh (vertical skinfold midway between the inguinal crease and the proximal border of the patella) and proximal calf (vertical skinfold 5cm inferior to the fossa popliteal) measurements should be taken in conjunction with Durnin & Womersley 4 site skinfold method (Norton & Eston, 2019).

3.4 Control measures and standardisation

Participants were required to avoid stimulants that would improve isokinetic strength including caffeine 12 hours prior (Grgic et al., 2022). to avoid alcohol 24 hours prior (Barnes, 2014) and to avoid heavy exercise 48 hours prior to testing (Michaut et al., 2003) as it might influence muscular force production. Controlling for these factors during each experimental trial

mitigated potential confounding impacts upon muscle function. Participants were asked to replicate a similar eating pattern for both experimental trials. Participants were required to arrive to the laboratory in a euhydrated state on each visit, by maintaining normal drinking and consuming an additional 10 mL of water per kilogram of bodyweight the night before and the morning of each experimental trial. The control limb for this thesis was also covered with insulating leggings but was not wrapped in an un-perfused garment, the wearing of an unheated garment was trialled to maintain biomechanical leverages, however, pilot testing revealed no difference between the conditions, and therefore the decision to have the leg be uncovered to remain as thermoneutral as possible was decided upon.

3.4.1 Contralateral heating design

Throughout this thesis, a contralateral heating design is utilised, whereby one limb is heated while the opposite limb serves as a thermoneutral control within the same experimental session, rather than conducting separate trials in which both limbs are heated or both remain thermoneutral. This approach was adopted to minimise experimental variability. As demonstrated in CHAPTER 4 - **Experimental study 1 – Passive thigh heating improves isokinetic but not isotonic muscle function**, muscle force production in a single thermoneutral limb exhibits greater variability between days than within a single testing session. By using a within-session contralateral control, excellent reliability was observed both between sessions and between days, while overall variability was kept as low as possible. The residual between day variability is likely attributable to changes in participant motivation or normal physiological fluctuations rather than measurement error.

The methodological rationale for this design is strongly supported by the unilateral exercise literature. Macinnis et al. (2017) provides a comprehensive evaluation of unilateral and contralateral experimental models and highlights their particular strength in human physiology research. A key advantage identified is that unilateral designs allow each participant to act as their own control, thereby controlling for systemic influences such as circulating hormones, neural drive, cardiovascular responses, environmental conditions, nutritional status, and motivation levels. This substantially reduces inter-individual variability, increases statistical power, and allows meaningful physiological effects to be detected with smaller sample sizes, advantages that are especially important when studying subtle changes in muscle function. The authors further emphasise that unilateral models are particularly well suited to investigations of local interventions, such as temperature manipulation, where the primary effects are expected to be peripheral rather than systemic. By comparing limbs within the same individual, contralateral designs improve sensitivity to

local muscular adaptations while maintaining identical central and systemic conditions. This aligns directly with the aims of the present thesis, where isolating the local effects of muscle heating is critical.

Whilst the potential for interlimb interactions via neural or circulatory pathways are possible, it is worth noting that meaningful cross-education effects are most commonly associated with repeated unilateral resistance training over several weeks, rather than acute or short-term experimental interventions. This interpretation is consistent with existing evidence showing that cross-education typically requires sustained training stimuli (Green & Gabriel, 2018). In addition, prior work using contralateral heating protocols has demonstrated no evidence of molecular or haemodynamic carry-over effects to the non-heated limb, further supporting the assumption of limb independence in acute experimental designs. Collectively, the strong reliability outcomes observed in Chapter 4, combined with the methodological advantages outlined in the unilateral exercise literature, support the use of a contralateral heating design as a robust and internally controlled approach. This design maximises sensitivity to local heating effects while minimising variability, making it well suited for investigating changes in muscle force production and physiological responses in human skeletal muscle.

3.5 Heating via water perfused trouser

Limb temperatures were manipulated by applying a custom hot water-perfused garment, consisting of a series of water tubing allowed a constant flow of hot water to pass over the participant's thigh. One limb was heated while the other served as an unheated control. To minimise the risk of burns or pinching the participants wore leggings to act as a barrier between the garment and the skin. The trouser was connected to a thermostatically controlled water circulator (Julabo F-34), which continuously circulated 50°C water for 90 min. The participants sat on the ovoid shaped heated trouser as to place it underneath the hamstring muscle group but avoid the gluteal group. Either side of the trouser was wrapped around the anterior thigh covering the *vastus lateralis* and *rectus femoris*, while not overlapping; the trouser was secured with a Velcro strap (see Figure 3. 1.). To access the thigh for muscle temperature measurements the trouser was pulled distally, exposing the site for the duration of the measurements (~60 s) whereupon completion the trouser was returned to cover the entire thigh. Prior work investigating muscle temperature changes using this specific heating methodology has reported consistent heating rates of muscle temperature change (Figure 2. 12. Local temperature change from baseline of the *vastus lateralis* in response to single leg, double leg and whole body passive heating over time. Figure displays data from prior work,

red circles whole body heating (Watanabe et al., 2024), orange circles single leg heating (Watanabe et al., 2024), blue circle thermoneutral control (Watanabe et al., 2024), black diamond single leg heating (Gibson et al., 2023). and Figure 2. 13. Local temperature change in $^{\circ}\text{C min}^{-1}$ from baseline of the *vastus lateralis* in response to single leg, double leg and whole body passive heating over time. .), supporting the reliability of the heat stimulus applied. The *vastus lateralis* is commonly selected as a measurement site in comparable studies (Gibson et al., 2023; Kenny et al., 2003; Rodrigues et al., 2024), due to its superficial location and accessibility for temperature measurement. However, it should be acknowledged that the *vastus lateralis* is not the primary contributor to knee extension torque and therefore may not fully represent the thermic conditions experienced by other quadriceps muscles, which may influence the generalisation of these findings across the muscle group.



Figure 3. 1. Application of heated water perfused garment prior to isokinetic muscle function testing. Garment is positioned over the participants thigh on top of nylon leggings

3.6 Isokinetic dynamometry

Maximal leg extension tests were performed on an a Biodex 3 Multi-Joint System Pro Isokinetic Dynamometer (Biodex Medical Systems, Shirley, NY, USA). Figure 3. 2. shows the optimal position for a knee flexion/extension test (Biodex, 2011), with the participant's torso being secured to the machine with a padded strap. The headrest was adjusted to sit comfortably behind the participants skull. The machine was adjusted to ensure the shank, and the torso were parallel to each other, and the thigh was perpendicular to both, creating a 90° angle at the hips and the knee. The knee joint flexion and extension axis was defined as a mediolateral axis passing through the lateral femoral epicondyle, representing the approximate centre of rotation within the sagittal plane. The dynamometer height was altered

such that the knee axis for the flexion-extension movement was aligned with the mechanical axis of the dynamometer. Additional straps were placed across the right thigh and the waist. The distal shin pad of the dynamometer was attached 2 to 3 cm proximal to the lateral malleolus by using a strap. The machine then had the range of motion safety limits set for full flexion and extension, ensuring that range of motion exceeded 100°. A calibration of vertical shaft position was then conducted with assistance of a spirit level, and the leg was weighed for gravity correction in accordance with the BASES statement (Baltzopoulos et al., 2012). For experiments involving multiple visits, the dynamometer set up was replicated for the second trial.



Figure 3. 2. Optimal Position for a Knee Flexion/Extension Test. (Biodex, 2011)

3.6.1 Warm up

A warmup of 10 submaximal knee extensions (five at 50% maximum effort, three at 75% maximum effort, two at 90 of maximum effort) was conducted on each leg at a self-selected intensity. The Biodex system 4 software was used throughout this thesis collecting data at 100Hz (Biodex Medical Systems, Shirley, NY, USA). Torque, position, and velocity data was

therefore collected within software every 10 millisecond then exported as a .txt file and imported to Microsoft Excel for analysis. Active warm ups, including cycling on an ergometer, dynamic stretching and 10 repetitions at various isokinetic contractile speeds have not demonstrated an ergogenic benefit to peak force production (Altamirano et al., 2012; Júnior, 2013.; Park et al., 2018). This warm up was conducted in the interest of increased injury prevention.

3.6.2 Isokinetic testing

Isokinetic dynamometers measure isokinetic muscle strength by recording the resistive moment required to counterbalance the force applied by the participant and maintain a constant joint angular velocity. Isokinetic force is considered the gold standard of muscle function testing as it can observe and record a complete profile of force production throughout a contraction providing insights into mechanistic changes within the muscle during research. Isokinetic muscle function of the knee extensors was assessed during slow (60°/s), medium (180°/s) and fast (300°/s) contractile speeds to assess potential mechanisms affecting muscle function after local hyperthermia, using these speeds to assess muscle function is in accordance with prior research (Menzel et al., 2013). Testing across multiple contractile speeds was used in this thesis to provide insights into how passive heating may benefit performance across a range of functional tasks. Slow contractions primarily assess maximal strength such as rising from a chair or lifting a heavy object (Reid & Fielding, 2012). Moderate contractions represent the demands of walking or stair climbing (Petrella et al., 2005), whilst fast contractions provide an insight into the function of the muscle to quickly activate and provide force as may be necessary in correcting balance or reversing a fall (Izquierdo et al., 1999). Participants were instructed to begin the test with their leg fully flexed before completing three continuous repetitions of maximal knee extensions, 60 sec of rest was provided between each set with 3 mins of rest between limbs. The heated leg was tested first to ensure accurate heat exposure duration; this was followed by the control (~5 min delay). Measurements were made at baseline, and 30, 60, 90 and 120 mins following heating onset. The Biodex system 3 has been demonstrated to be highly reliable for isometric (Drouin et al., 2004), isokinetic and isotonic measurements (Sáenz et al., 2010); however, the reliability of these measures whilst undergoing passive heating is not known.

3.6.1 Isometric testing

A 5 sec maximal isometric contraction was conducted on both the heated and normothermic limbs at 90° of knee extension, with the arm of the dynamometer perpendicular to the ground as checked by a spirit level, at the start of each testing session following isokinetic measures. The purpose of this was to provide a characterisation of the participants strength profile and it

was also used in the calculation of isotonic loads. Participants were instructed to “push as hard as they can” and were given verbal encouragement throughout. Maximal torque values were used as a measure of maximal muscle force production, torque is a rotational force calculated by multiplying the force generated by the muscle and the moment arm, the lever through which the force is applied. Correct dynamometer setup minimised the moment arm by aligning the dynamometer shaft with the medial condyle of the knee allowing for an accurate assessment of force produced by the muscle. A 3-min rest was provided between limbs to allow for a full recovery. The Biodex system 3 has been demonstrated to be valid and reliable for isometric assessment, displaying a 1% variance during isometric torque measurements (Drouin et al., 2004).

3.6.3 Isotonic testing

Isotonic testing records peak limb velocity produced by the muscle under assessment. The dynamometer measures velocity by recording the rate of change in the angle of the dynamometer shaft through its range of motion. Isotonic testing to assess maximal contraction velocity occurred at 25% of the participants maximal voluntary isometric contraction, in line with similar muscle function research (Cheng & Rice, 2005; Petrella et al., 2005). Isotonic velocity and power development have been demonstrated to be highly reliable (ICC= .85 - .98) in loads under 75% of maximal MVC (Van Driessche et al., 2018).

3.7 Electromyography data collection and analysis

3.7.1 Sensor placement

Surface EMG activation of the *vastus lateralis* (VL) was assessed with a Delsys Trigno Wireless System electrode (Delsys Trigno System, Boston, MA, USA). A small mark was made on the skin at 2/3 on the line from the anterior spina iliaca superior to the lateral side of the patella, this mark indicated the location of the sensor (in accordance with the Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles guidelines (Hermens et al., 2000)) and allowed a visual inspection to determine if the sensor had moved when in contact with the heated garment. Sensors were placed parallel to the muscle fibres (see figure 3. 5. for sensor placement) and sampled at 1000 Hz (Solnik et al., 2010). The data were amplified by a factor of 909 and filtered on-board with a 10–500 Hz bandpass filter. Prior to the placement of electrodes, the skin was prepared by removing any excess body hair over the desired region and cleaning the skin using an alcohol wipe The Delsys EMG Trigno system has been demonstrated to be comparable to previously used wet gel electrodes and dry metal electrodes, as well as being valid and reliable (Yamagami et al., 2018)

3.7.2 EMG analysis

Delsys EMG acquisition software (Delsys Trigno System, Boston, MA, USA) was used to collect the raw EMG data. The data were plotted within the acquisition software before being exported to Excel. Once exported the data were processed with custom Matlab code (see appendix A), the raw EMG signals were bandpass filtered with a 6-450Hz cut-off frequency in accordance with similar studies (Gordon et al., 2023) before subtracting the mean of the signal to correct baseline-offsets. The filtered signal then underwent full-wave rectification and low-pass filtering to produce a linear envelope using a dual-pass 2nd order Butterworth filter. EMG signals often contain noise and artifacts that can obscure meaningful data. Applying a second-order Butterworth filter helps in smoothing the EMG signal by effectively reducing high-frequency noise without distorting the signal's essential components. This filtering process enhances the signal-to-noise ratio, facilitating more accurate analysis and interpretation of muscle activity and has been used in similar isokinetic tasks (Engardt et al., 1995; Taha et al., 2015).

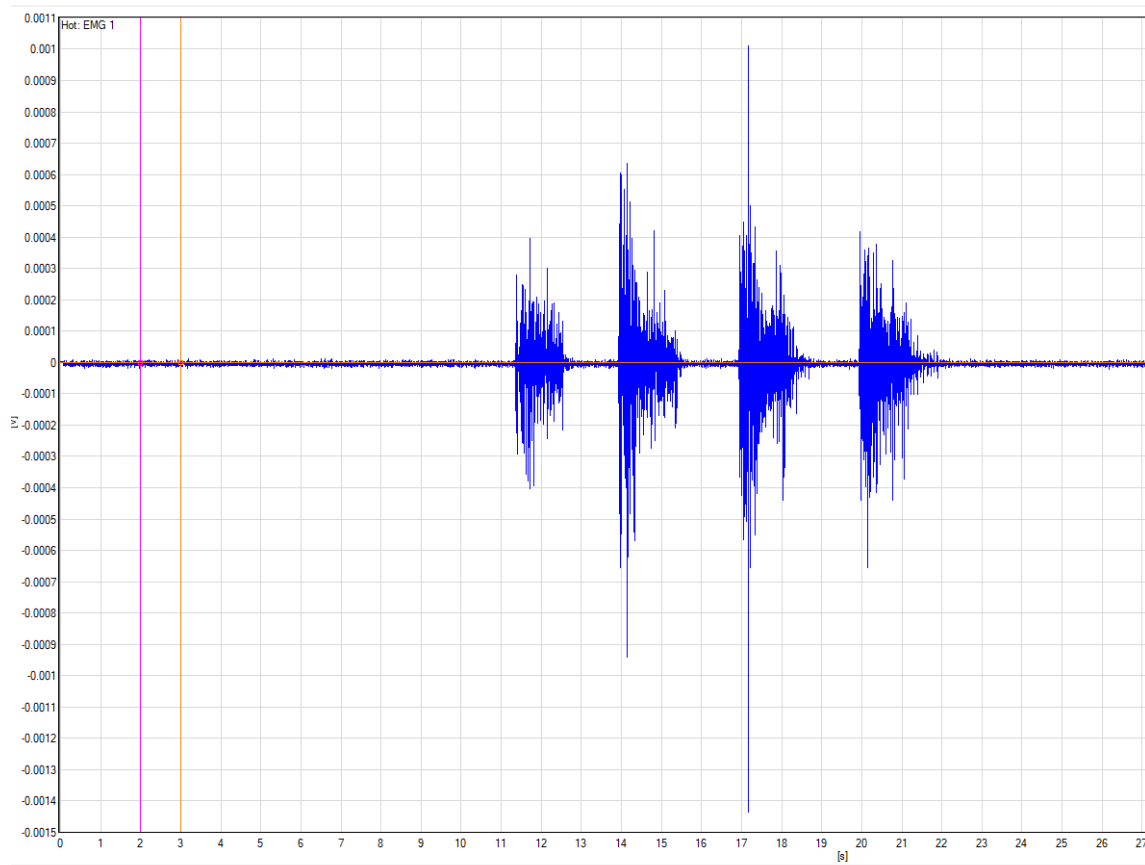


Figure 3. 3. Example of raw EMG data from the *vastus lateralis* during 60°/s knee extension

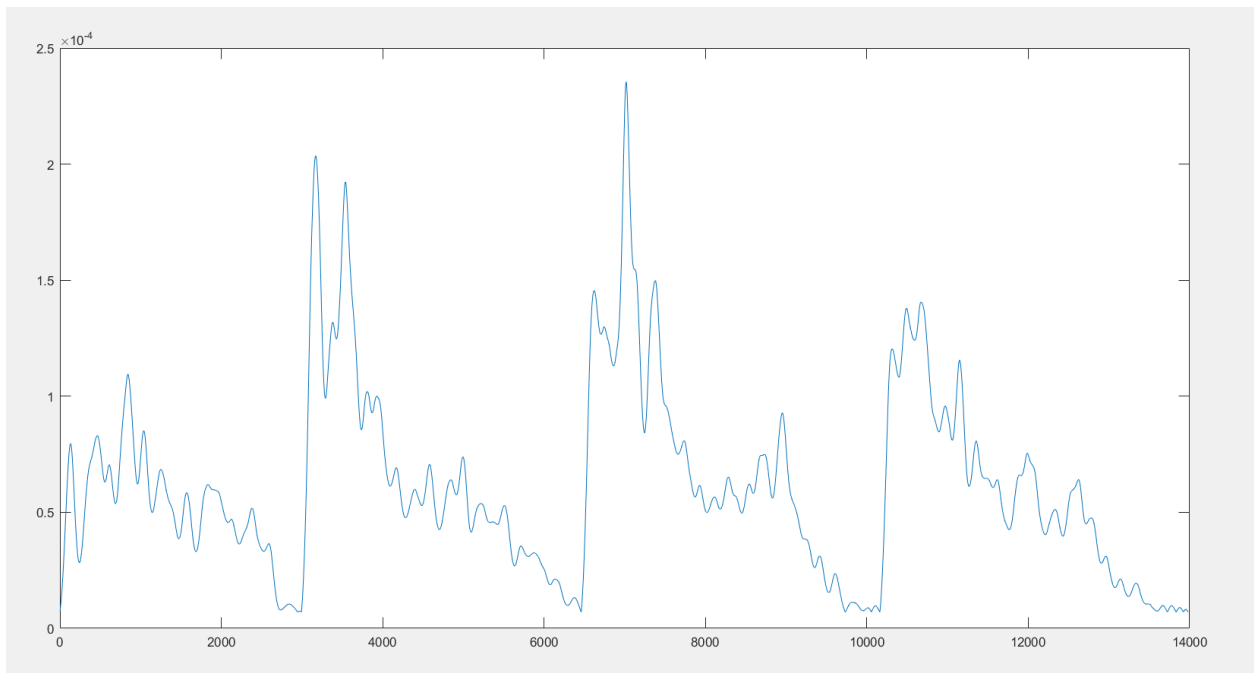


Figure 3. 4. full-wave rectification and low-pass filtering using a dual-pass 2nd order Butterworth filter of raw EMG signal from Figure 3. 3.

3.8 Physiological measurements

All physiological measurements were manually recorded and transferred to a spreadsheet for analysis.

3.8.1 Tympanic temperature

Tympanic temperature was used as a core temperature surrogate measure. An in-ear temperature probe (Brawn Thermoscan 7, Bussigny, Switzerland) was set to the appropriate age setting and then fully inserted into the right ear canal. Temperature measurement was taken every 30 mins after baseline. The Brawn Thermoscan focuses the infrared electromagnetic radiation emitted from the tympanic membrane into a thermopile detector, which in turn transfers the radiation into heat and then to electricity which is measured. The device and methodology were validated in a prior study finding only 1% of error within the general population, the tympanic measurement was found to be more reliable and accurate than commonly used oral and other auxiliary site measurements for approximating core temperature (Chamberlain et al., 1995).

3.8.1.1 Validity of tympanic temperature as a surrogate for direct measures of core temperature

Oesophageal and rectal thermometry are commonly used to assess core temperature in clinical and exercise settings due to their accuracy and responsiveness (Ganio et al., 2011; Moran & Mendal, 2002), but their invasive nature means that they can negatively impact recruitment for passive heating protocols where large thermal shifts are not anticipated (Welles et al., 2018). Tympanic thermometry, while less accurate under high-intensity or extreme environmental conditions (Easton et al., 2007), has been shown to correlate well with core temperature at rest (Ganio et al., 2009). We believe it to be a suitable alternative to direct measures of core temperature for passive, resting interventions where null or negligible changes in core temperature are probable. As such, this study adopted tympanic temperature monitoring, allowing sufficient duration for any potential core temperature changes to be reliably detected.

3.8.2 Skin temperature

iButton wireless devices (DS1922L Thermochron Data Logger, UK) were used to measure skin temperature. The skin sites were prepped by wiping clean with an alcohol wipe and allowed to dry before the iButton were affixed using Tegaderm film (3M healthcare, Minnesota, United States of America), at four sites; one on each medial calf and one placed distally to the EMG sensor on the vastus lateralis on both limbs. The iButtons were programmed to record temperature every min. Information was transferred between the iButton device and a PC with a momentary contact within a Blue Dot receptor (Thermochron Data Logger, UK). Data was extracted from the iButton devices after the testing had concluded, the data was plotted with the Onewire viewer software (Thermochron Data Logger, UK) then exported as an Excel file.

3.8.3 Muscle temperature

Prior to measurement the skin above the *Vastus Lateralis* was cleaned with an alcohol swab and allowed to air dry. Muscle temperature was recorded using a temperature probe (RS 103-433 K-type thermocouple, England) inserted ~30 mm deep at a 45° to the horizontal into the anatomical site marked superficially above the Vastus Lateralis via a 21-gauge (1.2 mm) hypodermic needle (Microlance 3, Ireland). This depth was selected in line with previous work (Gibson et al., 2014) and more recent investigations into muscle temperature kinetics during passive heating which observed that typical skinfold thickness over the vastus lateralis is 10 mm, and the muscle thickness is 25 mm (Rodrigues, Trajano, et al., 2020). A 30 mm depth therefore represents a mid-depth measurement and minimises the influence of deep and superficial measurements of muscle temperature which initially present as higher and lower respectively; up to 1°C between 10 mm increments, (Rodrigues et al., 2024), before inverting

this relationship between 15 and 60 min of heating. For heating ≥ 75 min, measurement depth does not appear to influence muscle temperature (Raccuglia et al., 2015). Temperature was allowed to stabilise (~5 sec) before values were manually recorded after stabilisation. See figure 3. 5. for a depiction of muscle temperature measurements.



Figure 3. 5. An example of muscle temperature measurement of the *vastus lateralis* in between muscle function assessments on a participant. This figure also demonstrates the placement of surface EMG sensors on the *vastus lateralis*. Skin temperature logger is distal to the surface EMG sensor visible.

3.8.4 Heart rate and blood pressure

Heart rate and blood pressure was taken with a self-inflating cuff placed over the left brachial artery (Carescape V100 VitalSigns Monitor, Bolton). An automated heart rate sensor simultaneously recorded real time heart rate, systolic and diastolic measurements were recorded manually and mean arterial pressure was subsequently mathematically estimated based off (DeMers & Wachs, 2022) .

Equation 3. 4. Calculation of Mean Arterial Blood Pressure adapted from (DeMers & Wachs, 2022)

$$\text{Mean Arterial Pressure} = \text{Diastolic Pressure} + \frac{1}{3}(\text{Systolic Pressure} - \text{Diastolic Pressure})$$

3.9 Perceptual measurements

3.9.1 Rating of Perceived Exertion

Rating of Perceived Exertion (RPE) quantifies an individual's perceived exertion during exercise using a Borg scale (G. Borg, 1990). The scale runs from 6 = very very light to 20 = maximal exertion, see below. Standardised instructions from the American College of Sports Medicine (ACSM) were told to every participant, "The feeling should reflect your total amount of exertion and fatigue, combining all sensations and feelings of physical stress effort and fatigue. Try not to underestimate or overestimate your feelings of exertion, be as accurate as you can." (American College of Sports Medicine, 2014).

After completing one set of 60°/s isokinetic knee extensions in both the heated and normothermic conditions, participants were shown a Borg rating of perceived exertion scale and asked to say or point where on the scale, if two numbers were given the highest was recorded. The rating of perceived exertion has been shown to be reliable for resistance exercise in both trained and untrained individuals (Lea et al., 2022). Below is the scale used within this thesis.

Rating Of Perceived Exertion

- 6
- 7 Very, very light
- 8
- 9 Very light
- 10
- 11 Light
- 12
- 13 Somewhat hard
- 14
- 15 Hard
- 16
- 17 Very hard

18

19 Very, very hard

20

2.9.2 Thermal Sensation scale

The Thermal Sensation Scale (TSS) measures thermal sensation at rest and during exercise, the TSS scale has been previously described and validated as a measure of thermal sensation (Toner et al., 1986). Every thirty mins participants were asked to identify how comfortable they felt in relation to the heating that they were experiencing, by asking “How hot or cold do you feel right now?” in accordance with Ganio et al., (2009). Participants were asked to point or say what degree on the scale, if two numbers were given the highest number was recorded. See below for the scale used within this thesis. Thermal sensation scales have demonstrated moderate correlations with core temperatures in younger adults (Ganio et al., 2009); although older adults often exhibit a blunted thermal sensitivity the thermal sensation scale remains responsive (Hoof & Hensen, 2006).

Thermal Sensation Scale

0.0 Unbearable Cold

0.5

1.0 Very Cold

1.5

2.0 Cold

2.5

3.0 Cool

3.5

4.0 Neutral (Comfortable)

4.5

5.0 Warm

5.5

6.0 Hot

6.5

7.0 Very Hot

7.5

8.0 Unbearably Hot

3.9.3 Rate of change scale

A global rate of change scale, previously deemed reliable by Bobos et al., (2020), was used to assess how “ready and capable for exercise the limb felt” the heated leg was compared to the normothermic limb. Participants were shown a visual scale and asked, “how does your heated leg feel compared to the other in terms of readiness and capability for exercise?”, if two numbers were given the highest number was recorded. See below for the scale used within this thesis.

Global Rate of Change Scale

- +7 A very great deal better
- +6 A great deal better
- +5 Quite a bit better
- +4 Moderately better
- +3 Somewhat better
- +2 A little bit better
- +1 A tiny bit better
- 0 About the same
- 1 A tiny bit worse
- 2 A little bit worse
- 3 Somewhat worse
- 4 Moderately worse
- 5 Quite a bit worse
- 6 A great deal worse
- 7 A very great deal worse

3.10 Data Reporting

3.10.1 Central Tendency

Measures of central tendency are used in this thesis to provide a summary of the dataset by identifying the central or typical value. This helps in understanding the general pattern or common trend in the data, making it easier to interpret the overall characteristics of the population or sample being studied (Manikandan, 2011).

3.10.1.1 Mean

The mean, or average, is calculated by summing all data values and dividing by the number of observations. It is used commonly within this thesis to represent the expected value in a dataset, allowing for comparison between different groups or variables (Manikandan, 2011).

3.10.1.2 Median

The median is the middle value when the data is arranged in order. It is used in the thesis to provide a more robust measure of central tendency, especially when the dataset includes outliers or is skewed. Unlike the mean, the median is not influenced by extreme values, making it a better indicator of the typical value in distributions that are not symmetrical (Manikandan, 2011).

3.10.2 Dispersion

Measures of dispersion are used in this thesis to describe the variability or spread of the data. While measures of central tendency provide a summary value, dispersion indicates how much the data deviates from this central point. This is crucial for understanding the reliability and consistency of the data, as well as identifying any potential variability within or between groups, this data is presented after the statement of values throughout this thesis, denoted by the \pm symbol.

Standard deviation quantifies the amount of variation or dispersion in a dataset relative to the mean. In this thesis, it is used to show how spread out the data points within the calculation of the mean. A smaller standard deviation indicates that the data points are close to the mean, suggesting more uniformity, while a larger value implies greater variability.

The standard error measures how much the sample mean is expected to vary from the true population mean (Lee et al., 2015). It is used in this thesis to assess the precision of the sample mean as an estimate of the population mean. A smaller standard error suggests that the sample mean is a more accurate reflection of the population mean and is most often used in within the creation of figures within this thesis.

3.11 Statistical analysis

All data were analysed using SPSS Statistical Software (Version 25, SPSS, Chicago, IL). Statistical significance was set at $p < 0.05$, data are reported as mean \pm SD unless otherwise stated. Global rate of change scales data was manually counted and reported as frequency of responses.

3.11.1 Checking of data

To assess responses to the thigh heating intervention and control, data were analysed for normality of distribution using the Shapiro-Wilk test, due to its superior power making it a reliable choice for assessing data distribution (Arnastauskaitė et al., 2021).

3.11.2 Data normality

The Shapiro-Wilk test was used to evaluate whether the dataset followed a normal distribution, a necessary assumption for parametric testing. Sphericity was assessed using Mauchly's test during the ANOVA, and where violations were detected, the Greenhouse-Geisser correction was applied to adjust the degrees of freedom accordingly.

3.11.3 Reliability analysis

3.11.3.1 ICC measures

The ICC evaluates the consistency or reproducibility of quantitative measurements. To confirm reliability of the protocol-intervention interaction, single measures ICC was chosen as the most appropriate measure of repeatability for the muscle function tests (Koo & Li, 2016), ICC estimates were calculated using absolute-agreement two-way mixed effects model (ICC3,1) and was used to identify the intra-day reliability of peak torque, EFP and RFD₅₀ produced for the heated and control leg during isokinetic contractions at 60°/s, 180°/s, 300°/s, and during isotonic contractions at 25% of MVIC force across four timepoints (0, +30, +60, +90 min).

3.11.3.2 Cronbach's alpha

Cronbach's Alpha is a measure of internal consistency or average correlation among items in a scale. In addition, Cronbach's Alpha ($C\alpha$) was used to calculate statistical reliability for the systemic physiological responses and psychological test answers. This was more appropriate due to the smaller number of datapoints and provided an averaged measures consistency result.

Single measures ICC and Cronbach's Alpha results were categorised as having excellent reliability if scores were >0.90 , good reliability if scores were >0.75 , moderate reliability >0.50 , and poor reliability for any values <0.50 (Koo & Li, 2016). Cronbach's Alpha has been described as a widely accepted measure of internal consistency (Tavakol & Dennick, 2011).

3.11.3.3 Coefficient of variation

CV expresses the extent of variability in relation to the mean of the population.

Coefficient of variation (CV) was calculated as the ratio between the standard deviation and the mean of each participant between visit 1 and visit 2 for each velocity and each timepoint, independently.

Equation 3. 5. Coefficient of Variation (CV)

$$\text{Coefficient of Variation} = (\text{Mean/Standard Deviation}) \times 100$$

3.11.3.4 Standard error of measurement (SEM)

SEM reflects the precision of individual scores on a test, estimating how much measured scores are spread around a "true" score.

The Standard Error of Measurement (SEM) was used to assess response stability via estimation of the standard error in a set of repeated scores. SEM was calculated by dividing the standard deviation of the sample by the square root of the sample size minus the ICC, in accordance with prior research (Weir, 2005). Intra-day SEM was calculated comparing peak baseline to peak 30 min in the control limb.

3.11.3.5 Minimal detectable change

MDC is the smallest change that can be detected by a measure that exceeds measurement error and reflects a meaningful change.

Minimum detectable change (MDC_{95}) represents the smallest change that can be interpreted as a true change beyond measurement error with 95% statistical confidence. In other words, an observed change exceeding the MDC_{95} has only a 5% probability of being attributable to random measurement variability rather than a real change in performance. The MDC_{95} therefore provides an estimate of the smallest quantifiable change that is likely to translate to a meaningful change in real-world performance and was calculated in line with prior work (Dontje et al., 2018).

3.11.4 Inferential analysis

Inferential statistics draw conclusions about the population based on sample data.

3.11.4.1 Analysis of Variance test (ANOVA)

Analysis of Variance (ANOVA) is a statistical method used to compare the means of three or more groups to determine whether at least one group differs significantly from the others. It works by partitioning the total variability in the data into components attributable to different sources (e.g., between-group and within-group variation). ANOVA is considered robust; especially in its repeated-measures or factorial forms, because it can tolerate modest violations of the normality assumption and is relatively insensitive to unequal group variances, particularly when sample sizes are equal or large (Kim, 2015). The data in this thesis was a mixture of normal and non-normally distributed with modest variance of the mean between participants within the testing groups. Further bolstering robustness, ANOVA focuses on overall patterns of variance rather than individual data points, making it more resilient to outliers or skewed distributions compared to other parametric tests. In ANOVA, a Type I error occurs when a true null hypothesis is incorrectly rejected, falsely suggesting a significant difference between groups, while a Type II error occurs when a false null hypothesis is not rejected, leading to a failure to detect a true effect or difference between groups. ANOVA is a robust statistical method that helps control for Type I error by assessing all group differences simultaneously, rather than through multiple individual comparisons, which would increase the risk of false positives. Additionally, ANOVA's capacity to detect true effects across multiple groups and conditions, especially when used with sufficient sample size and power, also reduces the risk of Type II error, making it a reliable tool for evaluating complex experimental designs involving several factors or repeated measures.

3.11.4.2 Two way ANOVA

Two-way ANOVA was used to determine main effect differences across timepoints (0, +30, +60, +90, +120 min), and between visits (visit 1, visit 2) for heart rate, blood pressure, thermal sensation and T_{tymp} .

3.11.4.3 Three way ANOVA

A three-way repeated-measures Analysis of Variance (ANOVA) was used to determine main effect differences across timepoints (0, +30, +60, +90, +120 min), between conditions (HEAT and CONT), between populations (YOUNGER and OLDER) and between visits (visit 1, visit 2) for muscle function T_{mu} , T_{skin} and RPE.

3.11.4.4 Post-hoc analysis

Bonferroni post-hoc adjusted pairwise comparisons were used where significant main effects occurred to identify interaction effects between individual timepoints between conditions and visits. Bonferroni post hoc analysis was used after a statistically significant result was found in ANOVA to determine exactly which group means differ from each other. Because multiple comparisons increase the risk of Type I error (incorrectly rejecting the null hypothesis), the Bonferroni method adjusts the significance level to control for this. Bonferroni has been identified as a reliable and appropriate adjustment following an ANOVA test to control for type I error (Armstrong, 2014).

3.11.5 Effect size analysis

Partial eta squared (η^2) is a measure of effect size used in ANOVA to indicate the proportion of the total variance in a dependent variable that is attributable to a specific independent variable or interaction, after accounting for other factors in the model. It tells us how much of the variability in the outcome can be "partially" explained by a particular factor. In this thesis effect sizes are considered small (≥ 0.01), medium (≥ 0.06), and large (≥ 0.14) in accordance with prior work (Richardson, 2011).

CHAPTER 4 - Experimental study 1 – Passive thigh heating improves isokinetic but not isotonic muscle function

4.1 Abstract

Skeletal muscle function is optimised when tissue temperature is elevated above resting within an optimal range; excessive elevations ($>40^{\circ}\text{C}$) may compromise muscle performance. This study examined muscle torque production and maximal velocity responses to passive thigh heating on two occasions to determine the efficacy and reliability of the intervention. Twenty participants (10 female) completed two identical visits, whereby one thigh was wrapped in a water perfused garment for 90 min; circulating 50°C water with the contralateral limb remaining unheated. Four maximal isokinetic repetitions were conducted at three speeds (slow, $60^{\circ}/\text{s}$, moderate, 180 and fast, $300^{\circ}/\text{s}$) and an isotonic set at 25% MVIC to assess muscle function on both limbs at baseline and every 30 min for 90 min. Muscle temperature (*vastus lateralis*) was assessed every 30 min. Heating increased muscle temperature from baseline ($32.2\pm 1.1^{\circ}\text{C}$) to 30 min ($36.8\pm 0.7^{\circ}\text{C}$) with further $0.4\pm 1.3^{\circ}\text{C}$ increases in the following 30 min periods ($p<0.05$). Heating increased peak torque during moderate ($+10\pm 12$ N.m) and fast ($+10\pm 11$ N.m) contractions from 30 min onwards relative to the unchanged control leg ($p<0.05$). Peak torque during slow isokinetic and isotonic contractions were unchanged. Rate of force production at 50ms and early force production was increased during the slow contraction by 14% and 15% respectively from 30 min in the heated leg whilst the control leg was unchanged. Isokinetic and isotonic force muscle function was found to have excellent reliability across all contractile speeds (ICC >0.9). Passive heating improves peak torque production during moderate and fast isokinetic contractions and increases early force production in slow isokinetic contractions.

4.2 Introduction

Optimal muscle function for physical activity occurs at temperatures above the physiological resting range i.e., 32-35°C (Bishop, 2003). Muscle temperature is usually increased prior to exercise requiring high force or high velocity production through an active (exercise) warm-up, which can raise muscle temperature by $\geq 3^\circ\text{C}$ (Marshall et al., 2015). Passive heating of skeletal muscle to raise muscle temperature prior to undertaking high force or high velocity contractions is an emerging field of research and has been proposed to be of benefit as a supplement to an active warm up, or even as a replacement for those who cannot partake in an active warm up before they engage in physical activity (McGorm et al., 2018), e.g., the elderly and those undergoing physical therapy. Passive heating can induce localised hyperthermia, i.e., an elevated temperature of target tissue e.g., skeletal muscle, by up to 6.5°C (Koch Esteves et al., 2021; Mitchell et al., 2008; Watanabe et al., 2024) with this likely an optimal temperature elevation given temperatures above 42°C potentiate protein degradation and declines in contractile function (Baracos et al., 1984; Essig et al., 1985; Ranatunga, 1984). Passive heating interventions can be implemented by a variety of means including hot water baths (Jackman et al., 2023; Rodrigues, Trajano, et al., 2020), water perfused garments (Gibson et al., 2023; K. Kim, Reid, et al., 2020), microwave diathermy (Draper et al., 1999; K. Kim, Monroe, et al., 2020) and environmental chambers (Ihsan et al., 2020; Sweet et al., 2024). Whilst all methods share the same primary outcome i.e., increased temperature of the skeletal muscle associated with impending contraction; whole body heating modalities often also increase core temperature. This systemic rather than local hyperthermia decreases central drive to the muscle impairing force production (Thomas et al., 2006). To avoid this, local heating e.g., via water perfused garments, increases muscle temperature whilst maintaining core temperature at normothermic levels (Gibson et al., 2023; Koch Esteves et al., 2021), with data demonstrating that this local response is ergogenic (Rodrigues et al., 2021).

Initial research investigating the effect of passive heat on muscle function primarily focused on cycle ergometry (Bergh & Ekblom, 1979; Sargeant, 1987) or field testing (Oksa et al., 1996) with later work investigating team sport simulations (West et al., 2016). Current research in this domain has primarily focused on isometric contractions which have demonstrated increased rate of force production and increased peak force production following hyperthermia (Chang et al., 2023; Mornas et al., 2022; Rodrigues et al., 2021). Whilst isometric contractions provide a controlled state to observe modifications in muscle function, they do not reflect the demands of dynamic daily living tasks, such as standing from a chair or locomotion, or sporting physical activities. Accordingly, insights into the potential benefits arising from passive heating interventions may be limited. Dynamic muscle function testing is often conducted using

dynamometry and this technique is considered the gold standard for knee extensor muscle function assessment, due to its high accuracy and reliability when assessing peak isometric (de Araujo Ribeiro Alvares et al., 2015), isokinetic (Feiring et al., 1990) and isotonic force production (Timm et al., 1992). Additionally, dynamometry readily facilitates the assessment of muscle function across a variety of contractile speeds, often between 30-300°/s (Grbic et al., 2017; Ivy et al., 1981; Molczyk et al., 1991), which may provide translational and mechanistic insight (Taylor et al., 1991; Tsiros et al., 2011). Finally, early rate of force production in lower limb muscles, i.e., force within 0-300 ms of contraction onset, has been highly correlated to sporting performance (Hernández-Davó & Sabido, 2014) and quality of life in older adults (Thompson et al., 2014), with changes quantifiable via dynamometry. Despite emerging data describing the ergogenic benefit of passive heating during the early stage of evoked isometric contractions (Rodrigues et al., 2021), at present there is a paucity of data examining the efficacy of passive heating to increase peak force production during dynamic contractions (Ramanauskiene et al., 2008). Although the physiological benefits of passive heating are being investigated, the perceptual responses have not yet been adequately studied. It is essential to thoroughly understand how participants subjectively experience passive heating to develop a protocol that is not only tolerable but also perceived as beneficial. Accordingly, there is a need to investigate the influence of local passive heating on dynamic contractions which represent real world contexts.

The reliability of physiological and functional outcomes during passive heating have seldom been reported. In the absence of this understanding, it is challenging to fully understand the true observed effect and minimal change required to be meaningful. In general, the reliability of knee extension exercise during dynamometry is considered excellent (ICC > 0.9) (Sáenz et al., 2010). Without assessing the reliability of this protocol only assumptions can be made regarding natural variation and error associated with the experimental design increasing the chance for type I errors to occur (Webb, 1992). Despite this, to the best of the authors' knowledge there is yet to be a comprehensive assessment of the inter-day reliability of knee extension exercise during prolonged passive heating interventions relative to an unheated time control.

The first aim of this experiment was to quantify the intra and inter-day reliability of muscle function and local (to the thigh) and systemic (whole body) physiological responses before and during 90 min passive thigh heating or control in a cohort of healthy young adults (Part 1). It was hypothesised that i) peak isokinetic torque, peak isotonic velocity, early force production (EFP) and rate of force development (RFD) measures taken during the heating protocol would display excellent inter and intraday reliability, ii) physiological measures would also display

excellent inter and intraday reliability. The second aim of this study was to observe the interaction between skeletal muscle hyperthermia and torque production with a view to understanding whether passive heating can enhance peak force production, utilising the established reliability data to explain observations (Part 2). In relation to this aim, it was hypothesised that muscle hyperthermia would increase peak isokinetic torque production including EFP and RFD across all contractile speeds, and that peak isotonic contractile velocity would be increased.

4.3 Methods

4.3.1 Participant Characteristics

Twenty active participants (ten female, age 23 ± 2 y, height 1.72 ± 8 m, mass 68.5 ± 73.2 kg, BMI 23.1 ± 3.3 kg/m², body fat 17 ± 4 %, peak isometric force, heated limb 238 ± 70 N, control limb 201 ± 65 N, all partaking in >60 min.week of physical activity) free of known illness and disease completed the study. All participants were non-smokers, with no history of heat intolerance or neuromuscular disorders and provided written informed consent prior to taking part. An estimated sample size of 20 participants was calculated using a formula for intraclass correlation coefficient (ICC)–based reliability analysis, provided from Walter et al. (1998) provided in Borg et al. (2022), whereby the expected intraclass coefficient for isokinetic dynamometry was predicted to be ≥ 0.9 (Maffiuletti et al., 2007); with a precision of 0.1 and a confidence level of 95% with two retest occasions. Sample size estimation associated with determining the ergogenic potential of the heating intervention identified that <20 participants were required. As this was fewer than the number of participants required for the reliability analysis, twenty participants were recruited. Female participants were requested to schedule both visits within the same menstrual cycle phase. The study was approved by the Brunel University of London Research Ethics Committee and was carried out in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to commencement of the study. A full description of participants recruitment, pre-trial controls and demographic assessment can be seen in 3.2 Participants.

4.3.2 Experimental design

Participants attended the laboratory to undertake two identical visits on two separate days at either at 9:00 or 13:00. Visits were separated by 7 ± 2 days and was time matched for each participant whereby the room temperature was measured to be $19 \pm 2^{\circ}\text{C}$ across all testing days. Participants abstained from heavy exercise (e.g. resistance or interval training, prolonged endurance activity or competitive sport), caffeinated drinks and supplements, and alcohol for 24 hours prior to the experimental visits. Following anthropometry and body composition assessments, physiological and perceptual measures were assessed first,

followed by muscle function. These measures were taken at baseline, then +30, +60, +90 min thereafter on both limbs, more detail on how isokinetic testing was implemented please see 3.6 Isokinetic dynamometry. Following instrumentation, due to space and equipment constraints, the right leg (60% of participants' dominant limb) was prepared to have the upper thigh heated for 90 min (HEAT) whilst the contralateral limb served as a control (CONT). The participants wore leggings with the experimental thigh (HEAT) wrapped in a custom garment consisting of silicon tubes enclosed in cotton material that circulated water at an outlet temperature of 50°C and a survival blanket for a period of 90 min. Figure 4. 1. displays the custom heated garment uncovered by survival blanket. The garment remained on the thigh for the entire testing protocol, including during muscle function assessment, whilst the contralateral control thigh (CONT) was left uncovered, see 3.5 Heating via water perfused trouser. The same limbs served as HEAT and CONT legs on both visits. The participants remained seated on the dynamometer throughout the testing protocol. Whilst not engaged in dynamometry exercise the participants had their feet resting on a chair with their knees bent at a ~90° angle. All contractions were conducted through a 75° to 175° range of motion. The first visit began with an assessment of anthropometric characteristics. Unshod standing stature was recorded using a stadiometer (SECA model 213, Hamburg, Germany), with mass was assessed using electronic scales (SECA model 875, Hamburg, Germany). Body fat percentage was determined in accordance with the Durnin & Womersley (1977) four-site skinfold method. See Figure 4. 1. for a schematic of the full testing protocol conducted during each visit.

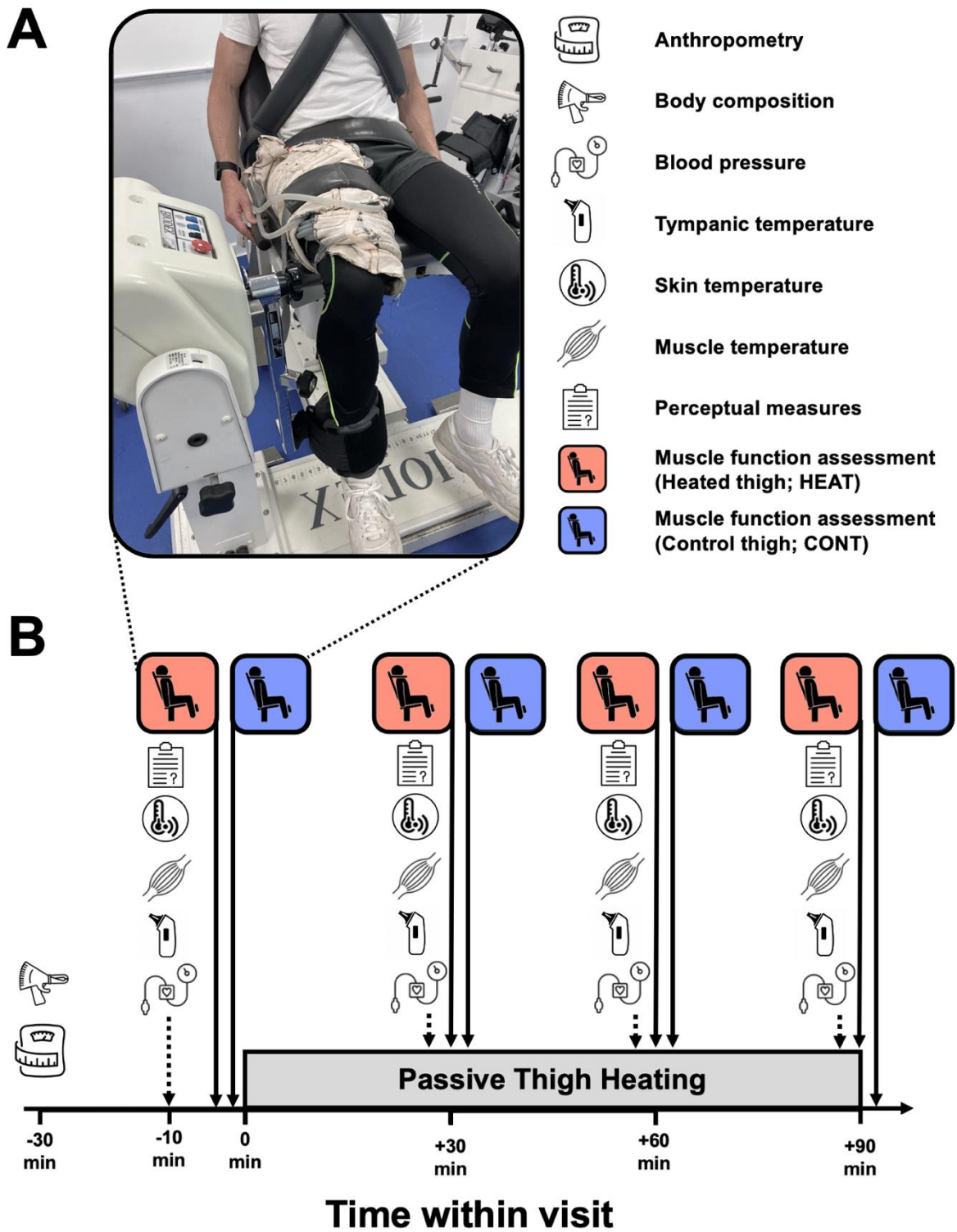


Figure 4. 1. Image of the custom-made water perfused heated garment as affixed on the thigh without the covering of the survival blanket (A). Sequence of the experimental protocols (B).

4.3.3 Physiological and perceptual measures

A tympanic membrane temperature device (Brawn Thermoscan 7, Bussigny, Switzerland) was set to the appropriate age setting and then fully inserted into the right ear canal whereby tympanic temperature (T_{tymp}) was recorded as a surrogate for core temperature (detail of the validity of this measure is explained in 3.8.1 Tympanic temperature). Heart rate (HR) and systolic (SBP) and diastolic blood pressure (DBP) and mean arterial pressure (MAP) were measured via an automated sphygmomanometer placed over the left brachial artery (Carescape V100 VitalSigns Monitor, Bolton). Muscle temperature (T_{mu}) was recorded using a muscle temperature probe (RS 103-433 K-type thermocouple, England). The probe was inserted, without local anaesthesia, ~30 mm below the skin surface at a 45° to the horizontal into the *vastus lateralis* via an 18-gauge hypodermic needle (Microlance 3, Ireland). Muscle temperature was manually recorded following temperature stabilisation (typically ~5 sec) with the probe and guide needle removed thereafter. Wireless iButton (DS1922L Thermochron Data Logger, UK) sensors were placed on the muscle belly of the *vastus lateralis* and used to measure thigh skin temperature (T_{skin}) at 1-min intervals. Participants responded to a global rate of change scale (Bobos et al., 2020), and thermal sensation scale (Young et al., 1987) prior to assessment of muscle function. A rating of perceived exertion (Borg, 1990) was shown and answered by participants after every set of knee extension at 60°/s, for more information on the scales used in this study please see 3.9 Perceptual measurements.

4.3.4 Muscle Function

Following the assessment of physiological measures, knee extensor function was assessed using a dynamometer (Biodex Medical Systems, Shirley, NY, USA). A warmup of 10 submaximal knee extensions (five at 50% maximum effort, three at 75% maximum effort, two at 90 of maximum effort) was conducted on each leg at a self-selected intensity and an assessment of their maximal voluntary isometric contraction force (MVIC) made on both limbs. At baseline (0 min), and +30, +60, +90 min thereafter participants performed four repetitions of maximal isokinetic knee extension at 60°/s, 180°/s, and 300°/s, separated by 60 s passive rest, then performed four isotonic contractions against 25% of their MVIC force. Testing was conducted in accordance with previous work investigating isokinetic (Blazquez et al., 2013) and isotonic muscle function (Cheng & Rice, 2005). At all timepoints the HEAT limb was assessed in full first, followed by CONT, therefore CONT testing occurred ~5 min following HEAT. The singular highest recorded torque value of each set were used independently for torque analysis, unless stated, across all contraction types at every timepoint. Rate of force development at 50 ms (RFD₅₀) was calculated as the first positive torque data point subtracted from the torque value at 50 ms after the first recorded value which was then divided by the

time elapsed in sec (Maffiuletti et al., 2016). RFD_{50} was calculated for repetitions at $60^\circ/s$ (RFD_{slow}), $180^\circ/s$ (RFD_{mod}), $300^\circ/s$ (RFD_{fast}) and for the isotonic contractions at 25% MVIC force ($RFD_{isotonic}$). Early force production (EFP) was calculated as peak torque produced at 0.18 s (Amaral et al., 2014) during the 4 repetitions at each velocity (i.e. at $60^\circ/s$ (EFP_{60}), $180^\circ/s$ (EFP_{180}), $300^\circ/s$ (EFP_{300}) and 25% MVIC force (EVP_{25})). The Biodex system 4 software was used to collect data at 100 Hz (Biodex Medical Systems, Shirley, NY, USA). Torque, position, and velocity data was collected within software every 10 millisecond then exported without filtering and imported to Microsoft Excel for analysis.

4.3.5 Statistical analysis

To assess muscle function test reliability, single measures ICC (ICC3,1) was used for intra-day (peak torque, EFP, RFD_{50}) and inter-day reliability (Koo & Li, 2016). Cronbach's Alpha (α) assessed systemic physiological and psychological test reliability. Coefficient of Variance (CV) was calculated between visits. Standard Error of Measurement (SEM) assessed response stability, and Minimum Detectable Change (MDC_{95}) estimated real-world performance changes. A three-way repeated-measures ANOVA analysed timepoints, conditions (HEAT, CONT), and visits for muscle function, T_{mu} , T_{skin} , and RPE. Two-way ANOVA assessed heart rate, blood pressure, thermal sensation, and T_{lymp} . Bonferroni post-hoc tests identified significant interactions (3.10 statistical analysis)

Data were analysed using SPSS (Version 25) with significance set at $p < 0.05$. Results are reported as mean \pm SD. Global rate of change scales were manually counted and reported as frequencies, for a complete description of the statistics used in this study please see 3.10 Data Reporting.

4.4 Results

4.4.1 Confirmation of reliability

Inter and intraday reliability of physiological and perceptual measures during passive thigh heating

The inter-day CV, ICC and MDC_{95} and intraday MDC_{95} was calculated for all physiological and perceptual measures (Table 4.1). When averaged across all timepoints all measures displayed low inter-day variability i.e., $CV \leq 10\%$. Skin temperature across timepoints in HEAT demonstrated excellent intra-day reliability (Cronbach's $\alpha > .90$) highlighting that the intervention delivered a consistent stimulus, with CONT T_{sk} demonstrating good reliability (Cronbach's $\alpha > .75$). Blood pressure and T_{lymp} also scored excellent reliability using the

Cronbach's α criteria ($>.9$). The T_{mu} in HEAT, demonstrated good reliability (Cronbach's α , $>.75$). The T_{mu} in CONT, thermal sensation score and RPE scored moderate reliability (Cronbach's α , $>.05$).

4.4.2 Inter and intraday reliability of lower limb force production during passive thigh heating

The inter-day CV, ICC and MDC_{95} and intraday MDC_{95} was calculated for peak torque production, RFD_{50} and EFP at 60, 180 and 300°/s contraction speeds, as well as for peak isotonic velocity and EVP at 25% MVIC force presented in Table 4.1. Inter day ICC was $>.9$ for all heated isokinetic peak torque production and $<.8$ for peak isotonic velocity, excellent CV values were displayed in all peak isokinetic torque and isotonic velocity values as well. Inter day ICC was $>.9$ for all heated EFP and $<.8$ for EVP. Excellent CV values were displayed within heated and control EFP_{180} and $EFP_{25\%}$. Good CV values were observed in EFP_{60} and EFP_{300} . All isokinetic RFD_{50} displayed good ICC $>.8$ scores, whilst the $RFD_{isotonic}$ displayed poor reliability (ICC $<.5$). Poor CV values were observed for all RFD_{50} values.

Table 4. 1.

The inter-day coefficient of variation (CV), minimal detectable change (MDC₉₅), statistical reliability (ICC) analysis, and intraday MDC₉₅ of peak force and peak velocity at 25% MVIC within isokinetic and isotonic contractions during 90 min of passive thigh heating (n = 20).

	CV%					SEM	Interday ICC				MDC ₉₅
	0 min	30 min	60 min	90 min	All timepoints	Inter-day	Estimation	Upper 95% confidence	Lower Confidence	95% Cronbach's α	Intraday
60/s HEAT (N.m)	9.6	7.7	8.6	8.0	8.5	3	0.93	0.95	0.88	0.96	9
60/s CONT (N.m)	8.5	6.7	6.0	7.9	7.3		0.94	0.96	0.90	0.97	
180/s HEAT (N.m)	11.0	5.9	4.8	5.6	6.9	2	0.95	0.97	0.93	0.98	6
180/s CONT (N.m)	7.5	6.9	6.1	5.2	6.4		0.94	0.96	0.90	0.97	
300/s HEAT (N.m)	8.5	6.1	4.7	5.0	6.1	3	0.95	0.97	0.92	0.97	7
300/s CONT (N.m)	9.8	7.3	8.1	6.4	7.9		0.91	0.95	0.85	0.96	
25% MVIC HEAT (°/S)	3.9	3.9	4.5	4.8	4.3	6	0.85	0.87	0.75	0.98	17
25% MVIC CONT (°/S)	3.9	3.1	3.7	5.5	4.0		0.82	0.85	0.70	0.97	

Table 4. 2.

The inter-day coefficient of variation (CV), minimal detectable change (MDC₉₅), statistical reliability (ICC) analysis, and intraday MDC₉₅ of Early force production and rate of force development at 50ms within isokinetic and isotonic contractions during 90 min of passive thigh heating (n = 20).

	CV%					SEM	Interday ICC				MDC ₉₅
	0 min	30 min	60 min	90 min	All timepoints		Inter-day	Estimation	Upper 95% confidence	Lower Confidence	
RFD 60/S HEAT (N.s ⁻¹)	25.0	10.3	11.4	11.7	14.6	42	0.84	0.90	0.74	0.92	99
RFD 60/S CONT (N.s ⁻¹)	10.7	15.2	8.3	10.1	11.1		0.82	0.90	0.76	0.91	72
RFD 180/S HEAT (N.s ⁻¹)	32.4	18.7	13.5	15.3	20.0	79	0.84	0.91	0.69	0.93	109
RFD 180/S CONT (N.s ⁻¹)	24.8	20.2	21.9	18.1	21.3		0.81	0.89	0.64	0.91	102
RFD 300/S HEAT (N.s ⁻¹)	48.9	23.3	27.4	30.0	32.4	34	0.83	0.95	0.83	0.92	97
RFD 300/S CONT (N.s ⁻¹)	35.4	26.9	32.7	33.8	32.3		0.89	0.94	0.79	0.95	68
RFD 25% MVIC HEAT (N.s ⁻¹)	32.5	40.1	29.3	34.7	34.1	114	0.39	0.56	0.19	0.56	67
RFD 25% MVIC CONT (N.s ⁻¹)	42.0	35.7	48.6	37.4	41.0		0.04	0.25	0.0	0.08	83
EFP 60/S HEAT (N.M)	13.3	8.8	6.5	8.8	9.3	5	0.94	0.96	0.90	0.97	8
EFP 60/S CONT (N.M)	14.7	13.0	7.9	7.6	10.8		0.89	0.93	0.83	0.94	9
EFP 180/S HEAT (N.M)	10.7	5.3	5.5	5.6	6.8	2	0.96	0.97	0.93	0.98	5
EFP 180/S CONT (N.M)	7.0	6.0	4.4	5.3	5.7		0.94	0.97	0.93	0.98	4
EFP 300/S HEAT (N.M)	8.6	15.1	5.9	9.1	9.7	3	0.90	0.93	0.85	0.95	5
EFP 300/S CONT (N.M)	9.8	8.4	10.0	9.9	9.5		0.88	0.92	0.82	0.94	5
EVP 25% MVIC HEAT (°/s)	8.4	6.7	9.9	8.4	8.4	13	0.85	0.90	0.78	0.92	17
EVP 25% MVIC CONT (°/s)	10.2	13.1	8.9	5.7	9.5		0.82	0.88	0.72	0.91	18

Table 4. 3.

The inter-day coefficient of variation (CV), minimal detectable change (MDC₉₅), statistical reliability (ICC) analysis, and intraday MDC₉₅ of Local heating responses, systemic physiological responses and perceptual responses to 90 min of passive heating (n = 20).

	CV%					SEM	Interday ICC	MDC ₉₅			
	0 min	30 min	60 min	90 min	All timepoints	Inter-day	Estimation	Upper 95% confidence	Lower Confidence	95% Cronbach's α	Intraday
T _{mu} HEAT	5.1	2.5	2.1	1.3	2.8	1	0.74	0.86	0.55	0.85	3
T _{mu} CONT (°C)	2.6	2.7	3.1	3.5	3.0		0.49	0.70	0.19	0.65	
T _{skin} HEAT (°C)	1.8	1.1	1.6	1.8	1.6	1	0.98	0.99	0.96	0.99	3
T _{skin} CONT (°C)	2.3	2.6	2.5	2.5	2.3		0.79	0.86	0.63	0.88	
T _{tymp} (°C)	0.7	0.4	0.6	0.5	0.6	0.2	0.59	0.73	0.42	0.93	1
SBP (mmHg)	6.1	5.8	3.7	6.1	5.4	9	0.57	0.75	0.40	0.92	25
DBP (mmHg)	8.3	5.8	6.8	5.5	6.6	9	0.62	0.79	0.45	0.94	25
MAP (mmHg)	6.8	4.9	4.1	5.2	5.2	5	0.64	0.80	0.48	0.94	14
HR (b.min ⁻¹)	10.0	8.8	9.4	8.1	9.1	8	0.46	0.66	0.29	0.88	22
Thermal Sensation	16.0	7.4	7.0	10.2	10.1	1	0.52	0.66	0.34	0.70	3
RPE HEAT	7.2	8.6	6.1	7.5	7.4	1	0.51	0.66	0.33	0.68	3
RPE CONT	6.3	6.9	6.9	6.3	6.6		0.34	0.52	0.13	0.51	

4.4.3 Responses to passive thigh heating

Local temperature responses to passive thigh heating

Muscle temperature differed when the main effect of Time ($F_{(3,27)} = 79.5$, $p = <.001$, $\eta^2 = .90$), Condition ($F_{(1,9)} = 44.5$, $p = <.001$, $\eta^2 = .83$), Visit*Time ($F_{(3,27)} = 5.3$, $p = .005$, $\eta^2 = .37$), Condition *Time ($F_{(3,27)} = 51.2$, $p = <.001$, $\eta^2 = .85$) was investigated. The T_{mu} in HEAT increased from baseline by $4.6 \pm 1.2^\circ\text{C}$ at 30 min, $5.0 \pm 1.3^\circ\text{C}$ by 60 min and $5.3 \pm 1.2^\circ\text{C}$ by 90 min, see Figure 4. 2. The T_{mu} in CONT increased from baseline by $1.5 \pm 1.4^\circ\text{C}$ at 30 min and maintained this temperature increase throughout testing. Skin temperature (Figure 4. 2) differed when observing main effects within Condition ($F_{(1,7)} = 300.4$, $p = <.001$, $\eta^2 = .98$), Time ($F_{(3,21)} = 313.0$, $p = <.001$, $\eta^2 = .98$), Condition*Time ($F_{(3,21)} = 181.4$, $p = <.001$, $\eta^2 = .96$). Post hoc testing revealed that T_{skin} increased in HEAT by $11 \pm 1^\circ\text{C}$ at 30 min which was sustained throughout the 90 min of heating. The CONT T_{skin} increased from baseline by $3 \pm 1^\circ\text{C}$ and maintained this temperature increase throughout testing.

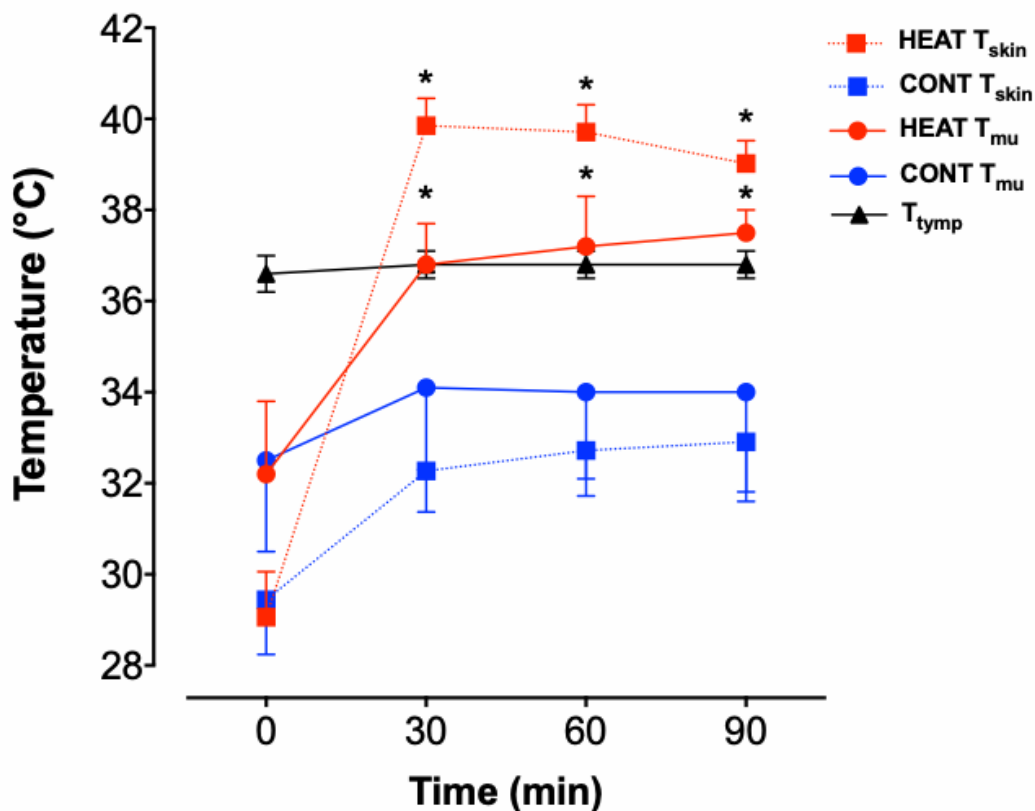


Figure 4. 2. Mean \pm SD Muscle temperature (T_{mu} , circles), thigh skin temperature (T_{skin} , squares) and tympanic temperature (T_{tymp} , triangles) during the passive thigh heating protocol (T_{tymp} and T_{skin} $n = 20$, $T_{mu} = 10$). * denotes significant difference between HEAT (red) and CONT (blue) at corresponding timepoint ($p < 0.05$)

4.4.4 Systemic physiological responses to passive thigh heating

Heart rate differed when the main effects of Time ($F_{(3,57)} = 7.7$, $p = .003$, $\eta^2 = .29$) were tested. Post hoc testing revealed that HR increased by 7 ± 10 b.min⁻¹ after baseline and remained at this until 60 min, no significant difference was observed at 90 min. Systolic blood pressure differed only when the main effects of Time ($F_{(3,57)} = 6.7$, $p = <.001$, $\eta^2 = .26$) was examined. SBP reduced by 9 ± 11 mmHg from baseline at 30 min, 7 ± 10 mmHg at 60 min and 5 ± 11 mmHg at 90 min. Diastolic blood pressure (DBP) differed when the main effects of Time ($F_{(3,57)} = 5.1$, $p = .003$, $\eta^2 = .21$) and Visit ($F_{(1,19)} = 4.7$, $p = .043$, $\eta^2 = .20$). There was a 3 ± 6 mmHg reduction in DBP in visit 2 and a 3 ± 6 mmHg reduction from baseline after 30 min that persisted until the end of testing. Mean arterial pressure (MAP) differed when the main effects of Time ($F_{(3,57)} = 8.7$, $p = <.001$, $\eta^2 = .32$) were tested. MAP reduced by 5 ± 6 mmHg after baseline and remained at this value thereafter. Tympanic temperature differed upon observing the main effect of Time only ($F_{(3,57)} = 10.8$, $p = <.001$, $\eta^2 = 1.0$) increasing by $0.2 \pm 0.3^\circ\text{C}$ at 30 min and maintained thereafter. No difference was observed between visits.

Table 4. 4. Physiological and perceptual responses to 90 min of passive thigh heating between two visits and averaged between visits (n = 20). Data are mean ± SD; **p*<0.05 vs control at corresponding timepoint; #*p*<0.05 vs baseline (0 min); ^ denotes difference between condition *p*<0.05, † denotes difference between visit *p*<0.05. SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure.

	0 min	30 min	60 min	90 min
Visit 1				
Heart rate (b.min ⁻¹)	75 ± 14	81 ± 13	81 ± 11	81 ± 13
SBP (mmHg)	125 ± 20	116 ± 8	117 ± 11	120 ± 15
DBP (mmHg) †	74 ± 10	71 ± 10	70 ± 8	68 ± 7 #
MAP (mmHg)	91 ± 12	86 ± 8	86 ± 8	85 ± 8
RPE (HEAT) ^	13 ± 2	15 ± 1	15 ± 2	16 ± 2
RPE (CONT)	15 ± 1	16 ± 1	16 ± 2	17 ± 2
Thermal sensation	4.0 ± 0.7	5.0 ± 0.6	5.2 ± 0.7	5.2 ± 1.0
Visit 2				
Heart rate (b.min ⁻¹)	74 ± 11	81 ± 9	80 ± 10	79 ± 10
SBP (mmHg)	123 ± 15	115 ± 13	118 ± 15	119 ± 12
DBP (mmHg) †	71 ± 10	68 ± 9	69 ± 10	68 ± 8
MAP (mmHg)	88 ± 11	83 ± 10	85 ± 9	85 ± 8
RPE (HEAT) ^	14 ± 1	15 ± 2	15 ± 2	15 ± 2
RPE (CONT)	15 ± 1	16 ± 2	17 ± 2	17 ± 2
Thermal sensation	3.5 ± 0.8	4.7 ± 0.6	5.1 ± 0.5	5.2 ± 0.6
Mean				
Heart rate (b.min ⁻¹)	74 ± 11	81 ± 9 #	81 ± 9 #	80 ± 10
SBP (mmHg)	125 ± 17	116 ± 9 #	118 ± 12 #	119 ± 13
DBP (mmHg)	73 ± 6	70 ± 4	70 ± 5	68 ± 4 #
MAP (mmHg)	90 ± 10	84 ± 8 #	86 ± 8 #	85 ± 7 #
RPE (HEAT) ^	14 ± 1 *	15 ± 1 *#	15 ± 1 *#	16 ± 1 *#
RPE (CONT)	15 ± 1	16 ± 1 #	16 ± 1 #	17 ± 1 #
Thermal sensation	3.8 ± 0.6	4.8 ± 0.4	5.2 ± 0.4	5.2 ± 0.5

4.4.5 Torque production following upper thigh muscle hyperthermia

Peak force production at 60°/s only differed where the main effects of Condition were observed ($F_{(1,19)} = 8.1$, $p = .010$, $\eta p^2 = .25$). Peak force production at 180°/s differed when the main effects of Condition ($F_{(3,57)} = 9.9$, $p = .005$, $\eta p^2 = .34$), and Condition*Time ($F_{(3,57)} = 4.5$, $p = .012$, $\eta p^2 = .19$) were examined. Post hoc comparisons are in Table 4. 5. and Figure 4. 3., whereby heating improved torque production by $+10 \pm 16$ N.m vs control from 30 min onwards ($p < 0.05$). The MDC_{95} for this measure was 6 N.m, 65% of the cohort could there be considered meaningfully changed beyond chance. Peak force production at 300°/s differed when the main effects of Visit ($F_{(1,19)} = 7.6$, $p = .013$, $\eta p^2 = .29$), Condition ($F_{(3,57)} = 12.0$, $p = .003$, $\eta p^2 = .39$), Visit*Time ($F_{(3,57)} = 2.9$, $p = .041$, $\eta p^2 = .13$) and Time* Condition ($F_{(3,57)} = 7.1$, $p = <.001$, $\eta p^2 = .27$) were examined. Full post hoc comparisons of all interactions of force production at 300°/s are located in Table 4.3 and revealed a $+4 \pm 6$ N.m increase in visit 2 vs visit 1 ($p < 0.05$) and an increase in force production during 300°/s contractions by $+10 \pm 11$ N.m in the heated condition vs control from 30 min onwards ($p < 0.05$). The MDC_{95} for this measure was 7 N.m, 75% of the cohort could therefore be considered as meaningfully increased beyond chance. Peak velocity at 25% of MVIC force differed when the main effect of Visit ($F_{(1,19)} = 4.7$, $p = .042$, $\eta p^2 = .20$) was examined. Post hoc testing, located in Table 4.3 revealed peak velocity at 25% of MVIC force was 8°/s greater in visit 1 vs visit 2 ($p < 0.05$). The effect of leg dominance on force production was examined across all angular velocities. No statistically significant differences were detected between dominant and non-dominant limbs at 60°/s ($p = .96$), 180°/s ($p = .83$), or 300°/s ($p = .79$). These findings indicate that, within this sample, limb dominance was not associated with measurable differences in peak torque at any of the tested velocities.

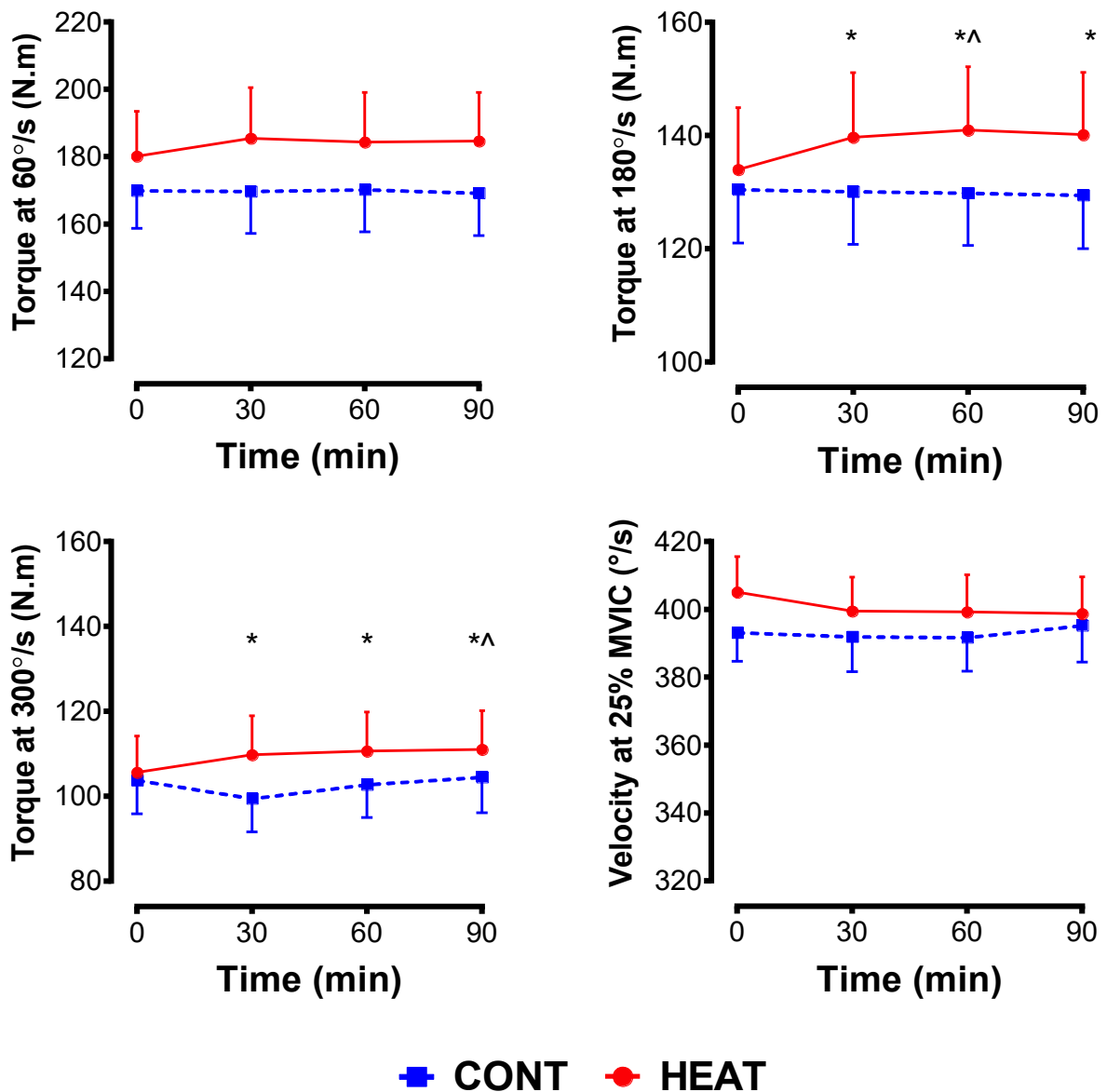


Figure 4. 3. Mean \pm SE Peak force production 60, 180, 300°/s and maximum velocity, over 90 min of passive thigh heating. * denotes significant difference between HEAT (red circles) and CONT (blue square) at corresponding timepoint ($p < 0.05$). ^ denotes significant difference from baseline ($p < 0.05$).

4.4.6 Rate of force development and early stage force production following upper thigh muscle hyperthermia

RFD_{slow} differed when the main effects of Condition ($F_{(1,19)} = 5.7$, $p = .027$, $\eta^2 = .23$), Time ($F_{(3,57)} = 4.7$, $p = .005$, $\eta^2 = .20$), Condition*Visit ($F_{(1,19)} = 8.6$, $p = .009$, $\eta^2 = .31$), Condition*Time ($F_{(3,57)} = 7.4$, $p = <.001$, $\eta^2 = .28$) and Visit*Time ($F_{(3,57)} = 8.4$, $p = <.001$, $\eta^2 = .31$). Post hoc comparisons are in Table 4.3 whereby only HEAT showed improved RFD₅₀ from baseline by $+122 \pm 133$ N.m.s⁻¹ at 30 min, $+154 \pm 168$ N.m.s⁻¹ at 60 min and $+155 \pm 187$

N.m.s⁻¹. No significant difference was observed between HEAT and CONT at baseline, HEAT was improved compared to CONT after 30 min by +143 ± 226 N.m.s⁻¹, 60 min by +151 ± 188 N.m.s⁻¹ and 90 min +156 ± 313 N.m.s⁻¹. RFD_{mod} differed when the main effects of Condition ($F_{(1,19)} = 7.4$, $p = .014$, $\eta^2 = .28$), Visit ($F_{(1,19)} = 14.7$, $p = .001$, $\eta^2 = .44$), Time ($F_{(3,57)} = 3.7$, $p = .035$, $\eta^2 = .16$), Condition*Time ($F_{(3,57)} = 3.2$, $p = .046$, $\eta^2 = .14$) and Visit*Time ($F_{(3,57)} = 4.1$, $p = .022$, $\eta^2 = .18$). Post hoc comparisons are in Table 4.3 whereby only HEAT showed improved RFD₅₀ from baseline by +142 ± 259 N.m.s⁻¹ at 30 min, +158 ± 256 N.m.s⁻¹ at 60 min and +138 ± 224 N.m.s⁻¹. No significant difference was observed between HEAT and CONT at baseline or 30 min, HEAT was improved compared to CONT after 60 min by +133 ± 174 N.m.s⁻¹ and 90 min +129 ± 192 N.m.s⁻¹. Visit 2 had an increased RFD₅₀ at baseline +241 ± 224 N.m.s⁻¹, 30 min +142 ± 197 N.m.s⁻¹ and at 90 min +120 ± 233 N.m.s⁻¹. RFD_{fast} differed when the main effects of Visit ($F_{(1,19)} = 13.6$, $p = .002$, $\eta^2 = .42$), no other main or interaction effects were observed. Post hoc testing revealed that visit 2 displayed higher RFD₅₀ results, visit 1 averaged 533 ± 416 N.m.s⁻¹ whilst visit 2 averaged 634 ± 443 N.m.s⁻¹. There was no significant main or interaction effects when observing RFD_{isotonic}.

Early force production at 0.18 s (60°/s) (EFP₆₀) differed when the main effects of Time ($F_{(3,57)} = 16.9$, $p < .001$, $\eta^2 = .47$), Visit*Time ($F_{(3,57)} = 5.6$, $p = .002$, $\eta^2 = .23$) and Condition*Time ($F_{(3,57)} = 4.0$, $p = .012$, $\eta^2 = .23$) were examined. EFP₆₀ did not differ over Time, or for the Visit*Condition interaction. Post hoc comparisons are in Table 4. 5. and Figure 4. 2., whereby heating improved EFP₆₀ by +12 ± 22 N.m vs control from 60 min, and +18 ± 15 N.m within HEAT from baseline after 90 min. EFP₆₀ was +4 ± 15 N.m greater in visit 1 vs visit 2 at the main effect level. The MDC₉₅ for this measure was 14 N.m, 95% of the cohort could be considered meaningfully changed beyond chance. EFP₁₈₀ only differed when the main effect of Condition ($F_{(1,19)} = 9.1$, $p = .007$, $\eta^2 = .32$) was examined. There was no difference in EFP₁₈₀ for all other main or interaction effects. EFP₁₈₀ was +8 ± 12 N.m greater HEAT vs CONT. EFP₃₀₀ differed for the main effect of Condition only ($F_{(3,57)} = 5.6$, $p = .002$, $\eta^2 = .23$). EFP₃₀₀ was +5 ± 10 N.m greater in HEAT vs CONT at the main effect level. EFP_{25%} differed for the main effect of Visit only ($F_{(1,19)} = 4.8$, $p = .041$, $\eta^2 = .20$). EFP_{25%} was +16 ± 27 °/s greater in Visit 2 vs Visit 1 at the main effect level.

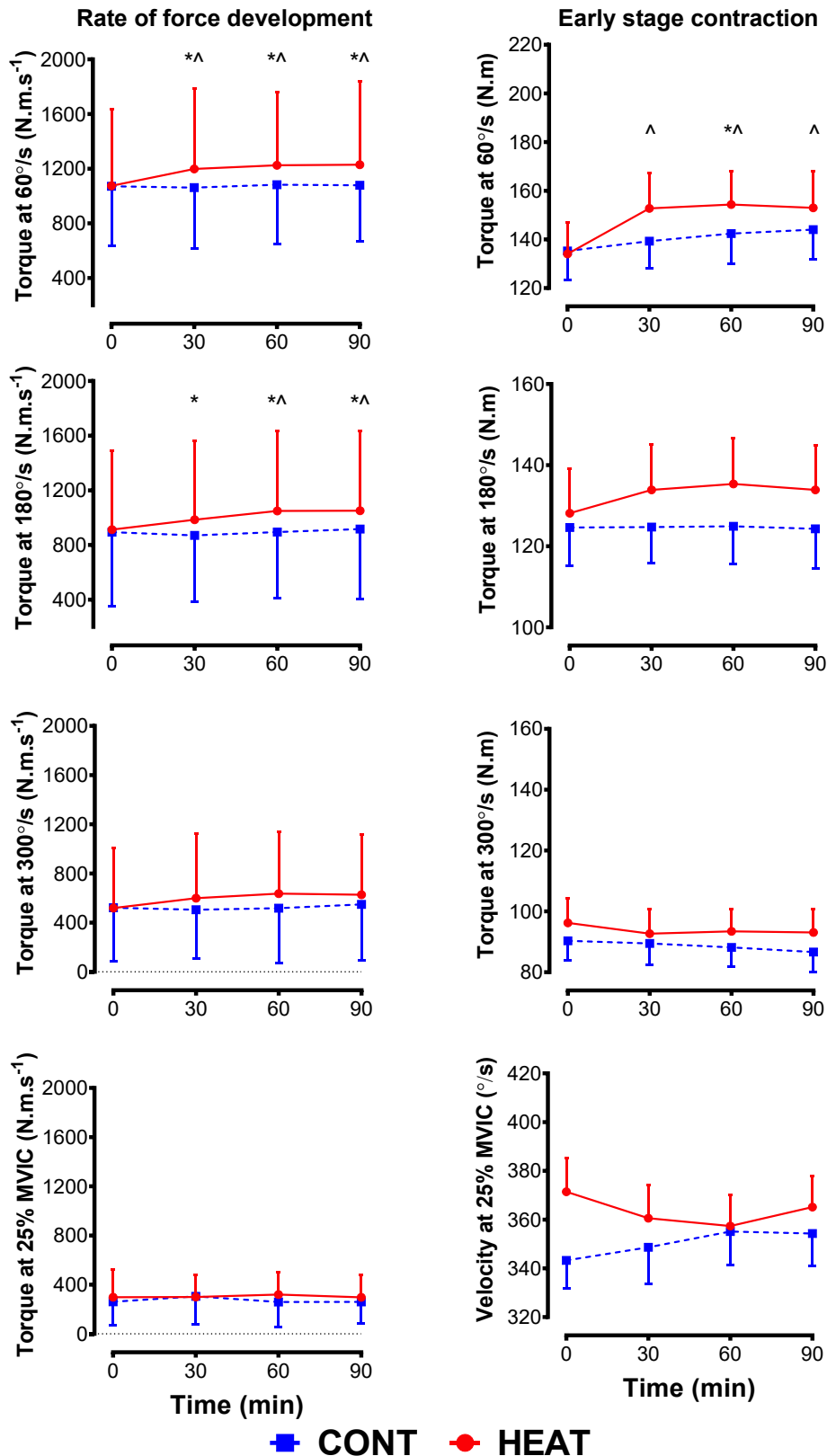


Figure 4. Mean \pm SE Rate of force development (left) and early force or velocity production (right) across 60, 180, 300°/s velocity production at 25% of MVIC force over 90 min of passive thigh heating. * denotes significant difference between HEAT (red circles) and CONT (blue square) at corresponding timepoint ($p < 0.05$). ^ denotes significant difference from baseline ($p < 0.05$)

Table 4 5. Mean \pm SD Peak torque and peak velocity generated at 60°/s, 180°/s and 300°/s and. 25% of MVIC force in heated (HEAT) and control (CONT) legs across 90 min of passive thigh heating separated by visit and averaged between visits (n = 20). * $p < 0.05$ vs control at corresponding timepoint; # $p < 0.05$ vs baseline (0 min); ^ denotes main effect difference between condition $p < 0.05$, † denotes difference between visit $p < 0.05$

	0 min	30 min	60 min	90 min
Visit 1				
Isokinetic Contractions - Peak torque (N.m)				
60°/s (HEAT)	186 \pm 62	189 \pm 71	191 \pm 72	187 \pm 66
60°/s (CONT)	175 \pm 56	176 \pm 63	174 \pm 63	170 \pm 59
180°/s (HEAT)	132 \pm 50	143 \pm 56	144 \pm 52	142 \pm 50
180°/s (CONT)	133 \pm 45	133 \pm 43	132 \pm 42	131 \pm 48
300°/s (HEAT) †	105 \pm 38	114 \pm 45	114 \pm 45	114 \pm 45
300°/s (CONT) †	105 \pm 38	105 \pm 38	103 \pm 36	103 \pm 37
Isotonic Contractions - Peak velocity (°/s)				
25% MVIC (HEAT) †	403 \pm 47	398 \pm 50	400 \pm 53	400 \pm 54
25% MVIC (CONT) †	393 \pm 39	394 \pm 49	391 \pm 51	392 \pm 56
Visit 2				
Isokinetic Contractions - Peak torque (N.m)				
60°/s (HEAT)	176 \pm 62	184 \pm 70	179 \pm 67	182 \pm 68
60°/s (CONT)	164 \pm 54	164 \pm 59	167 \pm 57	167 \pm 60
180°/s (HEAT)	139 \pm 50	140 \pm 51	141 \pm 51	142 \pm 52
180°/s (CONT)	131 \pm 45	130 \pm 47	127 \pm 44	131 \pm 44
300°/s (HEAT)	110 \pm 40	110 \pm 39	112 \pm 40	111 \pm 38
300°/s (CONT)	107 \pm 36	99 \pm 41	106 \pm 36	109 \pm 38
Isotonic Contractions - Peak velocity (°/s)				
25% MVIC (HEAT)	410 \pm 49	404 \pm 44	404 \pm 51	399 \pm 52
25% MVIC (CONT)	397 \pm 41	396 \pm 48	394 \pm 44	404 \pm 50
Mean				
Isokinetic Contractions - Peak torque (N.m)				
60°/s (HEAT) ^	180 \pm 60	185 \pm 67 *	184 \pm 66 *	185 \pm 65 *
60°/s (CONT)	170 \pm 51	170 \pm 57	170 \pm 56	169 \pm 57
180°/s (HEAT) ^	133 \pm 49	140 \pm 51 *	141 \pm 50 *	140 \pm 51 *
180°/s (CONT)	130 \pm 42	130 \pm 42	130 \pm 42	129 \pm 42
300°/s (HEAT) ^	106 \pm 38	110 \pm 41 *	111 \pm 41 *	111 \pm 41 *#
300°/s (CONT)	104 \pm 36	100 \pm 35	103 \pm 35	105 \pm 38
Isotonic Contractions - Peak velocity (°/s)				
25% MVIC (HEAT)	405 \pm 47	400 \pm 45	399 \pm 49	399 \pm 48
25% MVIC (CONT)	393 \pm 39	392 \pm 46	392 \pm 45	396 \pm 49

Table 4. 6.

Mean \pm SD Early force production (torque at 0.18 s) and early velocity production generated at 60°/s, 180°/s and 300°/s and. 25% of MVIC force in heated (HEAT) and control (CONT) legs across 90 min of passive thigh heating separated by visit and averaged between visits (n = 20). * $p < 0.05$ vs control at corresponding timepoint; # $p < 0.05$ vs baseline (0 min); ^ denotes main effect difference between visit $p < 0.05$

	0 min	30 min	60 min	90 min
Visit 1				
Isokinetic Contractions - EFP (N.m)				
60°/s (HEAT)	124 \pm 55	150 \pm 68	154 \pm 62	153 \pm 66
60°/s (CONT)	129 \pm 56	139 \pm 47	143 \pm 54	142 \pm 52
180°/s (HEAT)	123 \pm 50	132 \pm 53	135 \pm 52	133 \pm 49
180°/s (CONT)	123 \pm 43	122 \pm 38	125 \pm 42	121 \pm 44
300°/s (HEAT)	94 \pm 34	95 \pm 34	94 \pm 31	96 \pm 34
300°/s (CONT)	90 \pm 30	92 \pm 33	91 \pm 30	87 \pm 30
Isotonic Contractions - EVP (°/s)				
25% MVIC (HEAT) †	362 \pm 72	354 \pm 70	346 \pm 70	365 \pm 66
25% MVIC (CONT) †	381 \pm 57	367 \pm 58	369 \pm 55	366 \pm 58
Visit 2				
Isokinetic Contractions - EFP (N.m)				
60°/s (HEAT)	144 \pm 62	155 \pm 64	155 \pm 60	152 \pm 69
60°/s (CONT)	141 \pm 57	140 \pm 55	142 \pm 58	146 \pm 58
180°/s (HEAT)	133 \pm 49	135 \pm 48	136 \pm 50	135 \pm 50
180°/s (CONT)	127 \pm 42	127 \pm 43	125 \pm 41	128 \pm 43
300°/s (HEAT)	99 \pm 38	90 \pm 41	93 \pm 36	90 \pm 38
300°/s (CONT)	90 \pm 30	86 \pm 31	86 \pm 29	86 \pm 32
Isotonic Contractions - EVP (°/s)				
25% MVIC (HEAT)	328 \pm 64	330 \pm 89	353 \pm 74	354 \pm 64
25% MVIC (CONT)	358 \pm 44	367 \pm 56	357 \pm 60	354 \pm 60
Mean				
Isokinetic Contractions - EFP (N.m)				
60°/s (HEAT)	134 \pm 57	153 \pm 66 #	154 \pm 61 *#	153 \pm 67 #
60°/s (CONT)	135 \pm 54	139 \pm 49	142 \pm 55	144 \pm 54
180°/s (HEAT) ^	128 \pm 49	134 \pm 50	135 \pm 50	134 \pm 49
180°/s (CONT)	125 \pm 42	125 \pm 40	125 \pm 41	124 \pm 43
300°/s (HEAT) ^	96 \pm 36	93 \pm 36	93 \pm 33	93 \pm 35
300°/s (CONT)	90 \pm 29	89 \pm 31	88 \pm 29	87 \pm 30
Isotonic Contractions - EVP (°/s)				
25% MVIC (HEAT)	371 \pm 61	361 \pm 61	357 \pm 58	365 \pm 56
25% MVIC (CONT)	343 \pm 51	349 \pm 66	355 \pm 62	354 \pm 59

Table 4. 7.

Mean \pm SD Rate of force development at 50ms generated at 60°/s, 180°/s and 300°/s and 25% of MVIC force in heated (HEAT) and control (CONT) legs across 90 min of passive thigh heating separated by visit and averaged between visits (n = 20). * $p < 0.05$ vs control at corresponding timepoint; # $p < 0.05$ vs baseline (0 min); ^ denotes main effect difference between condition $p < 0.05$, † denotes difference between visit $p < 0.05$, ‡ denotes difference between visit $p < 0.05$

	0 min	30 min	60 min	90 min
Visit 1				
Isokinetic Contractions – RFD ₅₀ (N.m.s ⁻¹)				
60°/s (HEAT) †	988 \pm 534	1229 \pm 550	1296 \pm 502	1255 \pm 496
60°/s (CONT)	1280 \pm 436	1283 \pm 499	1280 \pm 420	1323 \pm 598
180°/s (HEAT) †	824 \pm 532	954 \pm 520	1083 \pm 577	1053 \pm 521
180°/s (CONT)	1106 \pm 535	1137 \pm 571	1114 \pm 506	1135 \pm 568
300°/s (HEAT) †	460 \pm 506	600 \pm 494	614 \pm 507	601 \pm 475
300°/s (CONT) †	650 \pm 448	651 \pm 537	719 \pm 473	717 \pm 477
Isotonic Contractions - RFD (N.m.s ⁻¹)				
25% MVIC (HEAT)	290 \pm 210	301 \pm 197	319 \pm 170	303 \pm 161
25% MVIC (CONT)	295 \pm 165	356 \pm 228	301 \pm 210	272 \pm 174
Visit 2				
Isokinetic Contractions – RFD ₅₀ (N.m.s ⁻¹)				
60°/s (HEAT)	1105 \pm 376	1153 \pm 394	1158 \pm 366	1128 \pm 312
60°/s (CONT)	1150 \pm 356	1074 \pm 362	1115 \pm 364	1138 \pm 349
180°/s (HEAT)	841 \pm 523	853 \pm 423	903 \pm 422	876 \pm 427
180°/s (CONT)	1042 \pm 484	954 \pm 474	978 \pm 472	1035 \pm 508
300°/s (HEAT)	493 \pm 435	498 \pm 353	467 \pm 409	534 \pm 401
300°/s (CONT)	581 \pm 414	553 \pm 427	596 \pm 460	602 \pm 487
Isotonic Contractions - RFD (N.m.s ⁻¹)				
25% MVIC (HEAT)	324 \pm 228	313 \pm 148	339 \pm 178	311 \pm 179
25% MVIC (CONT)	267 \pm 210	306 \pm 232	233 \pm 174	272 \pm 157
Mean				
Isokinetic Contractions – RFD ₅₀ (N.m.s ⁻¹)				
60°/s (HEAT) ^	1134 \pm 469	1256 \pm 511*#	1288 \pm 444*#	1289 \pm 535*#
60°/s (CONT)	1127 \pm 356	1113 \pm 356	1137 \pm 357	1133 \pm 316
180°/s (HEAT) ^	965 \pm 514	1045 \pm 537*	1098 \pm 525*#	1094 \pm 530*#
180°/s (CONT)	942 \pm 488	904 \pm 426	940 \pm 430	956 \pm 450
300°/s (HEAT)	555 \pm 441	625 \pm 507	667 \pm 478	659 \pm 462
300°/s (CONT)	537 \pm 416	526 \pm 380	532 \pm 425	568 \pm 438
Isotonic Contractions - RFD (N.m.s ⁻¹)				
25% MVIC (HEAT)	292 \pm 148	328 \pm 179	310 \pm 179	287 \pm 134
25% MVIC (CONT)	296 \pm 152	310 \pm 148	286 \pm 125	292 \pm 125

4.4.7 Perceptual measures following upper thigh hyperthermia

Rating of perceived exertion differed when the main effects of Condition ($F_{(1,19)} = 15.0$, $p = .001$, $\eta^2 = .44$) and Time ($F_{(3,57)} = 24.0$, $p < .001$, $\eta^2 = .56$) was examined. Thermal sensation differed when the main effect of Time ($F_{(3,57)} = 48.6$, $p < .001$, $\eta^2 = .72$), increases in thermal sensation as time progressed, see Table 2. In response to the global rate of change scale, 80% of participants self-reported that heating made them feel at least “a little bit better” in terms of readiness for exercise, 50% self-reported that heating made them feel at least “moderately better” in terms of readiness for exercise and 35% self-reported that heating made them feel at least “a great deal better” in terms of readiness for exercise.

4.5 Discussion

This is the first study to evaluate the inter and intraday reliability of knee extensor torque production across a variety of contractile speeds and physiological responses at multiple timepoints during a passive thigh heating protocol. The peak isokinetic torque values displayed excellent intraday reliability for both the heated and control conditions. Reliability for EFP at all contraction velocities was also excellent while $RFD_{(slow)}$, $RFD_{(mod)}$, $RFD_{(fast)}$, and EVP was moderately reliable whilst $RFD_{(isotonic)}$ displayed low intraday reliability. A poor coefficient of variation was observed within the RFD values; however, this is likely due to the exaggerated difference when torque values are divided by the elapsed time. Although good intraday reliability was observed within the ICC values of the RFD scores, the findings of significant differences between visits for RFD_{slow} , RFD_{mod} , RFD_{fast} may indicate poorer reliability; it is worth noting that this too, may be exaggerated by the inflating the torque values, and also their differences, by dividing by the elapsed time in sec. It does not appear that passive hyperthermia negatively influences reliability relative to the control limb, indeed in the context of RFD and EFP measures, reliability is typically superior in the heated vs control limb. Systemic physiological responses i.e., HR, blood pressure and T_{tymp} responses displayed very good reliability with the reliability of local physiological markers i.e., T_{mu} and T_{sk} identified as good.

The passive thigh heating intervention increased muscle temperature in the HEAT condition +4.6°C at 30 min, +5.0°C at 60 min and +5.3°C at 90 min, in contrast CONT saw an increase from baseline of only 1.5°C after 30 min which was sustained throughout testing. The increase in T_{mu} occurred in the absence of meaningful changes in systemic physiological markers highlighting the localised action of the intervention. This study also quantified the change in isokinetic and isotonic muscle function (peak isokinetic torque, EFP and peak isotonic velocity) following a bout of passive heating relative to a control. To this end, from 30 min onwards heating increased peak torque during moderate contractile speeds by 8% (+10 N.m) and

during fast contractile speeds by 10% (+10 N.m) relative to the control. Heating was also found to increase RFP₅₀ by 14% (+155 N.m⁻¹) and early force production by 15% (+20 N.m) during the slow contraction speed (60°/s) when compared to baseline and the control. To contextualise the magnitudes of change observed in this study, the group mean increases in muscle function are also above our calculated MDC in moderate and fast contractions as well as for the early force production at 60°/s with 65%, 75% and 95% of individuals exceeding the calculated MDC thresholds respectively. The group mean change in RFD_{slow} was also above calculated MDC, 85% of all individuals exceeded the MDC threshold. It was observed that heating improved “readiness for exercise” and reduced perception of exertion while being a thermally comfortable experience, likely due to the absence of change in systemic physiological responses. In agreement with our hypothesis and adding confidence to the reported ergogenic effects of the intervention, the inter-day repeatability of peak torque during slow, moderate and fast contractions and peak velocity achieved during leg extension exercise following passive heating was excellent according to the ICC testing.

4.5.1 Reliability of muscle function and systemic physiological assessments

Excellent reliability has been documented when testing isokinetic knee extension (Maffiuletti et al., 2007; Toonstra & Mattacola, 2013), peak torque (Sole et al., 2007) and isotonic contractions (Van Driessche et al., 2018) across days. We now extend this understanding by identifying that isokinetic and isotonic dynamometry following passive heating produces reliable results between days. Our study therefore finds that the addition of heating does not alter reproducibility of peak torque outcomes, maintaining similar intraday reliability to unheated isokinetic dynamometry (Holmbäck & Lexell, 2007; Kambič et al., 2020). When comparing to similar work investigating rate of force production during isokinetic exercise (Brown et al., 2005) lower ICC scores (0.58) than the current study (0.84) whilst CV scores that suggested lower variability were observed. A review on RFD reliability reports that ICC scores for RFD typically range between 0.8-0.9 (Davó & Solana, 2014), whilst the current study is in agreement for the isokinetic trials, the poor reliability of the isotonic trials is unexplained.

Further research into the intraday reliability of isokinetic and isotonic RFD measurements, including CV are required. In addition to quantifying the reliability of our protocol, and likely that of others using similar approaches (Chang et al., 2023; Mornas et al., 2022; Rodrigues et al., 2021).

4.5.2 Performance response to passive thigh heating

A key finding of this study is that passive heating enhanced peak isokinetic torque at moderate (180°/s) and fast (300°/s) contraction velocities. Whilst preliminary research into isometric contractions did not identify benefits to peak force production (Morrison et al., 2004; Rodrigues et al., 2021; Thornley et al., 2003), increases in early force development i.e. an increased rate of force development (+26%) at 50 ms have been observed (Rodrigues et al., 2021). Additionally, passive heat exposure has also been reported to accelerate fascicle shortening velocity and improving the rate of force development (+48%) during the first 100 ms of a voluntary explosive isometric contraction (Mornas et al., 2022). Although there are subtle differences between rate of force production and EFP these results align with our EFP₆₀ observations where early force production, an important metric due to its relationship with dynamic physical activities, enhanced by 20 N.m. (+15%) in the 60°/s heated condition after 30 min of heating and remained in this enhanced state throughout the heated protocol. Increases in early force production likely occurred in the 60°/s contractions only, as this was the only contractile speed slow enough to elicit maximal/near maximal voluntary force production with observation of this phenomenon typically restricted to isometric contractions (Maffiuletti et al., 2016). The isokinetic contraction type used in this study may explain why our study saw only a +14% increase in RFD₅₀ as opposed to the >25% increases observed during prior isokinetic work; this is likely a continuation of the effect of contractile speed on the rate of force development. From a translational perspective, there does not appear to be additional benefit in applying passive thigh heating for 60 and 90 min suggesting that a 30 min intervention may be sufficient to elicit muscle function benefits. Other heating interventions, e.g., whole body heating, may however require extended protocols to elicit equivalent intramuscular responses.

Increasing muscle temperature, both actively and passively, before physical activity has been demonstrated to enhance muscle temperature (Faulkner et al., 2013). To further support our findings of the benefits of heating on peak torque being most pronounced in the moderate and faster isokinetic contractions aligns with prior sprint cycling studies whereby faster cadences saw greater increases in power output following lower limb passive heating than slower cadences (Sargeant, 1987). The mechanisms underpinning an increase in isokinetic force production following passive heating remain unclear; however, it has been suggested that in addition to sodium channel activated increases in nerve conduction velocity (Kiernan et al., 2001; Todnem et al., 1989) augmented Ca²⁺ handling occurs (Kobayashi et al., 2005). Increased intramuscular temperature elevates Ca²⁺ storage reserve in the sarcoplasmic reticulum (Godt & Lindley, 1982; Kobayashi et al., 2005; Ranatunga, 1984), which then simulates the SERCA pump to increase Ca²⁺ movement into and out of the working muscle

(Davies and Young 1983). This alteration reduces half-relaxation time and increases muscle shortening velocity and force production (Rodrigues et al., 2022). An increase in intramuscular fluid has also been suggested to be an important element associated with enhanced muscular function by stiffening the muscle-tendon unit (Eng et al., 2018). The increased fluid within the epimysium and perimysium within the fixed area of the muscle increases muscle stiffness allowing for a more efficient transfer of force (Hughes et al., 2015). Whilst heating is known to influence metabolic systems within the muscle (Brocherie et al., 2024; Girard et al., 2015) this response is unlikely to be a factor in short periods of maximal exercise as investigated within this study. While the fastest isokinetic contraction ($300^{\circ}/s$) saw the greatest benefit supporting this mechanism, in contrast isotonic velocity did not see any improvement; despite being the fastest contractile velocity ($415 \pm 50^{\circ}/s$). This null response in isotonic contractions may be explained in part by the velocity limiting factor of ATP disassociation from the myosin head (Nyitrai et al., 2006) and decreases in passive muscle and active tendon stiffness which result in unchanged late and global rates of force development (Mornas et al., 2022).

Systemic physiological responses, particularly T_{tymp} were unmeaningfully changed displayed excellent reliability, suggesting that the passive thigh heating protocol did not increase core temperature. In addition to removing the known performance impairment associated with whole body hyperthermia (Thomas et al., 2006), minimising increases in core temperature reduces the potential for hypotension induced syncope or other negative events associated with hyperthermia. Muscle temperature in the heated condition demonstrated good reliability with the absolute ($37\text{-}38^{\circ}\text{C}$) and relative ($+4\text{-}6^{\circ}\text{C}$) increase in muscle temperature consistent with other research using water perfused garments (Gibson et al., 2023; Ihsan et al., 2020; Koch Esteves et al., 2021), and similar to changes observed during exercise (Gibson et al., 2014; Kapnia et al., 2023; Kenny et al., 2003). Our observed peak muscle temperature signals the attainment of a physiological plateau, as the temperature gradient between the muscle and core temperature diminishes, and the heating impulse decreases concurrently with increased intramuscular blood flow (Heinonen et al., 2011; Koch Esteves et al., 2021). Only when core temperature increases above this apparent plateau does muscle temperature increase further during passive hyperthermia via water perfused garments (Watanabe et al., 2024). To achieve higher temperatures, alternative heating interventions such as diathermy are likely necessary, although at the current time it remains unknown whether increasing skeletal muscle temperature above the magnitudes observed in this study would elicit greater increases in muscle function. From a perceptual perspective, when asked how the heated leg felt compared to the control leg in terms of “readiness for exercise” most respondents reported that heating improved their perceived readiness for exercise by at least “a little bit”. The heated condition saw increases in force production but a decrease in a rating for perceived exertion,

this may be linked to the analgesic effect of heating (Malanga et al., 2015). The reported enhancement in "readiness for exercise" underscores the potential of localised heating as a preparatory strategy for physical activity that does not undesirably perturb systemic physiology.

4.5.3 Practical implications and limitations

The findings from this study have practical implications across various fields, particularly in sports performance, physical therapy, and geriatrics. Traditional active warmups have been demonstrated to not improve isokinetic force at 60, 180 or 300°/s (Park et al., 2018; Rodrigues, Gabellone Hernandez, et al., 2020) and may have reduced intramuscular pressure and stiffness as measured by an increase in mechanomyographic signals (Altamirano et al., 2012). The demonstrated reliability of muscle function and physiological responses following passive heating suggests that this method could be integrated into warm-up routines for athletes to enhance performance and used in conjunction with in-competition heat maintenance strategies (Faulkner et al., 2013; Raccuglia et al., 2015). The increase in peak torque and early force production, especially at moderate and fast contraction speeds, highlights that passive heating an effective strategy for improving muscle force production before activities requiring high/maximal exertion e.g., athletic competition. The increases in muscular force presented in this study (+10 N.m, 8%) provides similar benefits to widely used performance aids such as caffeine (+5 N.m, 3%) (Grgic et al., 2022) and L-arginine (+4.2%) (Zart et al., 2023) and may therefore be considered a surrogate or complement to other interventions that are designed to improve muscle function. Additionally, the protocol could benefit individuals in physical therapy settings, where the combination of increased muscle function and reduced perceived exertion could accelerate recovery while minimizing pain. The use of this approach as a non-invasive, thermally comfortable, and ergogenic intervention strategy adds significant value to pre-exercise and therapeutic protocols, including as a part of late-stage rehabilitation protocols as a means to accelerate training adaptations via optimised molecular responses. Finally, passive preheating may be useful for older adults who are unable to conduct a traditional warm-up prior to physical activity such as climbing stairs or walking.

This research provides further foundations to continue research into the effects of passive heating on muscle function in other cohorts e.g., older adults, and contexts e.g., physical therapy and rehabilitation. As previously discussed, the mechanisms that underpin the augmentation of muscle function following heating are largely unknown, this work may help guide work on investigating the underlying mechanisms of passive heating aiding performance by providing different contractile types and speeds tested at the same muscle temperature

across multiple timepoints. Some limitations of this experimental design include a lack of familiarisation to the protocol as well as a potential inconsistent motivation between sets and days influencing effort. Intra-day variability in motivation could have influenced the psychological responses. These may not be a consideration that appears relevant to our serial measurements as evidenced by equal or lowering CV values throughout the protocol. It was possible that there was a physiological effect of having heating only applied to one leg as it is not possible to placebo the heating although we saw high inter-day reliability further research may wish to investigate muscle activation during the trials. This study is limited in its analysis of RFD₅₀ due to the 100HZ export rate from the Biodex, this rate does not provide the highest level of accuracy that may be provided by other machines. This study did not perfectly balance the dominant limb between HEAT and CONT groups, this remains unclear how this may have influenced responses. The serial muscle function measures within this study also modestly increased muscle temperature in the control leg from exercise from baseline every 30 min, thus did not simulate how heating may attenuate the decline in muscle function that long periods of true sedentary behaviour may incur. It could therefore be postulated that passive heating would have an even greater effect when assessed against inactive and normothermic musculature. Previous research on prolonged passive heating observed decreases in muscle temperature in the control (CONT) condition likely due to the absence of physical activity (Gibson et al., 2023). In contrast, our study did not observe a decrease in muscle temperature in the control condition, likely due to the exercise being performed. This study also only focused on a young healthy population and did not measure the retention/decay of increased muscle function post cessation of heating. Future research should investigate the mechanisms underpinning the observed changes following heating and explore different how heating modalities, timings and conditions affect muscle function across a variety of populations. Finally research exploring how heating may counteract longer acute sedentary muscle function deterioration could provide useful as the general population spends multiple hours sedentary.

4.6 Conclusion

In summary, peak isokinetic and isotonic muscle function, isokinetic EFP and RFD, local thigh temperature and systemic physiological responses following passive upper thigh heating produce highly reliable outcomes between days. Passive heating of the thigh for 30 min increased muscle temperature by 4.6°C which enhanced isokinetic muscle function during moderate and fast contractile velocities by ~10% and increases rate of force production at 50ms and early force production during slow isokinetic contractions by ~14% and ~15% respectively, whilst the control limb muscle function was unchanged.

CHAPTER 5 - Experimental study 2 – Passive thigh heating improves peak force production in younger adults and early isokinetic force production in younger and older adults

5.1 Abstract

Older adults often suffer from a reduced capability to complete physical activity or daily tasks relative to young adults in part due to impaired muscle function. This study investigated the ergogenic effects of passive thigh heating on knee extensor torque production healthy older vs younger adults. Twenty-two young (YOUNGER; 23±3 y) and sixteen older adults (OLDER; 68±8 y) completed an experimental visit whereby one thigh was heated via a garment circulating 50°C water for 90 min with the contralateral limb unheated. Four maximal contractions were performed at three isokinetic speeds (slow, 60°/s, moderate, 180 and fast, 300°/s) and an isotonic set (25% maximal voluntary isometric contraction); contractions were performed on both limbs at baseline and every 30 min thereafter for 120 min with the final timepoint used to quantify the retention/decay in response. *Vastus lateralis* temperature was measured every 30 min and surface electromyography implemented throughout to monitor muscle activation. Heating increased the pooled muscle temperature from baseline (31.7±1.7°C) at 30 min (36.5±1.5°C), 60 min (37.1±1.4°C), 90 min (37.5±0.7°C) and at 120 min (35.5±2.3°C), all $p < 0.05$. Heating increased peak torque during moderate (+11±12 N.m) and fast (+7±11 N.m) contractions in only YOUNGER participants relative to their control leg which remained unchanged ($p < 0.05$). After 30 min the rate of force development in HEAT was increased during slow contractions from baseline in both groups (+229 ± 210 N.m.s⁻¹ $p < 0.05$). Early force production increased in YOUNGER and OLDER during the slow contractions) from 60 min in HEAT ((+15±15 N.m $p < 0.05$). Peak EMG amplitude was unchanged throughout. Passive thigh heating improves peak knee extensor torque in young adults during moderate and fast isokinetic contractions and increases the rate of force development and early force production in slow isokinetic contractions in both younger and older adults.

5.2 Introduction

Muscle strength and power predicate's effective human function and contributes to a higher quality of life. Muscular strength facilitates postural control (Topp et al. 1997), supports locomotion (Anderson et al. 2007) and a higher level of muscular strength has been correlated to a reduced risk of falling (Pizzigalli et al. 2011). Further to this, maintaining muscular power can fall risk, facilitate independent living and maintain a higher quality of life during ageing (Rice and Keogh 2009; Gray and Paulson 2014; Freitas et al. 2024). The decline of muscle function is often observed from the age of 40 years (McGregor et al. 2014) and is attributed to a loss of muscle mass, degradation of neural pathways, decreases in mitochondrial function, chemical handling and reductions in hormone levels (Morley et al. 2001; Cruz-Jentoft et al. 2010; Cruz-Jentoft and Sayer 2019). These changes in muscle physiology are commonly quantified as reduced maximal force production, a proxy for maximal strength (Tøien et al. 2025), and slower rates of force production, a proxy for muscle power (Barry et al. 2005; Thompson et al. 2013). This decrease in muscle function can result in mobility disorders, increased risk of falls and injury, loss of independence and diminished quality of life (Cruz-Jentoft et al. 2010).

In conjunction with general age-related declines in muscular strength and power production, older adults are often exposed to prolonged periods of inactivity, which can contribute to a reduced muscle temperature (Gibson et al. 2023). Skeletal muscle has been reported to perform optimally during physical activity at temperatures above the resting physiological range i.e., 32-35°C (Bishop 2003), with hyperthermic skeletal muscle displaying increases in force production relative to normothermic tissue (Ranatunga 1984; Sargeant 1987). While an increase in muscle temperature is normally achieved through a traditional active warm up (Racinais et al. 2017) for some populations, e.g., those who have developed frailty and immobility (Strandberg et al. 2011), this is impractical. Acute increases in muscle temperature elicit a variety of physiological benefits, including, but not limited to, an increase in localised blood flow into and out of the heated muscle (Chiesa et al. 2015; Watanabe et al. 2024), improved myofibrillar calcium handling (Kobayashi et al. 2005), altered muscle-tendon stiffness (Rodrigues et al. 2022), optimised penetration angle (Eng et al. 2018), and increases in ATP turnover and muscle fibre conduction velocity (Gray et al. 2006).

Experimental literature investigating functional outcomes associated with acutely increased skeletal muscle temperature in young adults has observed that thigh/leg heating increases early force production and rate of force development at 50ms during isometric and slow (60°/s) isokinetic contractions (Rodrigues et al. 2021; Denny et al. 2025), increases peak torque during moderate and fast isokinetic contractions (Denny et al. 2025), and improves force

production and/or performance during functional movements such as increased jump height (Skurvydas et al. 2008) and cycle sprinting (Sargeant 1987). Taken together, it has been proposed that an acute increase in muscle function across the aforementioned contraction types and facilitated by increased muscle temperature, may aid physical activity and the performance of everyday tasks in cohorts with sub-optimal muscle function. At the current time, investigation into the efficacy of passive heating of skeletal muscle as a means to improve contractile function across multiple contraction types and velocities in older adults is limited and whether the intervention has efficacy beyond cohorts of young adults remains unknown. Should observations regarding the ergogenic benefit of passive heating in younger adults also be observed in older cohorts, passive heating may be considered a viable intervention to enhance the ability of older adults to complete daily living tasks (Hortobágyi et al. 2003; Katula et al. 2008; Khodadad Kashi et al. 2023).

Whilst prior work in young adults has identified passive heating as a potential ergogenic aid to improve muscle function, the dose-response relationship is also yet to be effectively characterised. Additionally, whilst it is understood that maintaining increased muscle temperature is important for maintaining peak force production (Faulkner et al. 2013), the retention and/or decay following heating cessation have yet to be fully quantified. Beyond the physiological and functional benefits, the study of psychological effects following passive heating have been restricted to pain management (Chabal et al. 2020). The discomfort associated with physical activity is inversely associated with enjoyment or pleasure when partaking in physical activity or exercise (Ekkekakis et al. 2005; Ekkekakis and Petruzzello 2012). Therefore, understanding whether heating improves these sensations, i.e., perceived effort and feelings of readiness to participate in physical activity is noteworthy in the context of exercise initiation and adherence. Perceptions of self-ability have been identified as barriers to physical activity for older adults (Rúa-Alonso et al. 2023), a simple heating intervention may help increase confidence and improve perceptions of readiness for exercise. It is also important that participants believe that the intervention is beneficial for adherence or usage of the tool if it is to be implemented.

The primary aim of this study was to quantify the effect of 90 min of passive thigh heating on peak torque and early force production and rate force development during isokinetic contractions in younger vs. older adults. The secondary aim of the study was to measure the retention/decay of the anticipated improvements in isokinetic peak torque and early force production in younger vs. older adults 30 min post cessation of heating. The final aim was to examine the perceptual responses to the heating protocol and quantify how both younger and older adults perceived passive heating to influence muscle function. It was hypothesised that

passive heating would enhance peak isokinetic torque at moderate and fast contractile speeds, while also improving rate of force development and early force production at the slowest contractile speed in both younger and older adults. These enhancements were expected to persist for up to 30 min following the heating intervention. Additionally, both age groups were anticipated to perceive passive heating as beneficial to muscle function.

5.3 Methods

5.3.1 Participant Characteristics

The study was approved by the Brunel University of London Research Ethics Committee (44577-MHR-Oct/2023- 47671-3) and was carried out in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to commencement of the study. Twenty-two younger (YOUNGER) (12 male, 10 female; age 23 ± 3 y; height 1.72 ± 0.08 m; mass 68.5 ± 12.5 kg; BMI 23.1 ± 3.3 kg·m⁻²; body fat $17 \pm 4\%$; peak isometric force: heated limb 238 ± 70 N·m, control limb 201 ± 65 N·m) and sixteen older (OLDER) adults (8 male, 8 female; age 68 ± 8 y; height 1.65 ± 0.10 m; mass 67.7 ± 13.1 kg; BMI 24.8 ± 4.4 kg·m⁻²; body fat $27 \pm 4\%$; peak isometric force: heated limb 153 ± 82 N·m, control limb 124 ± 78 N·m), free of known illness or disease, completed the study. All participants were non-smokers, with no history of heat intolerance or neuromuscular disorders. OLDER completed the WHOQOL-BREF questionnaire (von Steinbüchel et al., 2006) with all participants indicating a high quality of life relative to the international validation study of the WHOQOL-BREF (Skevington et al., 2004). Older adults were excluded from participation if they were taking medication that thinned the blood or was an anti-coagulant; a discussion was had with each older adult to ensure that their medication would not interfere with a natural heating response or muscle function. The target sample size was estimated from a prior experimental study involving younger adults only (Chapter 4). An effect size of 0.6 was calculated for the difference in torque produced between the heated condition and control condition within the analysis. Therefore, in conjunction with an α set to 0.05 and β at 0.8 a sample size of at least 16 participants per group was required. A subset of younger participants for this study had previously undertaken an associated reliability experiment where younger adults only were recruited (Chapter 4). Younger females were not controlled for menstrual phase and for older females this was not a relevant requirement as they were post-menopausal. A full description of participants recruitment, pre-trial controls and demographic assessment can be seen in 3.2 Participants.

5.3.2 Experimental design

Participants visited the laboratory (ambient room temperature YOUNGER = $19 \pm 2^\circ\text{C}$, OLDER = $19 \pm 2^\circ\text{C}$) for a single visit either at 9:00 or 13:00 having abstained from heavy exercise (e.g. resistance training, prolonged endurance activity or competitive sport), caffeine, and alcohol for 24 hours prior to the experimental visits, which was verbally confirmed by the participants. A separate familiarisation visit was not included due to both the single-visit experimental design and practical time constraints, particularly for the OLDER cohort. Older adults were required to tolerate a prolonged laboratory protocol involving invasive muscle temperature assessment and repeated maximal contractions; the inclusion of an additional visit would have substantially increased participant burden and may have negatively impacted recruitment and retention. Importantly, all participants were recreationally active and capable of independent locomotion, with the OLDER group reporting high quality of life and regular engagement in physical activity. As such, participants were accustomed to producing high-effort lower-limb contractions, reducing the likelihood that performance during maximal isokinetic testing was limited by task unfamiliarity. To further minimise learning effects, all participants completed a structured warm-up consisting of progressive submaximal contractions (50–90% perceived maximum) followed by maximal voluntary isometric contractions on each limb prior to data collection. Additionally, the contralateral limb control design ensured that any residual familiarisation effects would be present in both HEAT and CONT conditions. Consequently, any remaining learning-related improvements would be expected to bias results toward the null rather than exaggerate the effect of passive heating.

Physiological and perceptual measures were assessed first followed by muscle function; these measures were taken at baseline (0 min), then +30, +60, +90, +120 min thereafter on both limbs. Following instrumentation, the right leg (60% of participants' dominant limb) was prepared to have the upper thigh heated for 90 min (HEAT) whilst the contralateral limb served as a control (CONT), see 3.5 Heating via water perfused trouser. The participants wore leggings with the experimental thigh (HEAT) wrapped in a custom garment that circulated water at an outlet temperature of 50°C and a survival blanket for a period of 90 min that remained on for the entire testing protocol, including during muscle function assessment, whilst the contralateral control thigh (CONT) was left uncovered. The participants remained seated on the dynamometer throughout the testing protocol. Whilst not engaged in exercise the participants had their feet resting on a chair with their knees bent at a $\sim 90^\circ$ angle. All contractions were conducted through a 75° to 175° range of motion for a full explanation of how isokinetic dynamometry was used in this study see 3.6 Isokinetic dynamometry. Figure 5. 1. provides a schematic overview of the experimental design.

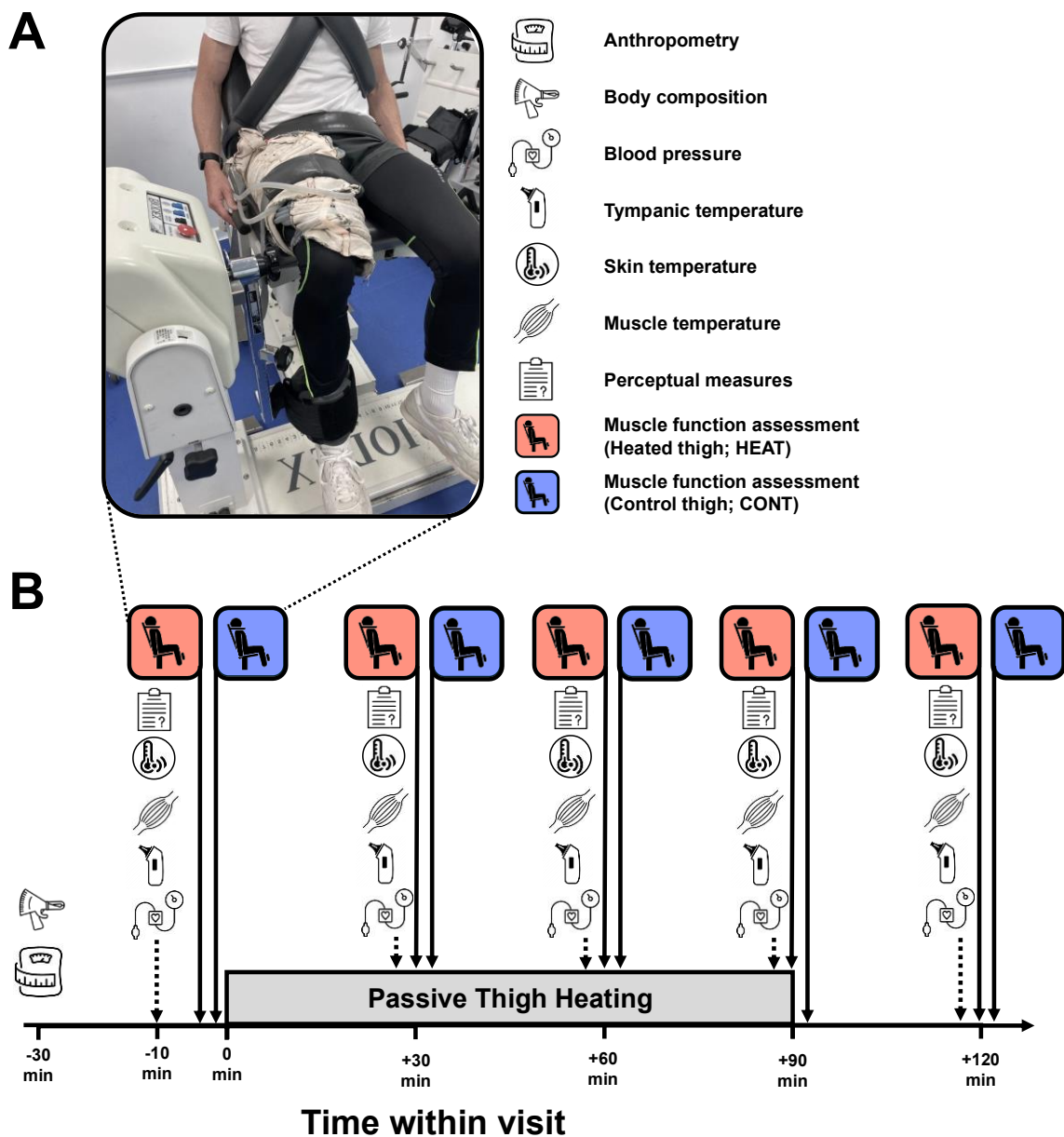


Figure 5. 1. Image of the custom-made water perfused heated garment as affixed on the thigh without the covering of the survival blanket (A). Sequence of the experimental protocols (B).

5.3.3 Physiological and perceptual measures

A tympanic membrane temperature device (Brawn Thermoscan 7, Bussigny, Switzerland) was set to the appropriate age and then fully inserted into the right ear canal whereby tympanic temperature (T_{tump}) was recorded as a surrogate for core temperature. Heart rate (HR) and systolic (SBP) and diastolic blood pressure (DBP) and mean arterial pressure (MAP) were measured via an automated sphygmomanometer placed over the left brachial artery (Carescape V100 VitalSigns Monitor, Bolton). Muscle temperature (T_{mu}) was recorded using a muscle temperature probe (RS 103-433 K-type thermocouple, England). The probe was

inserted, without local anaesthesia, ~30 mm below the skin surface at a 45° to the horizontal into the vastus lateralis via an 18-gauge hypodermic needle (Microlance 3, Ireland). Muscle temperature was manually recorded following temperature stabilisation (typically ~5 sec) with the probe and guide needle removed thereafter. Wireless iButton (DS1922L Thermochron Data Logger, UK) sensors were placed on the muscle belly of the vastus lateralis and used to measure thigh skin temperature (T_{skin}) at 1-min intervals. Participants responded to a global rate of change scale (Bobos et al. 2020), and thermal sensation scale (Young et al. 1987) prior to assessment of muscle function. A rating of perceived exertion (Borg 1990) was shown and answered by participants after every set of knee extension at 60°/s, for more information on the scales used in this study please see 3.9 Perceptual measurements.

5.3.4 Muscle Function and surface electromyography

Following the assessment of physiological measures, knee extensor function was assessed using a dynamometer (Biodex Medical Systems, Shirley, NY, USA). A warm-up of 10 submaximal knee extensions (five at 50% maximum effort, three at 75% maximum effort, two at 90% of maximum effort was conducted on each leg at a self-selected intensity), followed by an assessment of maximal voluntary isometric contraction force (MVIC) on both limbs. At baseline (0 min), and +30, +60, +90 min and +120 min thereafter participants performed four repetitions of maximal isokinetic knee extension at 60°/s, 180°/s, and 300°/s, separated by 60 passive rest then performed four isotonic contractions against 25% of their MVIC. Muscle function testing was conducted in accordance with previous work investigating isokinetic (Blazquez et al. 2013) and isotonic muscle (Cheng and Rice 2005). At all timepoints the HEAT limb was assessed in full first, followed by CONT. The singular highest recorded torque value of each set was used independently for torque analysis, unless stated, across all contraction types at every timepoint. Measures of early muscular force and power production were taken as prior research suggests rapid force and power development are severely diminished with ageing (Bellumori et al. 2013; Crozara et al. 2013). Prior research suggest that localised passive heating can increase rate of force development at 50 ms (RFD_{50}) in younger adults (Rodrigues et al. 2021; Mornas et al. 2022). RFD_{50} was calculated as the first positive torque data point subtracted from the torque value at 50 ms after the first recorded value, which was then divided by the time elapsed in sec (Maffiuletti et al. 2016). RFD_{50} was calculated for repetitions at 60°/s. Peak force produced at 0.18s was recorded as a measure of early force production (EFP), with EFP recorded during the 4 repetitions at each velocity (i.e. at 60°/s (EFP_{60}), 180°/s (EFP_{180})). Contractions at 300°/s and the isotonic contractions at 25% MVIC were not reported for EFP as older adults were not able to consistently produce measurable force at 0.18 s. The peak torque value of each set was analysed across all contraction types at every timepoint. EFP was calculated as peak force produced at 0.18 s during the 4

repetitions produced during contractions at 60°/s (EFP60) and at 180°/s (EFP180). The Biodex system 4 software were used to collect data at 100Hz. Torque, position, and velocity data was therefore collected within software every 10 millisecond, then exported without filtering and imported to Microsoft Excel for analysis. EMG acquisition software (Delsys Discover, Boston, MA USA) was used to collect the raw EMG data from the EMG sensors (Delsys Trigno, Boston, MA USA). The data was plotted within the acquisition software before being exported. Once exported the data were processed with custom MATLAB code. Raw EMG signals were bandpass filtered with a 6-450Hz cut-off frequency in accordance with similar studies (Gordon et al. 2023) before subtracting the mean of the signal to correct baseline-offsets. The filtered signal then underwent full-wave rectification and low-pass filtering to produce a linear envelope using a dual-pass 2nd order Butterworth filter, after which peak amplitude within each set was reported.

5.3.5 Statistical analysis

All data were analysed using SPSS Statistical Software (Version 25, SPSS, Chicago, IL), (3.10 Statistical Analysis). A three-way repeated-measures analysis of variance (ANOVA) was used to determine main effect differences across timepoints (0, +30, +60, +90 min), between conditions (HEAT and CONT) and between groups (YOUNGER, OLDER) for muscle function T_{mu} , T_{skin} and RPE. For all other experimental variables, a two way ANOVA was used to determine main effect differences across timepoints (0, +30, +60, +90 min), and between groups (YOUNGER, OLDER). A two way ANOVA was also used to determine main effect differences between peak isometric force between conditions (HEAT and CONT) and groups (YOUNGER and OLDER). An independent sample t-test was used to determine differences in participant characteristics between groups. Bonferroni post-hoc adjusted pairwise comparisons were used where significant main effects occurred to identify interaction effects between individual timepoints between conditions and visits. Statistical significance was set at $p < 0.05$, data are reported Mean \pm SD. Global rate of change scales had results manually counted and reported as frequency of responses.

5.4 Results

5.4.1 Participant characteristics

By design, between group differences were observed for age, with greater body fat also observed in the OLDER group. Participants were successfully matched for all other anthropometric characteristics. Peak isometric force analysis revealed an age*condition interaction ($F_{(1,35)} = 9.3$, $p = .004$, $\eta^2 = .21$) whereby significantly greater force was observed in YOUNGER vs OLDER in both HEAT (+86 \pm 144 N.m, +43%) and CONT (+75 \pm 134 N.m,

+47%). Further to this, differences between HEAT and CONT were observed for YOUNGER (+30 ± 13 N.m, +17%) and OLDER (+19 ± 16 N.m, +21%).

5.4.2 Systemic physiological and local temperature responses to passive thigh heating

Muscle temperature analysis revealed an effect of condition ($F_{(1,11)} = 23.5$, $p < .001$, $\eta^2 = .27$), with the effect of time also significant ($F_{(1,44)} = 36.7$, $p < .001$, $\eta^2 = .77$). Most noteworthy is the significant interaction effects of condition and time ($F_{(4,44)} = 18.4$, $p < .001$, $\eta^2 = .63$) whereby significant increases in T_{mu} from baseline were detected in HEAT at 30 min (+4.3 ± 1.9°C), 60 min (+4.9 ± 1.7°C), 90 min (+5.3 ± 1.6°C) and 120 min (+2.4 ± 2.3°C). A significant increase in T_{mu} was observed in CONT from baseline, at 30 min (+1.6 ± 1.1°C), 60 min (+1.8 ± 1.6°C) and 90 min (+1.8 ± 1.8°C) however the change was no longer significant at 120 min (+1.8 ± 1.9°C). Muscle temperature in HEAT was not different at baseline, but significantly higher than CONT at 30 min (+2.4 ± 1.9°C), 60 min (+2.9 ± 1.6°C) and 90 min (+3.3 ± 2.0°C), no significant difference was seen at 120 min (+0.4 ± 1.9°C). Skin temperature showed significant differences when considering the main effects of condition ($F_{(1,24)} = 80.0$, $p < .001$, $\eta^2 = .77$), the effect of time was also significant ($F_{(4,96)} = 30.8$, $p < .001$, $\eta^2 = .56$). The interaction effect between condition and time is most relevant ($F_{(4,96)} = 24.2$, $p < .001$, $\eta^2 = .50$) whereby no significant differences were observed at baseline between HEAT and CONT, but significant increases in HEAT from baseline were observed at 30 min (+10.1 ± 3.0°C), 60 min (+10.4 ± 4.0°C), 90 min (+9.8 ± 2.3°C), and 120 min (+5.5 ± 4.8°C). CONT saw no significant changes from baseline. There was no difference between YOUNGER and OLDER in any temperature response with combined temperature data presented in Figure 4. 2. Mean arterial pressure differed when the main effects of time was observed ($F_{(4,140)} = 6.1$, $p < .001$, $\eta^2 = .15$), a significant change of -5 mmHg was observed at 60 min and a -8 mmHg at 90 min. No main or interaction effects were found when observing T_{tymp} (grand mean 36.3 ± 1.1°C) or heart rate (grand mean 75 ± 10 b.min⁻¹). Results were not different between condition at any timepoint and did not differ between YOUNGER and OLDER. Full post hoc comparisons can be found in table 5 1.

Table 5. 1.

Physiological and perceptual responses to 90 min of passive thigh heating and 30 min heat decay, YOUNGER (n = 22), OLDER (n = 16), Muscle temperature in younger (n = 10) and older adults (n=3) and skin temperature in younger (n = 15) and older adults (n=14). Data are mean \pm SD; * $p < 0.05$ vs control at corresponding timepoint; # $p < 0.05$ vs baseline (0 min); ^ denotes difference between condition $p < 0.05$.

	0 min	30 min	60 min	90 min	120 min
YOUNGER					
Heart rate (b.min ⁻¹)	75 \pm 14	81 \pm 13	81 \pm 11	81 \pm 13	82 \pm 13
T _{mu} (HEAT) (°C) ^	31.7 \pm 1.7	36.5 \pm 1.5	37.1 \pm 1.4	37.5 \pm 0.7	35.5 \pm 2.3
T _{mu} (CONT) (°C)	32.3 \pm 1.8	34.1 \pm 1.5	34.1 \pm 1.8	34.3 \pm 2.1	34.0 \pm 2.0
T _{skin} (HEAT) (°C) ^	28.9 \pm 1.5	39.4 \pm 0.9	39.0 \pm 2.6	39.6 \pm 1.2	34.3 \pm 0.8
T _{skin} (CONT) (°C)	30.1 \pm 1.9	32.7 \pm 1.7	33.3 \pm 1.4	33.3 \pm 1.1	33.3 \pm 1.
T _{tymp} (°C)	36.7 \pm 0.6	36.8 \pm 0.4	36.9 \pm 0.4	36.8 \pm 0.4	36.7 \pm 0.5
MAP (mmHg)	91 \pm 12	86 \pm 8	86 \pm 8	85 \pm 8	88 \pm 7
RPE (HEAT) ^	13 \pm 2*	15 \pm 1*#	15 \pm 2*#	16 \pm 2*#	16 \pm 2*#
RPE (CONT)	15 \pm 1	16 \pm 1	16 \pm 2	17 \pm 2	17 \pm 2
Thermal sensation	3.9 \pm 0.9	5.0 \pm 0.6#	5.2 \pm 0.7#	5.2 \pm 0.9#	4.0 \pm 0.8#
OLDER					
Heart rate (b.min ⁻¹)	69 \pm 13	71 \pm 13	71 \pm 11	69 \pm 10	70 \pm 11
T _{mu} (HEAT) (°C) ^	32.2 \pm 0.8	36.1 \pm 0.4	36.7 \pm 0.5	37.2 \pm 0.7	33.3 \pm 2.6
T _{mu} (CONT) (°C)	32.2 \pm 1.0	33.6 \pm 1.1	33.9 \pm 0.8	33.8 \pm 0.6	34.1 \pm 1.4
T _{skin} (HEAT) (°C) ^	30.5 \pm 1.7	38.9 \pm 1.7	39.2 \pm 1.1	39.3 \pm 1.2	34.5 \pm 0.7
T _{skin} (CONT) (°C)	30.6 \pm 1.5	32.6 \pm 1.1	33.1 \pm 1.0	33.2 \pm 1.2	33.3 \pm 1.5
T _{tymp} (°C)	36.2 \pm 0.4	36.3 \pm 0.4	36.4 \pm 0.4	36.4 \pm 0.3	36.5 \pm 0.3
MAP (mmHg)	102 \pm 11	98 \pm 9	97 \pm 13	93 \pm 12	99 \pm 21
RPE (HEAT) ^	15 \pm 1	14 \pm 2	15 \pm 2	15 \pm 2	15 \pm 2
RPE (CONT)	14 \pm 1	15 \pm 2	16 \pm 2	15 \pm 2	15 \pm 2
Thermal sensation	3.7 \pm 1.0	4.7 \pm 0.7#	4.7 \pm 0.9#	5.0 \pm 1.1#	4.5 \pm 1#
ALL PARTICIPANTS					
Heart rate (b.min ⁻¹)	74 \pm 11	81 \pm 9	81 \pm 9	80 \pm 10	76 \pm 9
T _{mu} (HEAT) (°C) ^	32.0 \pm 1.2	36.6 \pm 0.9 *#	36.9 \pm 1.0 *#	37.3 \pm 0.7 *#	34.4 \pm 2.4 #
T _{mu} (CONT) (°C)	32.2 \pm 1.4	33.8 \pm 1.3 #	34.0 \pm 1.3 #	34.0 \pm 1.3 #	34.1 \pm 1.7
T _{skin} (HEAT) (°C) ^	29.7 \pm 1.6	39.2 \pm 1.3 *#	39.1 \pm 1.9 *#	39.5 \pm 1.2 *#	34.4 \pm 0.7 *#
T _{skin} (CONT) (°C)	30.3 \pm 1.7	32.6 \pm 1.4	33.2 \pm 1.2	33.3 \pm 1.2	33.3 \pm 1.3
T _{tymp} (°C)	36.4 \pm 0.5	36.6 \pm 0.4	36.6 \pm 0.4	36.6 \pm 0.4	36.6 \pm 0.4
MAP (mmHg)	97 \pm 12	92 \pm 9	92 \pm 11	89 \pm 10	94 \pm 16
RPE (HEAT) ^	14 \pm 1 *	15 \pm 1 *	15 \pm 1 *#	16 \pm 1 *#	15 \pm 2 *#
RPE (CONT)	14 \pm 1	15 \pm 2 #	16 \pm 2 #	16 \pm 2 #	16 \pm 2 #
Thermal sensation	3.8 \pm 0.7	4.9 \pm 0.5 #	5.0 \pm 0.6 #	5.1 \pm 0.7 #	4.3 \pm 0.7

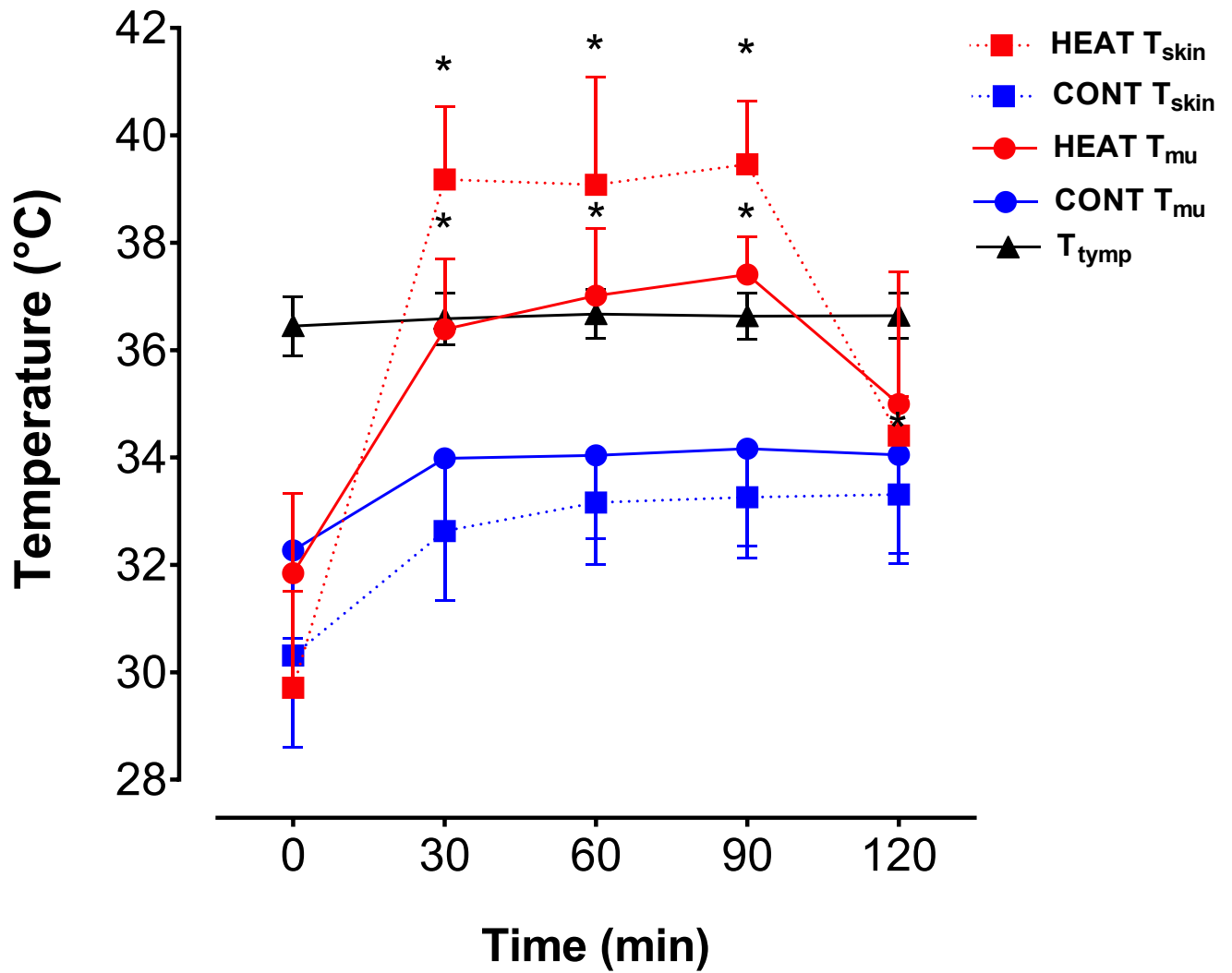


Figure 5. 2. Mean \pm SD Change in muscle temperature (T_{mu} , circles), thigh skin temperature (T_{skin} , squares) and tympanic temperature (T_{tymp} , triangles) during the passive thigh heating protocol (T_{tymp} and T_{skin} $n = 29$, $T_{mu} = 13$). * denotes significant difference between HEAT (red) and CONT (blue) at corresponding timepoint ($p < 0.05$).

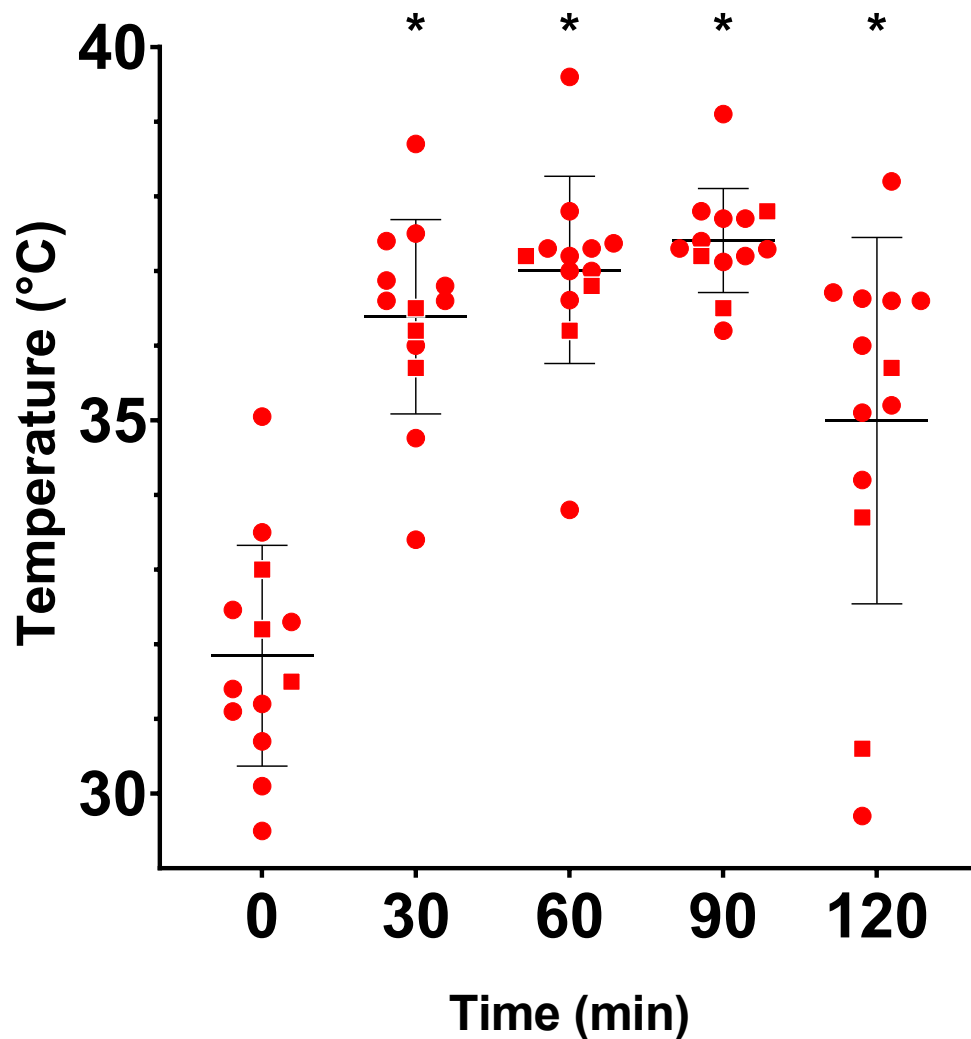


Figure 5. 3. Mean \pm SD (black lines) with individual muscle temperature responses in YOUNGER (circles n = 10) and OLDER (squares n = 3) in response to the 90 min passive thigh heating protocol and 30 min post heating

5.4.3 Torque production following upper thigh muscle hyperthermia

YOUNGER produced significantly more force than OLDER at every isokinetic contractile speed ($p < .001$). When comparing the effect of heating on peak torque production, force production at $60^\circ/s$ differed only between condition ($F(1,36) = 13.3$, $p = < .001$, $\eta^2 = .27$) with the HEAT leg producing $+12 \pm 21$ N.m. (+9%) more force. Differences were seen at $180^\circ/s$ when observing the main effect of time ($F(1,144) = 5.7$, $p = .004$, $\eta^2 = .138$) with an increase of 6 ± 11 N.m. seen at 30 and 60 min. When interpreting the time*condition*group interaction ($F(4,144) = 3.1$, $p = .018$, $\eta^2 = .079$), YOUNGER produced a significant $+11 \pm 17$ N.m (+8%) increase in peak torque from baseline in the HEAT vs CONT in all timepoints past baseline

whilst the OLDER saw a non-significant $+ 5 \pm 16$ N.m (+7%) change from baseline after 30 min, $+ 3 \pm 17$ N.m (+5%) at 60 min, $+ 2 \pm 15$ N.m (+3%) at 90 min and $+ 1 \pm 19$ N.m (+2%) at 120 min. Differences were seen at 300°/s, when observing the main effect of condition ($F(1,36) = 5.9$, $p = .021$, $\eta^2 = .14$) a significant increase in HEAT ($+ 4 \pm 9$ N.m) was seen. An interaction effect of condition*time ($F(4,144) = 2.8$, $p = .015$, $\eta^2 = .09$) was observed, and whilst no difference was observed between conditions at baseline, significant differences were seen in HEAT at 30 min ($+ 7 \pm 2$ N.m; +8%), at 60 min ($+ 7 \pm 2$ N.m; +8%), and at 90 min ($+ 6 \pm 2$ N.m; +7%) when compared to CONT. No significant difference between conditions in the decay measure at 120 min. HEAT significantly increased from baseline in YOUNGER at 30, 60 and 90 min by 8 ± 14 N.m (+10%) no significant changes occurred in CONT from baseline whilst the OLDER saw non significant $+ 5 \pm 12$ N.m (+10%) change from baseline after 30 min, $+ 7 \pm 14$ N.m (+13%) at 60 min, $+ 6 \pm 13$ N.m (+12%) at 90 min and $+ 2 \pm 16$ N.m (+4%) at 120 min. Peak velocity during the isotonic contractions at 25% MVIC had no main or interaction effects, results were not different between condition at any timepoint and did not differ between YOUNGER and OLDER ($p > 0.05$). Full post hoc comparisons can be found in table 5. 2. and figure 5. 4.

5.4.4 Rate of force development, early stage force production and surface EMG following upper thigh muscle hyperthermia

When comparing the effect of heating on RFD₅₀, rate of torque development during 60°/s contractions differed between condition ($F(1,34) = 5.1$, $p = .03$, $\eta^2 = .60$) whereby the heated leg produced 82 ± 120 N.m.s⁻¹. Differences were seen for the main effect of time ($F(4,136) = 11.1$, $p = <.001$, $\eta^2 = .99$) with an increase in RFD₅₀ observed from baseline throughout the 120 min protocol. Condition*time differed ($F(4,136) = 6.0$, $p = <.001$, $\eta^2 = .96$) whereby HEAT saw an increase from baseline of $+ 229 \pm 210$ N.m.s⁻¹ (+29%) after 30 min, $+ 265 \pm 282$ N.m.s⁻¹ (+33%) after 60 min, $+ 211 \pm 282$ N.m.s⁻¹ (+27%) after 90 min and $+ 223 \pm 306$ N.m.s⁻¹ (+25%) 30 min after heating had ceased. No significant changes were observed in CONT from baseline throughout the protocol. No significant differences were observed at baseline between HEAT or CONT; however, after 30 min of heating HEAT was $+ 99 \pm 348$ N.m.s⁻¹ higher and remained at this difference throughout the protocol.

When comparing the effect of heating on EFP60, torque during 60°/s contractions differed between condition ($F(1,35) = 9.7$, $p = .004$, $\eta^2 = .22$) whereby the heated leg produced $+ 8 \pm 15$ N.m. (8%) more force. Differences were seen for the main effect of time ($F(4,140) = 6.9$, $p = <.001$, $\eta^2 = .15$) with an increase in EFP60 peak torque observed from baseline throughout the 120 min protocol. Condition*time differed ($F(4,140) = 5.6$, $p = <.001$, $\eta^2 = .14$) whereby HEAT saw an increase of $+ 15 \pm 19$ N.m (+15%) at 60 min, a $+ 7 \pm 16$ N.m (+8%) at 90 min and a $+ 10$

± 21 N.m. (+10%) at 120 min when compared to CONT. HEAT also differed from baseline by +12 N.m from 30 min onwards (+12%). CONT saw no changes. An interaction effect of condition*time*group was observed ($F_{(4,140)} = 2.8$, $p = .043$, $\eta^2 = .78$); YOUNGER saw a significant increase in HEAT when compared to CONT of $+10 \pm 26$ N.m (+8%) at 60, 90 and 120 min whilst OLDER saw a significant increase in HEAT when compared to CONT of $+20 \pm 19$ N.m (+21%) at 60 min but not at 90 min ($+ 3 \pm 16$ N.m) and at 120 min ($+10 \pm 21$ N.m). When compared to baseline HEAT in YOUNGER was significantly improved by $+11 \pm 16$ N.m (+8%) at 60 min with OLDER improved by $+20 \pm 17$ N.m (+21%) at 60 min and $+13 \pm 17$ N.m (+15%) at 90 min. CONT increased from baseline by 10 ± 14 N.m (+13%) at 30 and 90 min. Differences were seen at EFP180 when observing condition ($F_{(1,35)} = 8.5$, $p = .006$, $\eta^2 = .20$) where a $+7 \pm x$ N.m (+12%) difference was observed between HEAT and CONT. The main effect of time also differed ($F_{(4,140)} = 3.0$, $p = .039$, $\eta^2 = .08$), a significant increase from baseline was seen at 30 min ($+6 \pm 9$ N.m; +8%). No significant differences were observed between HEAT and CONT. No main or interaction effects were observed upon investigating EMG results. Data were not significantly different between condition at any timepoint and did not differ between YOUNGER and OLDER

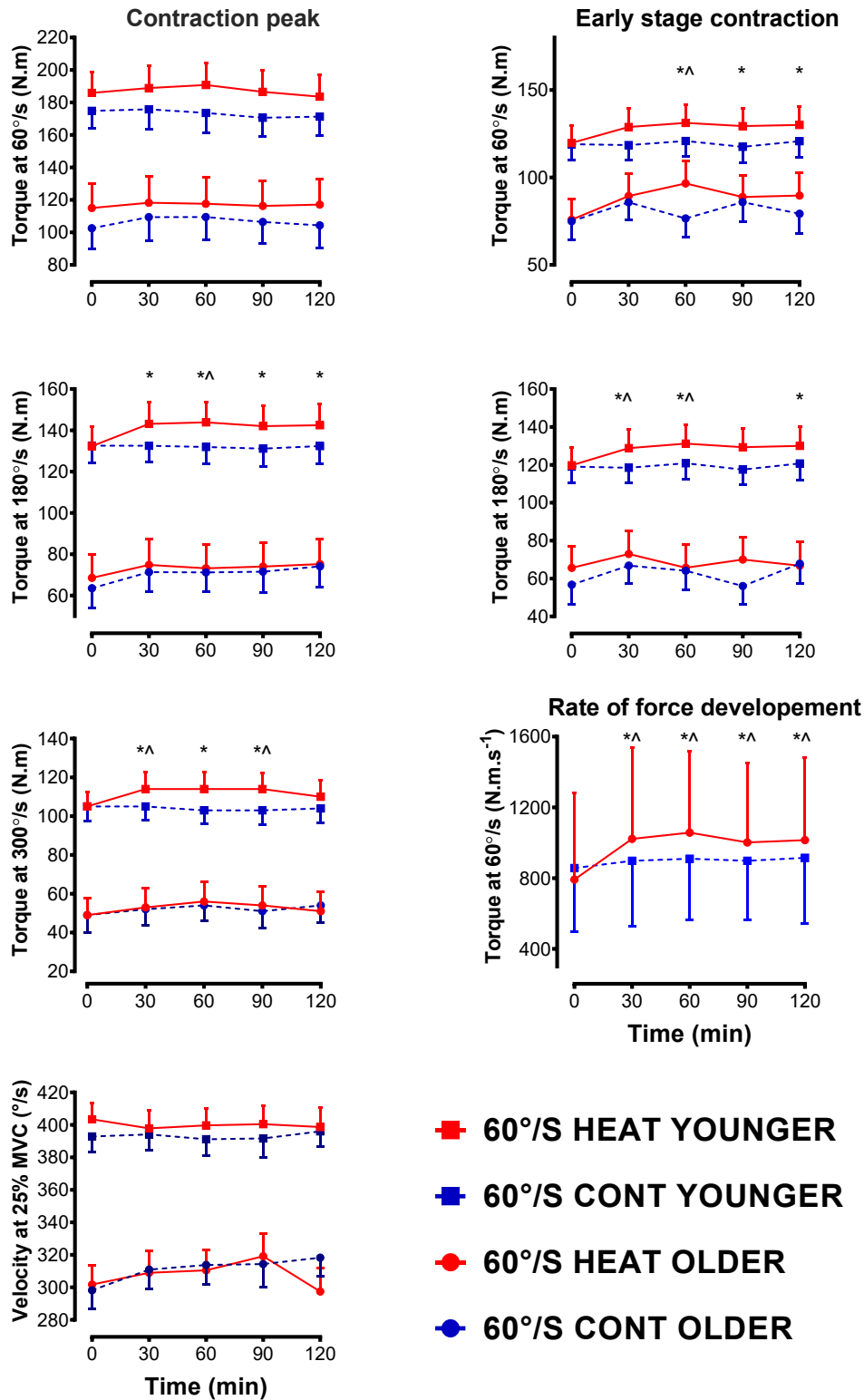


Figure 5. 4. Mean \pm SE Change in peak force production across 60, 180, 300°/s and maximum velocity, early force across 60, 180°/s and rate of force production in 60°/s over 90 min of passive thigh heating and 30 min of passive cooling. * denotes significant difference between HEAT (red) and CONT (blue) at corresponding timepoint ($p < 0.05$). ^ denotes significant difference from baseline ($p < 0.05$).

Table 5. 2.

Mean \pm SD Peak torque and peak velocity generated at 60°/s, 180°/s and 300°/s and vs. 25% MVIC in heated (HEAT) and control (CONT) legs across 90 min of passive thigh heating and 30 min heat decay, YOUNGER (n = 22), OLDER (n = 16). * $p < 0.05$ vs control at corresponding timepoint; # $p < 0.05$ vs baseline (0 min); ^ denotes main effect difference between condition $p < 0.05$, † denotes difference between visit $p < 0.05$

	0 min	30 min	60 min	90 min	120 min
YOUNGER					
Isokinetic Contractions - Peak torque (N.m)					
60°/s (HEAT)	186 \pm 50	189 \pm 54	191 \pm 54	187 \pm 52	184 \pm 53
60°/s (CONT)	175 \pm 43	176 \pm 48	174 \pm 48	171 \pm 45	171 \pm 47
180°/s (HEAT)	132 \pm 38	143 \pm 42#*	144 \pm 39#*	142 \pm 39*	142 \pm 41*
180°/s (CONT)	133 \pm 32	133 \pm 32	132 \pm 32	131 \pm 35	132 \pm 35
300°/s (HEAT)	105 \pm 30	114 \pm 34	114 \pm 34	114 \pm 33	111 \pm 34
300°/s (CONT)	105 \pm 30	105 \pm 28	103 \pm 27	103 \pm 29	104 \pm 30
Isotonic Contractions - Peak velocity (°/s)					
25% MVIC (HEAT) †	403 \pm 52	398 \pm 56	400 \pm 54	400 \pm 56	399 \pm 54
25% MVIC (CONT) †	393 \pm 49	394 \pm 48	391 \pm 49	392 \pm 55	396 \pm 48
OLDER					
Isokinetic Contractions - Peak torque (N.m)					
60°/s (HEAT)	115 \pm 59	118 \pm 63	118 \pm 64	116 \pm 61	117 \pm 63
60°/s (CONT)	103 \pm 50	109 \pm 56	109 \pm 56	106 \pm 52	104 \pm 54
180°/s (HEAT)	69 \pm 44	75 \pm 48	73 \pm 45	74 \pm 45	75 \pm 48
180°/s (CONT)	64 \pm 38	71 \pm 37	71 \pm 37	72 \pm 40	74 \pm 43
300°/s (HEAT)	49 \pm 35	53 \pm 40	56 \pm 40	54 \pm 39	51 \pm 42
300°/s (CONT)	49 \pm 35	52 \pm 33	54 \pm 32	51 \pm 34	54 \pm 35
Isotonic Contractions - Peak velocity (°/s)					
25% MVIC (HEAT)	281 \pm 59	286 \pm 64	287 \pm 62	297 \pm 64	280 \pm 62
25% MVIC (CONT)	282 \pm 55	297 \pm 56	297 \pm 56	297 \pm 62	301 \pm 55
ALL PARTICPANTS					
Isokinetic Contractions - Peak torque (N.m)					
60°/s (HEAT)	150 \pm 53	154 \pm 58	155 \pm 58	152 \pm 56	151 \pm 57
60°/s (CONT)	140 \pm 46	143 \pm 52	142 \pm 52	139 \pm 49	138 \pm 51
180°/s (HEAT)	101 \pm 41	109 \pm 45#*	109 \pm 42*	108 \pm 42*	109 \pm 44
180°/s (CONT)	99 \pm 35	102 \pm 35	102 \pm 34	102 \pm 38	103 \pm 39
300°/s (HEAT)	78 \pm 32	84 \pm 37#*	85 \pm 37#*	84 \pm 36#*	81 \pm 38
300°/s (CONT)	78 \pm 32	79 \pm 30	79 \pm 30	77 \pm 31	79 \pm 32
Isotonic Contractions - Peak velocity (°/s)					
25% MVIC (HEAT)	342 \pm 41	342 \pm 42	344 \pm 41	349 \pm 42	339 \pm 41
25% MVIC (CONT)	337 \pm 41	345 \pm 40	344 \pm 41	344 \pm 42	348 \pm 40

Table 5. 3.

Mean \pm SD early force production (torque at 0.18 s) and early velocity production generated at 60°/s, 180°/s and 300°/s and vs. 25% MVIC in heated (HEAT) and control (CONT) legs across 90 min of passive thigh heating and 30 min heat decay, YOUNGER (n = 22), OLDER (n = 16). * $p < 0.05$ vs control at corresponding timepoint; # $p < 0.05$ vs baseline (0 min); ^ denotes main effect difference between condition $p < 0.05$, † denotes difference between visit $p < 0.05$

	0 min	30 min	60 min	90 min	120 min
YOUNGER					
Isokinetic Contractions - early force production (N.m)					
60°/s (HEAT)	120 \pm 50	129 \pm 54*	131 \pm 52#*	129 \pm 51*	130 \pm 54*
60°/s (CONT)	119 \pm 44	118 \pm 42	121 \pm 44	118 \pm 46	121 \pm 46
180°/s (HEAT)	120 \pm 46	129 \pm 49	131 \pm 50	129 \pm 48	130 \pm 51
180°/s (CONT)	119 \pm 42	118 \pm 38	121 \pm 41	118 \pm 40	121 \pm 43
Isokinetic Contractions - RFD ₅₀					
60°/s (HEAT)	988 \pm 660	1229 \pm 684	1297 \pm 612	1255 \pm 600	1229 \pm 630
60°/s (CONT)	1105 \pm 486	1153 \pm 498	1158 \pm 462	1128 \pm 444	1145 \pm 492
OLDER					
Isokinetic Contractions - early force production (N.m)					
60°/s (HEAT)	76 \pm 57	89 \pm 62	97 \pm 60#*	89 \pm 59#	90 \pm 61
60°/s (CONT)	75 \pm 51	86 \pm 49#	77 \pm 51	86 \pm 53#	79 \pm 53
180°/s (HEAT)	66 \pm 54	73 \pm 57	66 \pm 58	70 \pm 56	67 \pm 59
180°/s (CONT)	57 \pm 49	67 \pm 45	64 \pm 48	56 \pm 47	68 \pm 50
Isokinetic Contractions - RFD ₅₀					
60°/s (HEAT)	597 \pm 738	815 \pm 768	819 \pm 684	750 \pm 672	801 \pm 702
60°/s (CONT)	610 \pm 540	644 \pm 552	664 \pm 516	669 \pm 498	687 \pm 552
ALL PARTICPANTS					
Isokinetic Contractions - early force production (N.m)					
60°/s (HEAT)^	98 \pm 54	109 \pm 58#*	114 \pm 56#*	109 \pm 55#*	110 \pm 58#
60°/s (CONT)	97 \pm 48	102 \pm 34	99 \pm 35	102 \pm 37	100 \pm 37
180°/s (HEAT) ^	93 \pm 50	101 \pm 53#	99 \pm 54#	100 \pm 52#	99 \pm 55#
180°/s (CONT)	88 \pm 46	93 \pm 42	93 \pm 45	87 \pm 44	95 \pm 47
Isokinetic Contractions - RFD ₅₀					
60°/s (HEAT)	792 \pm 492	1022 \pm 516#*	1057 \pm 462#*	1002 \pm 450#*	1015 \pm 468#*
60°/s (CONT)	857 \pm 360	898 \pm 372	910 \pm 348	898 \pm 336	915 \pm 372

Table 5. 4.

Mean \pm SD Surface EMG (mV) measured during knee extensions at 60°/s, 180°/s and 300°/s and vs. 25% MVIC in heated (HEAT) and control (CONT) legs across 90 min of passive thigh heating and 30 min heat decay, YOUNGER (n = 16), OLDER (n = 7).

	0 min	30 min	60 min	90 min	120 min
YOUNGER					
Isokinetic contractions					
60°/s (HEAT)	0.19 \pm 0.10	0.23 \pm 0.16	0.20 \pm 0.13	0.36 \pm 0.13	0.36 \pm 0.15
60°/s (CONT)	0.29 \pm 0.36	0.20 \pm 0.11	0.18 \pm 0.11	0.40 \pm 0.27	0.18 \pm 0.12
180°/s (HEAT)	0.21 \pm 0.12	0.19 \pm 0.12	0.19 \pm 0.11	0.19 \pm 0.09	0.19 \pm 0.11
180°/s (CONT)	0.25 \pm 0.15	0.22 \pm 0.14	0.26 \pm 0.22	0.32 \pm 0.27	0.21 \pm 0.14
300°/s (HEAT)	0.23 \pm 0.14	0.21 \pm 0.13	0.22 \pm 0.16	0.20 \pm 0.10	0.20 \pm 0.10
300°/s (CONT)	0.21 \pm 0.16	0.20 \pm 0.10	0.27 \pm 0.22	0.30 \pm 0.28	0.20 \pm 0.13
Isotonic contractions					
25% MVIC (HEAT)	0.25 \pm 0.14	0.28 \pm 0.26	0.25 \pm 0.23	0.23 \pm 0.10	0.23 \pm 0.16
25% MVIC (CONT)	0.27 \pm 0.17	0.18 \pm 0.10	0.27 \pm 0.18	0.21 \pm 0.15	0.23 \pm 0.17
OLDER					
Isokinetic contractions					
60°/s (HEAT)	0.19 \pm 0.03	0.23 \pm 0.04	0.20 \pm 0.03	0.36 \pm 0.16	0.36 \pm 0.16
60°/s (HEAT)	0.29 \pm 0.09	0.20 \pm 0.03	0.18 \pm 0.03	0.40 \pm 0.03	0.18 \pm 0.12
180°/s (HEAT)	0.21 \pm 0.03	0.19 \pm 0.03	0.19 \pm 0.03	0.19 \pm 0.02	0.19 \pm 0.03
180°/s (CONT)	0.25 \pm 0.06	0.22 \pm 0.04	0.26 \pm 0.08	0.32 \pm 0.12	0.21 \pm 0.03
300°/s (HEAT)	0.23 \pm 0.04	0.21 \pm 0.03	0.22 \pm 0.04	0.20 \pm 0.03	0.20 \pm 0.03
300°/s (CONT)	0.21 \pm 0.04	0.20 \pm 0.03	0.27 \pm 0.06	0.30 \pm 0.07	0.20 \pm 0.03
Isotonic Contractions					
25% MVIC (HEAT)	0.25 \pm 0.04	0.28 \pm 0.07	0.25 \pm 0.04	0.23 \pm 0.03	0.23 \pm 0.04
25% MVIC (CONT)	0.27 \pm 0.07	0.18 \pm 0.03	0.27 \pm 0.04	0.21 \pm 0.04	0.23 \pm 0.04

5.4.5 Perceptual measures following upper thigh muscle hyperthermia

Rating of perceived exertion differed when examining the main effects of condition ($F_{(1,35)} = 12.4$, $p = .001$, $\eta^2 = .26$) and time ($F_{(4,140)} = 18.0$, $p = <.001$, $\eta^2 = .34$) and the interaction effects of condition*group ($F_{(1,35)} = 4.9$, $p = .033$, $\eta^2 = .12$), time*group ($F_{(4,140)} = 5.4$, $p = <.001$, $\eta^2 = .14$) and condition*time*group ($F_{(4,140)} = 2.6$, $p = .043$, $\eta^2 = .07$). In YOUNGER HEAT and CONT saw a significant change from baseline of +2 on the RPE scale from 30 min onwards. OLDER saw no increase from baseline in HEAT or CONT. YOUNGER saw significant differences between HEAT and CONT with HEAT being -1 AU at every timepoint compared to CONT. Thermal sensation differed when the main effect of time ($F_{(4,144)} = 30.8$, $p = <.001$, $\eta^2 = .46$) and time*group ($F_{(4,144)} = 3.6$, $p = .008$, $\eta^2 = .09$) see Table 2. A total of 77% of YOUNGER participants self-reported that heating made them feel at least “a little bit better” in terms of readiness for exercise via the global rate of change scale, 50% self-reported that heating made them feel at least “somewhat better” in terms of readiness for exercise and 35% self-reported that heating made them feel at least “moderately better” in terms of readiness for exercise. The OLDER group response to the global rate of change scale 88% of participants self-reported that heating made them feel at least “a little bit better” in terms of readiness for exercise, 69% self-reported that heating made them feel at least “somewhat better” in terms of readiness for exercise, 56% self-reported that heating made them feel at least “moderately better” in terms of readiness for exercise and 31% self-reported that heating made them feel at least “quite a bit better” in terms of readiness for exercise

5.5 Discussion

This study primarily aimed to assess the effects of 90 min of passive thigh heating on peak torque, early force production, and rate of force development during isokinetic contractions in younger and older adults. Secondary objectives included evaluating the retention of these effects 30 min post-heating and examining age-related differences in perceived impacts of passive heating on muscle function. The passive thigh heating intervention significantly increased muscle temperature in HEAT by +5.3°C, which occurred in the absence of change in any systemic physiological responses in both, YOUNGER and OLDER groups. In conjunction with increased muscle temperature, peak torque increased in HEAT during tests at moderate (+6%) and fast (+8%) contractile speeds when compared to CONT. When exploring between age group differences, at the moderate contractile speed, YOUNGER was the only group significantly changing from baseline by 11%, whilst OLDER saw no significant change at any timepoint. This outcome suggests that the thermally mediated mechanism driving change was not activated/present in older adults. When observing RFD₅₀, combined data from the YOUNGER and OLDER groups saw a 29% increase from baseline in HEAT.

EFP also increased from baseline in HEAT by 9% in the YOUNGER and by 28% in the OLDER groups, suggesting that older adults may have greater capacity to enhance EFP than younger adults. The reported changes peak torques at moderate and fast speeds and early force production were above previously reported minimum detectable change (MDC_{95}) in 65%, 75% and 95% of YOUNGER individuals, with 44%, 38% and 75% of OLDER adults (Denny et al. 2025). Interestingly, while fast contractions improved following HEAT, the fastest/maximal velocity contraction type, i.e., isotonic contractions, saw no change at any timepoint or between conditions. Older adults perceived heating to increase their readiness for exercise more than younger adults, although both considered it beneficial.

5.5.1 Performance responses to passive thigh heating in younger and older adults

The finding of overall positive changes in peak torque in moderate and fast contractions following passive heating aligns with prior experimental findings (Denny et al. 2025). Peak torque in the heated limb within younger adults was significantly enhanced by 8% from baseline after just 30 min of heating and this was sustained throughout the protocol while the CONT limb saw no improvements from baseline, Older adults; however, only reported a non-significant 7% increase after 30 min. These results suggest that while passive heating may offer advantages for peak isokinetic force production in younger individuals, the benefits of passive thigh heating on this outcome specifically may be limited or absent in older adults when contextualised against inferential statistical outcomes. Rate of force development in HEAT saw an increase of 29% from baseline and a difference from CONT of +16% from 30 min onwards. All increases in muscle force production displayed large effect sizes ($>0.14 \eta p^2$), providing confidence in the impact that passive thigh heating had on peak torque and early force production. Beyond 30 min of heating examination of the OLDER outcomes indicates a more compelling absence of benefit given relative change diminishes ($\leq 5\%$ thereafter). Our findings are in broad agreement with prior work examining passive heating have observed rate of force development improvements of +26% at 50 ms (Rodrigues et al. 2021) and +48% at 100 ms (Mornas et al. 2022) in younger adults. The significant improvements in EFP function observed during heating did not decay 30 min following heating cessation highlighting that benefits persist beyond the withdrawal of the heating intervention, at least in the short term. Muscular benefits persisting after heating had ceased may be in part caused by enhanced microcirculatory and microvascular function (Brunt et al. 2016), as well as improved endothelium-dependent vasodilation in skeletal muscle observed following the cessation of heating (Richey et al. 2022). The upper duration for which a positive benefit is observed requires identification. In contrast, the benefits seen in increased peak torque in the moderate and fast contractile speed had diminished 30 min following heating cessation drawing parallels to prior work using active warm ups (Faulkner et al. 2013). The absence of significant

increases in systemic physiological measures, i.e., heart rate, T_{lymp} and MAP suggest the risk of adverse events is unlikely to be increased following the intervention. Rating of perceived exertion scores in HEAT were lower than that in CONT, this may be due to the analgesic effect of heating reducing the negative or painful experiences caused by high force contractions (Chabal et al. 2020). Both younger and older adults display similar muscle temperature heating responses as displayed in Figure 5. 3.

5.5.2 Age related differences in the responses to passive thigh heating

The improvement in peak torque was likely restricted to the YOUNGER group. Peak torque in the heated limb within younger adults was significantly enhanced by 8% from baseline after just 30 min of heating and this was sustained throughout the protocol while the CONT limb saw no improvements from baseline, Older adults however only reported a non-significant 7% increase after 30 min. These results suggest that while passive heating may offer advantages for peak isokinetic force production in younger individuals, the benefits of passive thigh heating on this outcome specifically may be limited or absent in older adults when contextualised against inferential statistical outcomes. Rate of force development in HEAT saw an increase of 29% from baseline and a difference from CONT of +16% from 30 min onwards. Similarly, both YOUNGER and OLDER groups benefited from heating when examining EFP at the slowest isokinetic contraction speed (EFP₆₀). We calculated MDC_{95} for peak isokinetic, EFP and RFD; from this, the peak isokinetic torque changes observed in YOUNGER moderate speed were above the calculated minimal detectable change (+11 N.m, $MDC_{95} = 6$ N.m) whilst the non-significant changes of OLDER were lower than MDC_{95} . The group mean increases in peak isokinetic, EFP and RFD are also above calculated MDC_{95} in moderate and fast contractions (6 and 7 N.m respectively).

Younger adults recorded an 8% increase in EFP₆₀ in HEAT from 60 min onwards whilst the older adults saw a superior 20% increase at 60 min. Prior work (Denny et al., 2025), displayed a 15% increase in EFP₆₀ during passive heating in younger adults, which agree with the outcomes in older adults in the current study meaning that older adults display similar significant improvement to younger adults suggesting a similar mechanism is active in both age groups that is separate from the mechanism that drives increases in peak torque in the younger adults. Importantly EFP during slow contractions were positively affected by heating in both groups. The greater increase of EFP observed in OLDER may suggest a greater sensitivity or potential to respond to temperature regulated EFP₆₀ mechanisms e.g., enhanced nerve conduction velocity. This may also explain why older adults saw increases in the CONT condition as slight muscle temperature increases were also observed. Older adults have displayed a reduced time to muscle activation (Thelen et al., 2000) and therefore may benefit

from the increases in nerve conduction velocity following passive heating, a reported 5% increase in conduction velocity has been observed in healthy younger adults for each 1°C increase in muscle temperature (Kiernan et al., 2001). Further research is required to understand if the changes in conduction velocity observed are present in older adults and if those responses are present during maximal knee extension contractions.

Higher RFD have been positively associated with increased mobility and increased ability to complete daily tasks (Aagaard et al. 1989; Blazevich et al. 2009; Maffiuletti et al. 2016). Whilst muscular weakness is a moderate predictor of fall risk in older adults (Suetta et al. 2004), the ability to reverse a fall and prevent injury is more closely linked to rapid force production (Bellumori et al. 2013). Muscular power has been identified as a key determinant of functional performance, more so than muscular strength for the execution of daily tasks, particularly those requiring rapid and dynamic movements (Bean et al. 2002; Sayers 2008). Functional tasks such as rising from a chair, climbing stairs, and maintaining balance during unexpected perturbations often necessitate the rapid generation of force, rather than merely the ability to sustain or produce maximal force. This distinction underscores the practical importance of muscular power in maintaining independence and reducing fall risk, particularly in older populations (Gray and Paulson 2014). RFD is widely recognised as the main determinant of muscular power, as it reflects the ability of the neuromuscular system to generate force within the critical timeframes required for successful task completion (McLellan et al. 2011). Therefore, the observed benefits in this metric have considerable importance from a translational perspective. In this context, it is imperative to recognise that declines in muscular power with aging are more pronounced than those observed in maximal strength (Macaluso and De Vito 2004) this pronounced decline in muscular power has been termed powerpenia, unlike sarcopenia, which focuses on the loss of muscle mass, powerpenia specifically impairs rapid force production required for tasks such as rising from a chair, climbing stairs, or preventing a fall. It typically precedes declines in muscle strength and mass, making it a sensitive early marker of functional impairment in older adults (Freitas et al. 2024). This disparity highlights the need to prioritise strategies that specifically target power development e.g., increasing muscle temperature, as peak muscular force development alone may fail to address the time-sensitive demands of functional tasks (Freitas et al. 2024).

5.5.3 Underpinning physiological mechanisms in response to passive heating

Current research suggests multiple mechanisms may be responsible for improved muscle function following passive heating including an increase in localised blood flow into and out of the heated muscle (Chiesa et al. 2015; Watanabe et al. 2024), improved myofibrillar calcium handling (Kobayashi et al. 2005), altered muscle-tendon stiffness (Rodrigues et al. 2022),

optimised penetration angle (Eng et al. 2018), and increases in ATP turnover and muscle fibre conduction velocity (Gray et al. 2006). The outcomes of this experiment, specifically the divergent responses between our groups, indicate that these proposed mechanisms underpinning increased peak and early isokinetic torque following passive heating may not be similarly inducible in the muscle of older adults in the way that they appear to be in experiments involving only younger adults. This study did not seek to extensively understand the fundamental mechanisms associated with muscle force enhancement following passive heating, however a notable finding of no change in peak surface EMG may suggest that the observed increases in force production are not due to an increased maximal neural drive at the working muscle (Aagaard et al. 2002) and indicates that an alteration in local muscle structure/function is more likely to be responsible for the change in isokinetic performance (Fitts et al. 1991).

Mechanisms underpinning differential age specific outcomes in response to passive heating require further consideration and may be a reflection of the multiple and complex declines in skeletal muscle anatomy and physiology that occur with advanced age. The theory that an increase in intramuscular fluid both stiffens the muscle and re-orders the angle of the muscle fibres to be more optimal for exercise (Eng et al. 2018), may be less applicable to older adults who generally have higher deposits of intramuscular fat (Pinel et al. 2021), which is compressible, and so the muscle does not deform into the theorised optimum. The older adults within this study had a ~10% higher body fat percentage and whilst intramuscular fat was not measured directly, this points to a greater absolute fat mass and given aging results in fat redistribution, whereby visceral fat infiltrates skeletal muscle (Li et al. 2022) it is likely greater fat deposits reside in this tissue in those with a higher overall body fat %, however, it is important to remember that this was not directly measured in this study. Ageing leads to impairments in the Sarcoplasmic Reticulum Calcium ATPase (SERCA) pump, which then slows the release of calcium into the working muscle (Miljkovic et al. 2015) and the subsequent calcium reuptake (Hunter et al. 1999; Carmeli et al. 2002). In young healthy adults the SERCA pump is stimulated during skeletal muscle hyperthermia (Davies and Young 1983) therefore increasing reuptake of calcium and reducing muscle half relaxation time (Rodrigues et al. 2021). Given the SERCA pump is vulnerable to numerous muscle pathologies associated with general aging (Xu and Van Remmen 2021) this may in turn lead to a blunted ability for stimulation following passive heating, this line of reasoning requires direct research to either confirm or rebut.

Ageing often leads to greater motor neuron function variability (Welsh et al. 2007) and a decrease in nerve conduction velocity (Rivner et al. 2001); however, in this study it is possible

that a similar neural effect was eventually observed in both younger and older adults resulting in the equivalent EFP₆₀ changes across groups. The degradation of the neural pathways may however explain why 60 min of heating was required in the older group whilst only 30 min was required to see significant benefits in the younger group. Whilst this may be a temporal factor, it is worth considering that it may be temperature dependant whereby older adults require higher temperatures to elicit the same outcome. Further mechanistic work examining the influence of incremental muscle temperatures on contractile function across the lifespan is needed. When observing perceptual data, it is worth noting that older adults perceived a greater sense of readiness for exercise following passive heating than younger adults. Whilst it is unclear how this relates to the physiology of muscle function, improving perceptions of readiness for exercise may bolster motivation and confidence which could improve exercise participation and adherence (Lee et al. 2008). The reported enhancement in "readiness for exercise" underscores the potential of localised heating as a preparatory strategy for physical activity that does not undesirably perturb systemic physiology.

5.5.4 Limitations and future directions

While peak muscle activation was not changed in this study, it is not possible to determine the impact of increased nerve conduction rate following passive heating (Todnem et al. 1989) on EFP due to the technological limitations of the equipment used. Time-syncing the EMG sensors to the isokinetic dynamometer would have allowed for an observation of muscle activation during the early stages of force production and should be implemented in future experimental studies. Whilst participants were instructed to maximally contract during the exercise, this was not confirmed by any maximal EMG baselines acquired through external muscle stimulation in this study nor was an assessment of voluntary activation made. It is also acknowledged that muscle temperature measurements were taken without ultrasound guidance; therefore, it cannot be guaranteed that all muscle temperatures were conducted at the same muscle depth between each participant. Previous research on prolonged passive heating reported decreases in muscle temperature in the control condition, likely due to the absence of physical activity (Gibson et al. 2023). However, in this study, muscle temperature in the control condition increased modestly and then plateaued, likely because of the contractions performed. It can be postulated that passive heating benefits could have an even more pronounced effect when compared to completely inactive, normothermic muscles; therefore, we suggest that research investigating true "cold starts" be undertaken. Further research is required to understand the mechanisms behind the observed change in heated muscle in both younger and older groups. While a dedicated familiarisation visit was not included, other research may be strengthened by its inclusion, especially if investigating sedentary or less active older adults who may be unused to leg extension exercise. Research

should also expand into more functional tasks, exploring how heating affects muscle contractions and muscular fatigue over longer time periods than observed in the present study, further research should also investigate how heating may potentially enhance static and dynamic balance in older adults; improved proprioception may reduce the risk of falls. The effect of heat acclimation on muscle function increase following passive heating also requires investigation, the potential interactive mechanisms between heat acclimation and passive heating on neuromuscular performance remain unclear (Tyler et al. 2016), as well as further investigation into how local vs systemic heating affects maximal muscle function. This study is also somewhat limited in its analysis of RFD₅₀ due to the 100 HZ export rate from the dynamometer device. Future research should also endeavour to examine how passive heating may transfer to commonly used functional tests, such as the timed up and go or sit to stand test. The minimum detectable change that was used within this study was calculated using only younger adult's data, as younger adults tend to produce more force; this may be higher than the true MDC for the older adults.

5.6 Conclusion

In summary, passive thigh heating increased muscle temperature by $>5^{\circ}\text{C}$ during the protocol whilst tympanic temperature and other systemic physiology remained unchanged. Peak isokinetic force in younger adults improved during heating at moderate (+8%) and in younger and older during fast (+10%) contractile speeds. Rate of force development during slow isokinetic contractions increased from baseline by 29%. Early force production during slow isokinetic contractions increased from baseline in younger (+13%) and older adults (+28%) during passive heating. Whilst there are observable differences in the responses of younger and older adults to passive heating, in aggregate, heating is a beneficial tool prior to exercise and improves muscle function for younger and older adults.

CHAPTER 6 - Experimental study 3 – Localised passive heating does not improve isokinetic fatigue resistance during repeated and prolonged knee extensor exercise.

6.1 Abstract

Ageing is associated with declines in muscle function, leading to increased fatigability, specifically the ability to resist the reduction of force production during continued exercise. Passive heating has demonstrated positive effects on muscle function during exercise in prior chapters and may extend to alternative forms of exercise. This study examined the effects of passive thigh heating on torque production during repeated and prolonged maximal dynamic isokinetic exercise in healthy older adults. Fifteen healthy older adults (68 ± 8 years) completed a single visit involving 90 min of unilateral thigh heating (50°C) via a custom garment (HEAT), while the contralateral limb served as an unheated control (CONT). Participants performed 30 maximal isokinetic knee extensions at $180^{\circ}/\text{s}$ before and after heating to assess peak torque during a single contraction, and average torque and total work done across the full protocol. Surface electromyography was implemented to monitor muscle activation during the protocol. Peak torque during a single contraction increased by 6 ± 7 N.m in HEAT (from 74 ± 33 N.m to 81 ± 33 N.m, +9%, $p < 0.05$), the control limb saw a no significant change (73 ± 31 N.m to 76 ± 28 N.m). Average torque across the full 30 repetition protocol declined significantly, with declines from the first third (68 ± 30 N.m) to the middle third (57 ± 26 N.m) and from the middle to the final third (51 ± 22 N.m). There was no difference in averaged torque thirds between HEAT and CONT highlighting that fatigue resistance was unaltered. Total work done showed a non-significant 7% increase in the heated limb (from 1619 ± 1100 J to 1751 ± 976 J, $p = 0.273$), a non-significant decrease of -4% was noted in the control limb (from 1589 ± 790 J to 1528 ± 784 J ($p = 0.284$)). Peak EMG amplitude remained unchanged across the protocol. While passive heating enhanced peak torque production, it did not improve fatigue resistance or work done during repetitive dynamic contractions in older adults. These findings indicate that the acute benefits of passive heating, such as increased peak force, may not extend to sustained or repetitive tasks in aged muscle.

6.2 Introduction

Muscular fatigue refers to the decline in a muscle's ability to generate force or power, often resulting from prolonged or intense activity (Sesboüé & Guinestre, 2006). Fatigue is often demonstrated and assessed during exercise as a decline in muscular activation or a reduction or inability to sustain muscular force production (Enoka & Duchateau, 2008). Muscular fatigue is a complex phenomenon, influenced by multiple factors and is commonly categorised as either central or peripheral in origin (Enoka & Duchateau, 2008). In daily living/activity, fatigue is often a combination of concurrent factors resulting in a reduction in the ability to exert force in response to voluntary effort (Ma et al., 2011). Peripheral fatigue results from changes within the muscle itself, including metabolic disturbances and impaired excitation-contraction coupling (Gandevia, 2001). While historically peripheral fatigue was attributed to the accumulation of lactate and a decrease in pH (Gibson & Edwards, 1985), contemporary research has identified additional mechanisms, including ATP depletion (Yin et al., 2021), oxidative stress (Finsterer, 2012), and local inflammation (Sluka & Rasmussen, 2010), as key contributors to muscular fatigue. Central fatigue arises from impairments in the central nervous system, such as reduced motor neuron output or affected by the motivation of the individual (Gandevia et al., 1995). In this chapter, fatigue will be quantified as the reduction in total work done and averaged torque produced during a continuous, repeated maximal strength-based dynamic exercise task (Wu et al., 2022). Additionally, the term 'fatigability' will be used to describe the susceptibility of the working muscle to fatigue.

Skeletal muscle force production and fatigability are highly sensitive to temperature. Increases in muscle temperature have been shown to enhance cross-bridge cycling kinetics, accelerate calcium release and reuptake within the sarcoplasmic reticulum, and increase motor nerve conduction velocity, collectively resulting in improved contractile speed and peak force production (Sargeant, 1987; Rodrigues et al., 2022). These temperature-dependent mechanisms are particularly relevant during dynamic contractions, where force production relies on rapid excitation–contraction coupling and efficient cross-bridge turnover. In addition to these intramuscular effects, elevated muscle temperature increases local blood flow and microvascular perfusion (Koch Esteves et al., 2021), which may influence the development of peripheral fatigue. Enhanced perfusion has the potential to improve oxygen delivery and facilitate the removal of fatigue-related metabolic by-products such as inorganic phosphate and hydrogen ions, which are implicated in reductions in force production during repeated maximal contractions (Kent-Braun, 2009). As such, passive heating represents a plausible intervention through which both peak force output and fatigue-related declines in force production may be altered.

It is well-established that ageing negatively impacts both neuromuscular and mechanical systems, leading to declines in muscle function (Lexell et al., 1988; R. Wu et al., 2020). These musculoskeletal changes often result in reduced performance of daily tasks and impaired quality of life (Brach & VanSwearingen, 2002). Exercise induced muscular fatigue has been observed in a variety of contraction types (Gomes et al., 2021; Kroll, 1973; Westerblad et al., 1998); however, resistance to dynamic fatigue has been identified as an important part of fall prevention in older adults (Rawson, 2010). Increased muscular fatigability, defined as the susceptibility of a muscle to experience a decline in its ability to generate force over time, particularly during sustained or repetitive activity (Hunter, 2018), manifests in declines in walking endurance, balance, and physical activity in healthy older adults (Senefeld et al., 2017). While older adults may exhibit greater fatigue resistance during isometric (Brach & VanSwearingen, 2002) and dynamic tasks (Lanza et al., 2004) compared to younger adults, contradictory findings have also been reported during maximal speed isotonic contractions whereby older adults experienced greater fatigue (Cupido et al., 1992; McNeil & Rice, 2007). The disparity in fatigue resistance between older and younger adults across different contraction types arises from age-related physiological changes and methodological designs (Paris et al., 2022). Older adults often exhibit greater fatigue resistance during isometric tasks, likely due to a higher proportion of type I muscle fibres, which are more fatigue resistant. Conversely, during dynamic or high-velocity contractions, older adults may experience greater fatigue, potentially due to diminished neuromuscular activation and slower muscle contractile properties (Avin & Frey Law, 2011). The increase in fatigue resistance observed in older adults has been attributed to the reduction of type II fibre size (Miljkovic et al., 2015) and the age-related remodelling of motor units that result mostly in denervation of type II muscle fibres and re-innervation of the more fatigue resistant type I muscle fibres (C. Kostek & J. Delmonico, 2011). Older adults are more susceptible to muscular fatigue due to age-related factors such as reduced muscle mass, diminished nerve function, and decreased cardiovascular efficiency, leading to impaired muscle performance and endurance (Santanasto et al., 2015). While it is well established that muscle mass and strength decline with age, it is less commonly recognised that muscular power deteriorates at nearly twice the rate of strength in older adults (Reid & Fielding, 2012), this phenomenon has been named as powerpenia.

Ageing is associated with a range of neuromuscular and metabolic alterations that may modify the muscle's response to acute changes in temperature. Older adults exhibit slower muscle contractile properties, reduced calcium handling efficiency, diminished motor unit firing rates, and impaired microvascular function, all of which contribute to reduced muscle power and increased fatigability during dynamic tasks (Avin & Frey Law, 2011; Reid & Fielding, 2012).

These age-related changes suggest that older muscle may be particularly sensitive to interventions that acutely enhance contractile speed and local perfusion. Furthermore, ageing muscle demonstrates a greater reliance on type I muscle fibres and a reduced reserve of type II fibres, which may limit rapid force generation and power output during high-intensity tasks (Miljkovic et al., 2015). By increasing muscle temperature, passive heating may partially offset age-related slowing of contractile kinetics and improve neuromuscular efficiency, thereby influencing both peak torque production and the rate at which fatigue develops during repeated contractions. Consequently, the physiological effects of heating observed in younger adults cannot be assumed to translate directly to older populations, necessitating targeted investigation in ageing muscle.

Interventions that attenuate age-related declines in muscle function and fatigue resistance in older adults are of significant interest. Older adults often complete daily living tasks near their maximal exercise capacity, and therefore an ergogenic aid may assist in these essential tasks (Hortobágyi et al., 2003). Passive heating of skeletal muscle has emerged as a potential ergogenic aid in this regard, with increased peak torque (see Chapters 4 & 5), maximal power output and increased distance within a 6 min walk (Pellinger et al., 2019; Sargeant, 1987), and early-stage muscle force production (Chapter 4 & 5) (Mornas et al., 2022; Rodrigues et al., 2021) evidenced to date. In addition to temperature-dependent mechanisms that enhance peak force production, such as increased calcium handling, greater muscle-tendon stiffness, faster nerve conduction velocity, and optimized muscle fibre orientation; increased muscle temperature also improves local blood flow (Koch Esteves et al., 2021). This enhanced perfusion may facilitate more efficient removal of metabolic waste and better delivery of energy substrates, thereby contributing to improved exercise tolerance (Rathmacher et al., 2012).

Previous work within this thesis (Chapters 4 & 5) have identified that 90 min is sufficient time when exposed to a heated garment to significantly elevate muscle temperature. Recent work has demonstrated that in clinical cohorts i.e., peripheral artery disease (Pellinger et al., 2019) and heart failure (O'Connor et al., 2025), aerobic exercise tolerance (duration < 10 min) can be increased with acute limb heating. However, the effects on non-clinically compromised older adults performing repeated maximal force contractions for < 60 s is unknown. To date, only one study has investigated the effects of passive increases in muscle temperature and fatigue resistance in younger healthy adults. The study conducted by (Ramanauskiene et al., 2008) found no significant impact on dynamic fatigue resistance during 50 isokinetic contractions at 180°/s when participants underwent either whole-body heating via hot water immersion at 44°C for 45 min or cooling by immersing their legs in 15°C water for 30 min. Whilst these data (Ramanauskiene et al., 2008) are unresponsive of the phenomenon, it

should be noted that assessment of the efficacy of heating interventions to improve local muscle function is problematic when core body temperature increases given this is known to reduce muscular performance through a reduced central drive (Thomas et al., 2006). Therefore in the study by Ramanauskiene et al., (2008) any peripheral benefits local to the exercising limb may have been masked by negative central perturbations. Notably, no studies have examined the effects of localised passive heating on fatigue resistance in older adults, leaving a critical gap in our understanding as to whether this intervention might benefit ageing muscle.

Despite evidence that passive heating enhances peak torque and power output in both young and older adults, its influence on fatigue resistance during short-duration, high-intensity dynamic exercise remains poorly understood. Existing studies have primarily examined whole-body heating protocols, which elevate core temperature and may negatively affect central motor drive, thereby obscuring any peripheral benefits to the exercising muscle (Thomas et al., 2006; Ramanauskiene et al., 2008). Localised passive heating offers a means of selectively increasing muscle temperature while minimising systemic thermal strain, providing a more targeted approach to examining temperature-dependent changes in muscle performance. Importantly, no studies to date have examined whether localised passive heating alters fatigue resistance during repeated maximal contractions in healthy older adults. Given the functional importance of maintaining force output during repeated efforts for daily living tasks and fall prevention, understanding whether passive heating can attenuate fatigue-related declines in force production or total work performed has clear relevance for ageing populations. Addressing this gap is necessary to determine whether passive heating represents a viable ergogenic aid for improving acute muscle performance in older adults.

The aim of this study was therefore to investigate the effects of 90 min of passive thigh heating on fatigue resistance i.e., the change in average torque and total work done of the knee extensor muscles during 30 moderate speed isokinetic extension exercises in healthy older adults. Additionally, the study aimed to assess the impact of passive heating on single contraction peak torque and muscle activation as measured by surface EMG. It was hypothesised that the protocol will result in decreases in force production across the 30 repetitions and that passive heating would improve fatigue resistance, i.e., attenuate the decline in force production, and preserve/increase total work, while having no effect on muscle activation. It was further hypothesised that single contraction peak torque would be increased by the heating intervention.

6.3 Methods

6.3.1 Participant characteristics

Fifteen older adults (eight female, age 68 ± 8 , height 1.65 ± 0.10 m, mass $67.7 \text{ kg} \pm 13.1$, BMI $24.8 \pm 4.4 \text{ kg/m}^2$, body fat 27 ± 4 %, peak isometric force, HEAT 153 ± 82 N, CONT 124 ± 78 N), free of known illness and disease, completed the study. All participants were non-smokers, with no known history of heat intolerance or neuromuscular disorders and provided written informed consent prior to taking part. The study was approved by the Brunel University of London Research Ethics Committee (44577-MHR-Oct/2023- 47671-3) and was conducted in accordance with the Declaration of Helsinki. The participants in this study are a subset of the older adults from Chapter 5 (i.e., 15 of the 16 participants), completing the exercise required for this study in conjunction with data collection for Chapter 5. One participant who completed the protocol associated with Chapter 5 was not eligible for inclusion in this analysis having not completed the post-testing fatigue protocol.

6.3.2 Experimental design

Participants visited the laboratory once at either at 9:00 or 13:00 having abstained from heavy exercise (e.g. resistance or interval training, prolonged endurance activity or competitive sport), caffeinated drinks and supplements, and alcohol for 24 hours prior to the experimental visit. One leg was selected to have the upper thigh heated for 90 min (HEAT) whilst the contralateral limb served as a control (CONT). The participants wore nylon leggings with the experimental thigh (HEAT) wrapped in a custom garment that circulated water at an outlet temperature of 50°C and a survival blanket for a period of 90 min whilst the contralateral control thigh (CONT) was left uncovered. Physiological, perceptual, and functional measurements were assessed every 30 min during the protocol, however for the purposes of this experimental chapter only baseline data and data at 90 min i.e., the end of heating is presented. Figure 6. 1. presents a schematic overview of the study and the place of the measurements in this Chapter relative to those in Chapter 5.

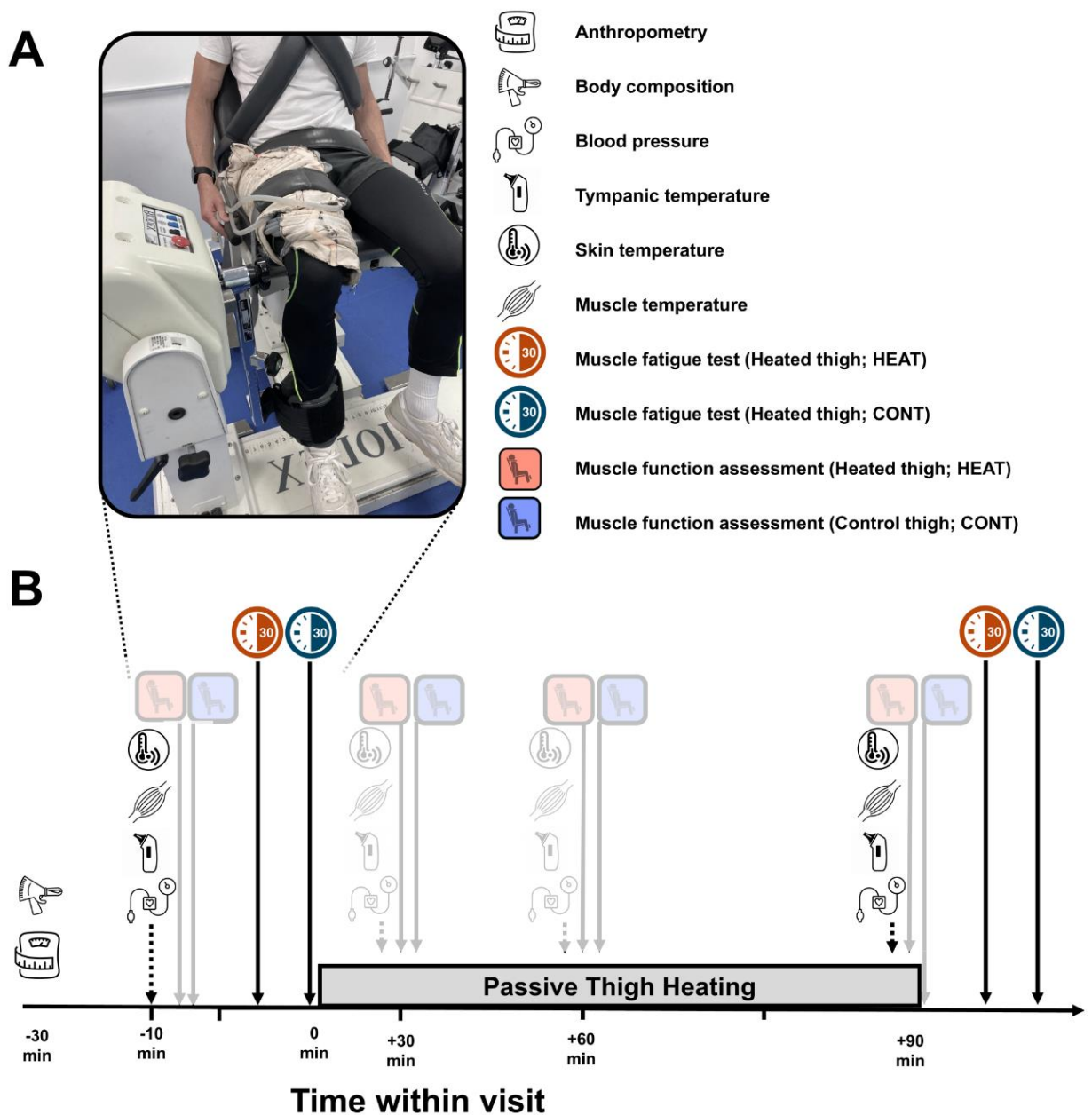


Figure 6. 1. Image of the custom-made water perfused heated garment as affixed on the thigh without the covering of the survival blanket (A). Sequence of the experimental protocols (B).

6.3.3 Physiological measures

The visit began with an assessment of anthropometric characteristics. Unshod standing stature was recorded using a stadiometer (SECA model 213, Hamburg, Germany), with mass was assessed using electronic scales (SECA model 875, Hamburg, Germany). Body fat percentage was calculated with the use of a bioelectrical impedance handheld body composition unit in accordance with manufacturer instructions (Bodystat 1500 touch, Isle of Man, UK). Full participant characteristics are provided in table 6.1. A tympanic membrane

temperature device (Brawn Thermoscan 7, Bussigny, Switzerland) was set to the appropriate age setting and then fully inserted into the right ear canal whereby tympanic temperature (T_{timp}) was recorded as a surrogate for core temperature. Heart rate (HR) and mean arterial pressure (MAP) were measured via an automated sphygmomanometer placed over the left brachial artery (Carescape V100 VitalSigns Monitor, Bolton). Muscle temperature (T_{mu}) was recorded using a muscle temperature probe (RS 103-433 K-type thermocouple, England). The probe was inserted, without local anaesthesia, ~30 mm below the skin surface at a 45° to the horizontal into the vastus lateralis via an 18-gauge hypodermic needle (Microlance 3, Ireland). Muscle temperature was manually recorded following temperature stabilisation (typically ~5 sec) with the probe and guide needle removed thereafter, only three participants volunteered for this assessment and so statistical analysis will not be provided for this measure. Wireless iButton (DS1922L Thermochron Data Logger, UK) sensors were placed over the muscle belly of the vastus lateralis and used to measure thigh skin temperature (T_{skin}) at 60 s intervals. Surface electromyography (EMG) was measured using wireless EMG sensors (Delsys Trigno, Boston, MA USA) “placed at 2/3 on the line from the anterior *spina iliaca superior* to the lateral side of the patella” in accordance with Seniam recommendations (Stegeman & Hermens, 2007). For full explanation for how surface EMG techniques were applied and analysed, please see 3.7 Electromyography data collection and analysis.

6.3.4 Muscle Function assessment

This chapter focuses on the isokinetic fatigue test that was conducted by the older adults during the data collection of Chapter 5. Participants completed isokinetic and isotonic knee extensions as part of the wider experiment as described in chapter 3 (3.6.2 Isokinetic testing) and 5 (5.3.4 Muscle Function and surface electromyography). The 30-repetition isokinetic fatigue test [selected in accordance with best isokinetic fatigue practise (McLeland et al., 2016)] occurred twice, once at baseline (for both the heated limb (HEAT) and the control limb (CONT) and again after 90 min of heating. The fatigue test occurred 3 min following the isokinetic and isotonic muscle function assessments had concluded at both timepoints. The isokinetic fatigue test was conducted using a dynamometer (Biodex Medical Systems, Shirley, NY, USA). An assessment of maximal voluntary isometric contraction (MVIC) was made on both limbs before any muscle function assessments. Participants completed one set of 30 maximal effort repetitions at 180°/s at baseline for both the HEAT and control CONT limb and after 90 min, participants were instructed to “kick as hard and as fast as possible for all 30 repetitions”. At both timepoints HEAT limb was assessed first, followed by CONT.

Peak torque values were assessed as the single highest recorded value for each repetition; the mean peak torque was also calculate for the first, middle and final thirds of the protocol

i.e., across repetitions 1-10, 11-20 and 21-30. Total work (torque x angular displacement) was assessed as the total work done, measured in joules (J), conducted in the knee extension phase of the kicking exercise for all 30 repetitions. The Biodex system 4 (Biodex Medical Systems, Shirley, NY, USA) software was used to collect data at 100 Hz. Torque, position, and velocity data was therefore collected within software every 10 millisecond then exported without filtering and imported to Microsoft Excel for analysis, for a comprehensive explanation of how isokinetic dynamometry was implemented and interpreted for this study please see 3.6 Isokinetic dynamometry. EMG acquisition software (Delsys Discover, Boston, MA USA) was used to collect the raw EMG data from the EMG sensors (Delsys Trigno, Boston, MA USA). The data were plotted within the acquisition software before being exported. Once exported the data were processed within a custom MATLAB code. Raw EMG signals were bandpass filtered with a 6-450Hz cut-off frequency (Gordon et al., 2023), before subtracting the mean of the signal to correct baseline-offsets (see 3.7.2 EMG analysis). The filtered signal then underwent full-wave rectification and low-pass filtering to produce a linear envelope using a dual-pass 2nd order Butterworth filter after which peak amplitude within each set was reported. EMG signals were standardised by expressing repetitions 2–30 as the percentage decrease relative to repetition 1, enabling comparisons across participants and experimental conditions.

6.3.5 Statistical analysis

All data were analysed using SPSS Statistical Software (Version 25, SPSS, Chicago, IL). Analysis of peak torque and peak EMG used a three-way repeated-measures Analysis of Variance (ANOVA) was used to determine main effect differences across timepoints (Two; 0, 90 min), between conditions (Two; HEAT and CONT) and between repetitions (Three torque blocks; First third, Middle third, Final third). A two-way ANOVA was used to determine main effect differences across timepoints (Two; 0, 90 min), between conditions (Two; HEAT and CONT) in T_{skin} and total work done. A paired sample t-test was used to compare differences between timepoint (Two; 0, 90 min) for T_{tymp} , heart rate and MAP. Bonferroni post-hoc adjusted pairwise comparisons were used where significant main effects occurred to identify interaction effects between individual timepoints between conditions. Statistical significance was set at $p < 0.05$, data are reported Mean \pm SD. For a complete description of the use of statistical analysis in this study please see 3.10 Data Reporting.

6.4 Results

6.4.1 Systemic physiology and local heating responses to passive thigh heating

Tympanic temperature demonstrated no significant change from pre-heating (36.2 ± 0.4 °C) to post heating (36.4 ± 0.3 °C). Heart rate also demonstrated no significant difference between pre (69 ± 12 b.min⁻¹) and post (69 ± 10 b.min⁻¹) heating. Mean arterial pressure decreased

from baseline by 9 ± 13 mmHg when measured after 90 min of passive heating ($t_{(15)} = 2.7$, $p = .015$).

Skin temperature differed when observing main effects within Condition ($F_{(1,13)} = 246.8$, $p = <.001$, $\eta p^2 = .95$), Timepoint ($F_{(1,13)} = 202.2$, $p = <.001$, $\eta p^2 = .94$), Condition*Timepoint ($F_{(1,13)} = 102.0$, $p = <.001$, $\eta p^2 = .89$). Post hoc testing revealed that T_{skin} increased from baseline in HEAT by 8.8 ± 2.5 °C. The CONT T_{skin} increased from baseline by 2.6 ± 1.2 °C. A significant interaction effect demonstrated that there was no difference between conditions at baseline (HEAT = 30.5 ± 1.8 °C, CONT = 30.6 ± 1.6 °C), however, that after 90 min of heating significant differences were observed between conditions (HEAT = 39.3 ± 1.3 °C, CONT = 33.2 ± 1.2 °C). Given the small sample number ($n = 3$), T_{mu} are reported for descriptive purposes whereby the HEAT T_{mu} was 32.2 ± 0.8 °C at 0 min, increasing to, 37.2 ± 0.7 °C at 90 min. The CONT muscle temperature was 32.2 ± 1.0 °C at 0 min, and 33.8 ± 0.6 °C at 90 min. The T_{mu} was therefore comparable between CONT and HEAT at baseline, with T_{mu} increasing by ~ 5.0 °C in HEAT and by ~ 1.6 °C in CONT.

Table 6. 1. Systemic physiological and muscular functional responses at baseline and following to 90 mins of passive thigh heating

	Baseline	Post heating
Heart rate (b.min ⁻¹)	69 ± 12	69 ± 10
MAP (mmHg)	102 ± 10	93 ± 12*
T_{skin} (HEAT) (°C) ^	30.5 ± 1.7	39.3 ± 1.2*
T_{skin} (CONT) (°C)	30.6 ± 1.5	33.2 ± 1.1*
T_{mu} (HEAT) (°C)	32.23 ± 0.75	37.17 ± 0.65
T_{mu} (CONT) (°C)	32.17 ± 0.95	33.80 ± 0.61
T_{tymp} (°C)	36.2 ± 0.4	36.4 ± 0.3
Single Contraction Peak Torque HEAT (N.m)	79 ± 39	86 ± 37*
Single Contraction Peak Torque CONT (N.m)	73 ± 31	76 ± 28
Average total work HEAT (J)	1619 ± 1064	1751 ± 943
Average total work CONT (J)	1589 ± 790	1528 ± 784

Values are Mean ± SD (N=15), * denotes significantly different PRE and POST ($p < 0.05$).

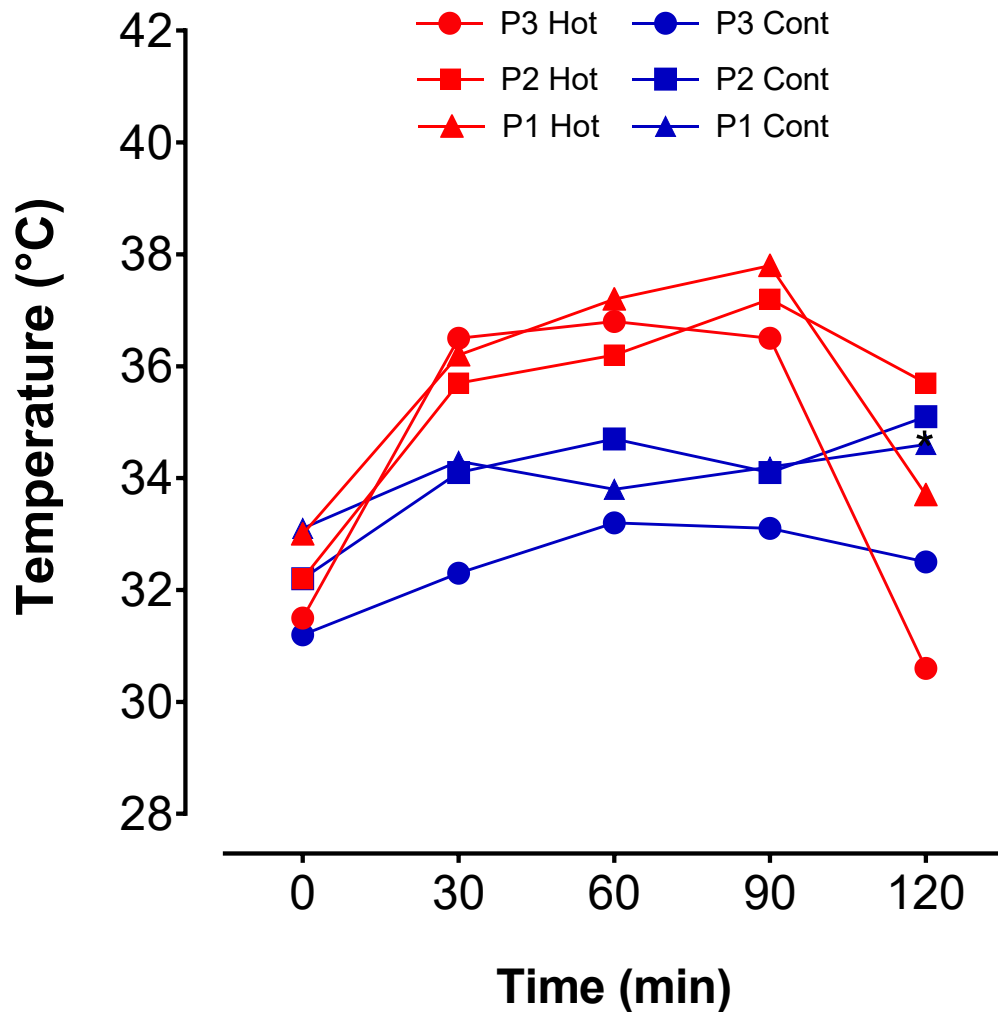


Figure 6. 2. Individual data of older adult muscle temperature of the *vastus lateralis* (n = 3) during 90 min of passive heating and 30 min post heating. HEAT in red, CONT in blue, P1 triangles, P2 square and P3 circles

6.4.2 Averaged torque, total work done and *vastus lateralis* EMG following upper thigh muscle hyperthermia.

As anticipated, the fatiguing task resulted in observable decreases in averaged peak torque production throughout the thirty isokinetic repetitions, see figure 6. 3. When investigating the differences between averaged torque blocks ($F_{(2,28)} = 26.9$, $p = <.001$, $\eta p^2 = .66$) the mean torque within the first third of the fatigue test (68 ± 30 N.m) was significantly higher than the middle third (57 ± 26 N.m) which was significantly higher than the final third (51 ± 22). An interaction effect was observed when examining Timepoint*Thirids ($F_{(2,28)} = 4.4$, $p = .043$, $\eta p^2 = .24$) whereby the averaged torque produced when HEAT and CONT are combined in the

baseline testing during the first third of the fatigue test (64 ± 31 N.m) significantly reduce in the second third (54 ± 27 N.m) and again in the final third (50 ± 24 N.m). The averaged torque produced post heating during the first third of the fatigue test post heating (71 ± 31 N.m) significantly reduce in the second third (59 ± 26 N.m) and again in the final third (52 ± 21 N.m). The effect of heating on average total work done during the fatigue task was not significantly different for HEAT (1685 ± 1015 J) and CONT (1558 ± 809 J) or between pre (1604 ± 953 J) and post (1640 ± 875 J) intervention timepoints. Furthermore, no significant differences were observed in HEAT (pre = 1619 ± 1100 J post = 1751 ± 976 J; change = $+133 \pm 449$ J (+7%)) or CONT (pre = 1589 ± 817 J, post = 1528 ± 813 J, change = -61 ± 213 J (-4%)); $p = .12$, $\eta^2 = .16$, Figure 6. 4.). When observing the peak EMG of the *vastus lateralis* during the fatigue test no significant differences were observed between the two conditions or timepoints or across blocks of contractions (Figure 6. 5.). As such, EMG magnitudes were statistically unchanged (first third, -1.99 ± 2.81 % Δ ; second third, -3.34 ± 7.35 % Δ ; final third, -3.38 ± 8.53 % Δ ; $p = .075$). Furthermore, there were no interaction effects.

Table 6. 2. Torque production and surface EMG during a 30-repetition fatigue task at baseline and following 90 mins of passive thigh heating

	First third Contractions 1 – 10	Second third Contractions 11 - 20	Final third Contractions 21 - 30
Baseline			
Averaged Torque CONT (N.m)	63 ± 29	52 ± 25	49 ± 22
Averaged Torque HEAT (N.m)	66 ± 31	56 ± 27	50 ± 24
EMG amplitude CONT (%Δ)	-0.05 ± 0.07	-1.29 ± 1.06	-1.98 ± 1.85
EMG amplitude HEAT (%Δ)	-0.78 ± 0.90	-3.32 ± 4.48	-4.27 ± 7.60
Post heating			
Averaged Torque CONT (N.m)	66 ± 29	56 ± 25*	50 ± 20*
Averaged Torque HEAT (N.m)	77 ± 33	63 ± 28*	55 ± 24*
EMG amplitude CONT (%Δ)	-0.60 ± 1.32	-3.58 ± 4.96	-8.34 ± 17.81
EMG amplitude HEAT (%Δ)	-1.09 ± 1.67	-1.85% ± 1.72	-1.19 ± 0.25

Values are Mean ± SD (n = 15), * denotes significantly different PRE and POST ($p < 0.05$).

6.4.3 Peak torque production following upper thigh muscle hyperthermia.

When observing the effect of heating on peak torque production a main effect of time was revealed ($F_{(1,14)} = 13.5$, $p = .003$, $\eta p^2 = .49$) whereby a $+6 \pm 7$ N.m improvement was observed post intervention HEAT measurement (81 ± 33 N.m) when compared to pre-intervention measurement timepoint (74 ± 33 N.m). No other significant main or interaction effects were observed.

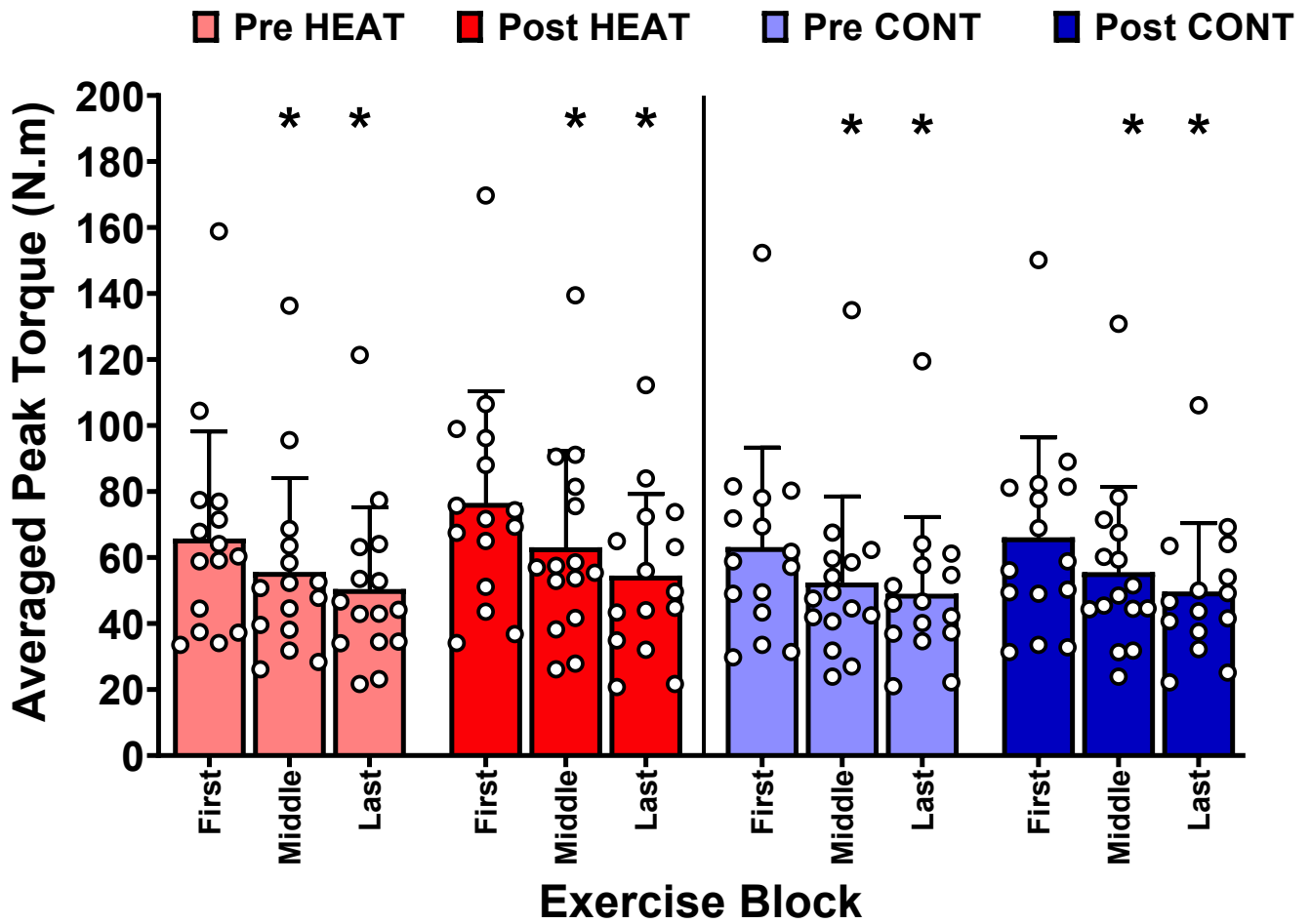


Figure 6. 3. Torque production (N.m) during the thirty-repetition isokinetic fatigue task separated into ten repetition blocks in the pre HEAT (light red bars), post HEAT (dark red bars), pre CONT (light blue bars) and post CONT (dark blue bars), individual data points are open circles. Data are presented as mean \pm SD. * denotes significant difference to prior exercise block

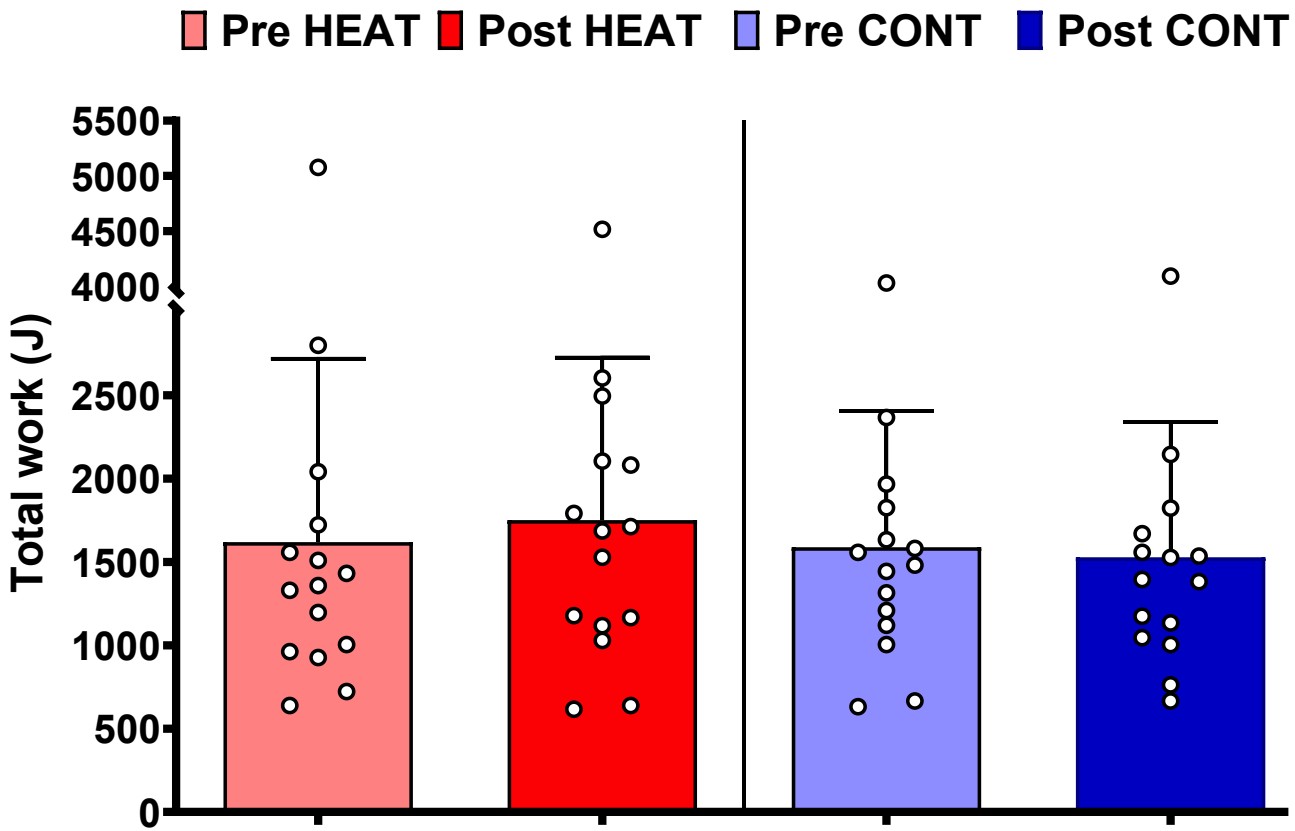


Figure 6. 4. Total work done (J) during the thirty-repetition isokinetic fatigue task separated into ten repetition blocks in the pre HEAT (light red bars), post HEAT (dark red bars), pre CONT (light blue bars) and post CONT (dark blue bars), individual data points are open circles (n = 15). Data are presented as mean \pm SD.

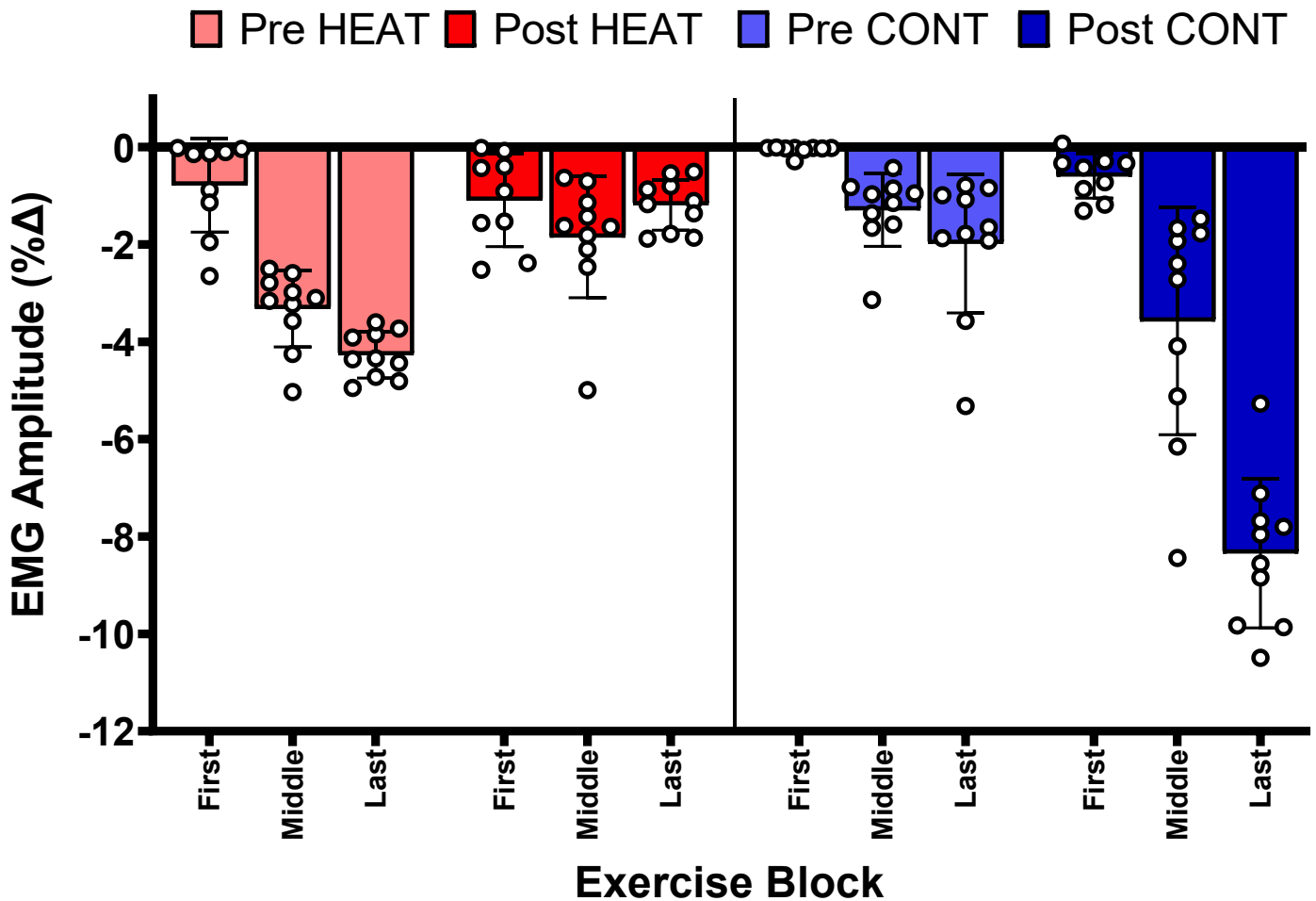


Figure 6. 5. Surface EMG measurements (n = 10 due to technical issues) averaged into thirds during 30 moderate speed isokinetic knee extensions in the pre HEAT (light red bars), post HEAT (dark red bars), pre CONT (light blue bars) and post CONT (dark blue bars). Data are presented as mean \pm SD

6.5 Discussion

This study investigated the effects of passive thigh heating on the fatigue resistance of knee extensor muscles during isokinetic exercise in healthy older adults. The fatiguing protocol employed within this study demonstrated a fatiguing effect on the working muscle, as was hypothesised and observed in similar studies (Gomes et al., 2021; Rawson, 2010). The results indicated that 90 min of passive thigh heating increased the skin and muscle temperature of the heated limb, however fatigue resistance and the attenuation of force decline were not observed. A non-significant +7% increase in total work done was observed in HEAT whilst a -4% change was observed in CONT following 90 min of heating, post hoc power analysis provided a required sample size of 14 to observe a 2 way interaction effect (with an effect size of 0.163 and a correlation among measures of 0.5.) suggesting that it may be adequately

powered to observe a significant change. In keeping with previous experimental chapters in this thesis, a significant increase in peak muscle force (torque produced during moderate isokinetic contractions) was observed.

6.5.1 Muscular fatigue resistance following passive thigh heating

The absence of statistically significant improvements in fatigue resistance following localised passive heating aligns with the findings of Ramanauskiene et al, (2008b) where whole-body heating, which increased muscle temperature to $39.5 \pm 0.2^{\circ}\text{C}$, did not affect dynamic fatigue resistance in younger adults but did result in higher peak force output, similar to the current study. Since both modalities demonstrated no change in fatigue resistance, this suggests that heating, whether local or whole body does not impact fatiguing exercise in both younger and older adults. Whilst fatigue resistance was unchanged statistically, an increase in peak torque was observed in both the HEAT and CONT condition alongside increases in skin temperature. Passive heating was observed to benefit maximal 6 min walking performance in clinical populations (O'Connor et al., 2025; Pellingier et al., 2019) the lack of observed improvement in this study may be attributed to passive heating being more effective in clinical populations than in healthy older adults. Clinical groups often present with lower baseline function or specific pathologies, such as peripheral artery disease, that are more directly influenced by the physiological effects of passive heating, particularly the increase in local blood flow. Additionally, differences in outcomes may stem from the nature of the task, as walking involves repeated submaximal contractions over a limited range of motion, whereas the present study employed maximal, large-range isokinetic knee extensions. Future research should explore whether clinical populations exhibit enhanced performance benefits during maximal isokinetic fatigue protocols following passive heating.

Localised heating of skeletal muscle has been speculated to facilitate calcium handling within the muscle fibre and at the sarcoplasmic reticulum (Rodrigues et al., 2022), which may enhance muscle contractile properties (Kobayashi et al., 2005; Rodrigues et al., 2022). However, the lack of observed improvements in fatigue resistance during this experiment suggest that if this mechanism is present following an acute exposure to passive heating it does not provide benefit to this type of exercise test or was not enough of a muscle force enhancement to offset the progressive onset of fatigue within this 30 repetition task. Metabolic accumulation hydrogen ions and inorganic phosphate; generated from anaerobic respiration within the muscle fibre, have been suggested to be a driver of fatigue within older adults during a isokinetic 30 repetition knee extension task (Kent-Braun, 2009). It was theorised that an increased local blood flow, as seen in prior passive heating work (Koch Esteves et al., 2021; Pearson, 2010), would aid in the removal of metabolic byproducts as seen in low intensity

active recovery (Zhang et al., 2025) and improve fatigue resistance. However, the benefits of increased blood flow may occur over a longer time period than during maximal exercise, where blood flow may be occluded during peak contraction. The findings of this study underscore the complexity and nuances of applying passive thigh heating as an ergogenic aid in older adults. While prior work has highlighted improvements in acute muscle function such as early force production in young (Mornas et al., 2022; Rodrigues et al., 2021) and older adults (CHAPTER 5 - **Experimental study 2 – Passive thigh heating improves peak force production in younger adults and early isokinetic force production in younger and older adults**), and peak torque in young cohorts (CHAPTER 4 - **Experimental study 1 – Passive thigh heating improves isokinetic but not isotonic muscle function**), these benefits may not translate to tasks requiring sustained or repetitive contractions in older adults of <60 s in the way that aerobic endurance activity (5 – 10 min) is following limb heating in clinical cohorts (Pellinger et al., 2019).

6.5.2 Surface EMG measurements and changes in muscle activation

The reduction in central neural drive is often measured and assessed through observed decreases in signal amplitude or frequency of surface electromyography (EMG) where the electrical potential throughout the muscle is recorded. EMG signal has been used as a tool to assess and differentiate peripheral and central fatigue. Two physiological factors are commonly assumed to determine the myoelectric factors of fatigue, a decrease of the conduction velocity of muscle motor units (peripheral fatigue), and the increase of motor unit synchronisation by the central nervous system (central fatigue) (Mesin et al., 2009). Surface EMG can be observed as decreasing in parallel with force magnitude as the muscle fatigues; however, increases in amplitude can be observed during peripheral fatigue as additional motor units are recruited to aid in force production, which is referred to as “cross talk” when analysing EMG data (Berchicci et al., 2013). Neural drive to the muscle is a key component of effective muscle function, with higher amplitude surface EMG recordings corresponding to increased force production (Alkner et al., 2000). Decreased amplitude in surface EMG readings are expected during fatiguing exercise (Pincivero et al., 2006) and this phenomenon is sometimes used as a determinant of local muscular fatigue (Cifrek et al., 2009). Whilst a statistically significant difference was not observed between the thirds, a decreasing trend in peak EMG was observable. Based on the work of others (Hsu et al., 2023) this suggests that central fatigue was a contributing factor to the observed reduction in force production as the task progressed. The recorded EMG values did not change between the HEAT or the CONT group

demonstrating that this passive heating protocol did not reduce or increase neural drive to the working muscle, the non-significance observed between exercise blocks in this study likely results from an underpowered analysis derived from the small sample size of 10, post hoc power analysis suggested a sample size of 12 for EMG measures. Whole body heating which results in increases in core temperature have demonstrated decreases in neural drive through decreased peak EMG amplitude during maximal isokinetic and isometric exercise (Racinais, 2013; Thomas et al., 2006). Prior work investigating localised passive heating has not demonstrated any reductions in EMG amplitude similar to that observed in whole body heating (Rodrigues et al., 2021).

6.5.3 Limitations and future directions

This study had several limitations that may have influenced the outcomes. This current study included additional exercise between the two fatigue testing sets, this altered the muscle temperature (and likely the metabolic state), especially in the CONT limb, which prevents a direct comparison between a truly rested heated limb and a truly rested thermoneutral limb. The scope of the surface EMG analysis within this study was not designed to assess or differentiate central or peripheral fatigue across all active muscles during knee extensor activity with analysis of frequency and amplitude absent it was designed to provide a simple view of muscle activation between a heated and thermoneutral control limb. Future work should therefore seek to investigate the mechanisms of fatigue during maximal exercise prior to and following local but not systemic hyperthermia. The lack of synchronicity between the EMG and dynamometry restricted the scope of analysis, additionally the absence of an evoked twitch precludes confirmation of a true maximal reference against which voluntary contractions can be compared. This study did not take any direct measure of sarcopenia, powerpenia or frailty; the physiology of an older adult who is pre-sarcopenia and one who has severe sarcopenia may not be able to be accurately compared, limiting how applicable these results may be to older adults as a larger group. This study likely did not have any older adults who were severely sarcopenic as all were able to independently commute to the laboratory for testing, those who struggle with independent living and would benefit from improvements to muscle function were likely not tested within this study, further research may wish to include older adults who are less physically active or have trouble living unassisted, as they stand to benefit from an ergogenic tool to reach optimum muscle function before conducting daily living tasks. Future research should also expand on the clinical applications in the possibility of passive heating to improve total work done in sustained efforts (Pellinger et al., 2019). A mechanistic study that improves knowledge around known and unknown mechanisms influencing muscle function following heating would add greatly to the field. This might include analysis of blood flow, calcium concentrations or muscle-tendon mechanics following localised

exposure to passive heating. With aging, increased fatigability due to muscle atrophy, neuromuscular decline, and reduced metabolic capacity leads to impaired daily function and greater fall risk during prolonged daily living tasks such as climbing stairs, sustaining balance when reaching or carrying objects between locations. Therefore, improving fatigability is essential for preserving independence and reducing adverse health outcomes in older adults; and whilst this is primarily achieved through targeted exercise (Reid & Fielding, 2012) a tool that increases acute fatigue resistance could provide immediate assistance and further research should investigate if passive heating can assist in real life living tasks.

6.6 Conclusion

In conclusion, whilst increases in peak torque were observed, passive thigh heating did not enhance fatigue resistance during a thirty-repetition maximal isokinetic knee extension exercise in healthy older adults. These findings suggest that the acute benefits of passive heating on peak torque production observed in young adults may not extend to repeated dynamic contractions in aged muscle.

CHAPTER 7 - General Discussion

This general discussion will summarise the key and novel findings of the experimental chapters of this thesis and collate the common themes relating to the use of localised passive heating as an ergogenic aid to acutely improve muscle function during dynamic exercise. The general discussion will then consider the practical applications of passive heating as a tool and propose areas of the research field that warrant further investigation.

7.1 Summary of Key Findings

Chapter 4 measured the inter and intraday reliability of systemic physiological, local temperature, isokinetic force production and isotonic velocity production in response to 90 mins of heating in healthy young adults (23 ± 2 years old). This study evaluated good to excellent reliability of these measures which allowed for the calculation of the minimal detectable change for the variables. The quantification of the variance within these measures taken during exposure to passive heating provides the first look at the true magnitude of the change that passive heating is affecting in dynamic muscle function. The passive heating protocol displayed high inter and intraday reliability, $ICC > 0.90$, and low variability, $CV < 10\%$, for all isokinetic muscle function assessments. This data showed heat reliably increased muscle temperature from baseline ($32.2 \pm 1.1^{\circ}\text{C}$) after 30 min ($36.8 \pm 0.7^{\circ}\text{C}$) with further $0.4 \pm 1.3^{\circ}\text{C}$ increases in the following 30 min periods ($ICC = 0.74$). All isokinetic and isotonic muscle function assessments were considered to have excellent inter day reliability ($ICC > 0.90$) and low variability ($CV < 10\%$). This study also observed the effect of 90 mins of localised passive heating on isokinetic muscle function across a variety of contractile speeds and isotonic function at 25% MVIC, heating increased peak torque during moderate (+8%) and fast isokinetic contractions (+10%) from 30 mins onwards relative to the unchanged control leg. The slowest contractile speed saw no significant changes to peak torque; however, when observing earlier parts of the contraction, force at 0.18s and rate of force development at 50ms the heated limb saw improvements of 14% and 15% respectively in the heated leg whilst the control leg was unchanged. The isotonic contractions saw no significant change in either condition, the systemic physiology e.g. heart rate, blood pressure or tympanic temperature was unchanged also. This work provided valuable insights into the reliability of the heating responses and allowed for the calculation of the minimal detectable change to confidently express the efficacy of passive heating as a tool to improve isokinetic force production.

Chapter 6 expanded upon the scope of experimental study 1 by using the same methodology with healthy older adults and comparing the effects of localised passive heating on isokinetic and isotonic muscle function between younger (23 ± 3 y) and older (68 ± 8 y) adults over 90

mins with an additional measure 30 mins post heating. This study included the same systemic physiological and local temperature measures as Chapter 4 with the inclusion of surface EMG. Heating increased muscle temperature from baseline ($31.7 \pm 1.7^{\circ}\text{C}$) at 30 min ($36.5 \pm 1.5^{\circ}\text{C}$), 60 min ($37.1 \pm 1.4^{\circ}\text{C}$), 90 min ($37.5 \pm 0.7^{\circ}\text{C}$) and at 120 min ($35.5 \pm 2.3^{\circ}\text{C}$). This study found that heating increased peak torque during moderate contractions by 6% and fast contractions by 8% in only the younger adult participants relative to their control leg which remained unchanged whilst older adults saw no improvements in peak torque at any isokinetic speed. When observing force produced within the early contraction, early force production saw peak increases of 9% in younger and peak increases of 28% within the older adults during the slow isokinetic contractions and the rate of force development at 50ms development was increased from baseline in the heated condition by when the younger and older adult data was combined and saw peak increases of 29%; however both groups saw similar increases, suggesting this study was underpowered to properly discern a between group interaction; post hoc power analysis provided a required sample size of 52 to observe a 3 way interaction effect (with and effect size of 0.153 and a correlation among measures of 0.5. when increasing the correlation among measures to 0.8 the required sample size was 22). This study displayed how after 30 mins post heating ergogenic benefits were lost in older adults but preserved in younger adults. Isotonic, surface EMG and systemic physiology was unchanged. This study provided an interesting comparison between younger and older adults and how heating did/did not affect isokinetic force production. This study also suggests that the benefits in peak torque and the enhancements to RFD and EFP are driven by separate mechanisms as the older adults did not see any improvement in peak torque but large increases in EFP.

Experimental study 3 (CHAPTER 6 - **Experimental study 3 – Localised passive heating does not improve isokinetic fatigue resistance during repeated and prolonged knee extensor exercise.**) was collected during data collection of experimental study 2 and it aimed to examine whether localised passive heating reduces muscular fatiguability during a moderate speed 30 isokinetic repetition task in older adults. This study demonstrated no significant change in torque profile during the fatiguability task. A non-significant, increase in total work was observed, which may suggest this work was underpowered to observe such an effect. An increase in peak torque was observed in both limbs after heating, this increase in force production was observed alongside an increase in limb skin and muscle temperature. Surface EMG was once again unaffected, further suggesting that localised heating does not interact with neural drive to the working muscle during prolonged bouts of maximal contractions. This study highlighted the potential limitations of passive heating as a tool, whilst previous increases in peak force and early force

production had been observed the theoretical increase in fatigue resistance was not observed in our older population. Figure 7. 1. summarises the physiological performance outcomes that were observed within all experimental chapters of this thesis.

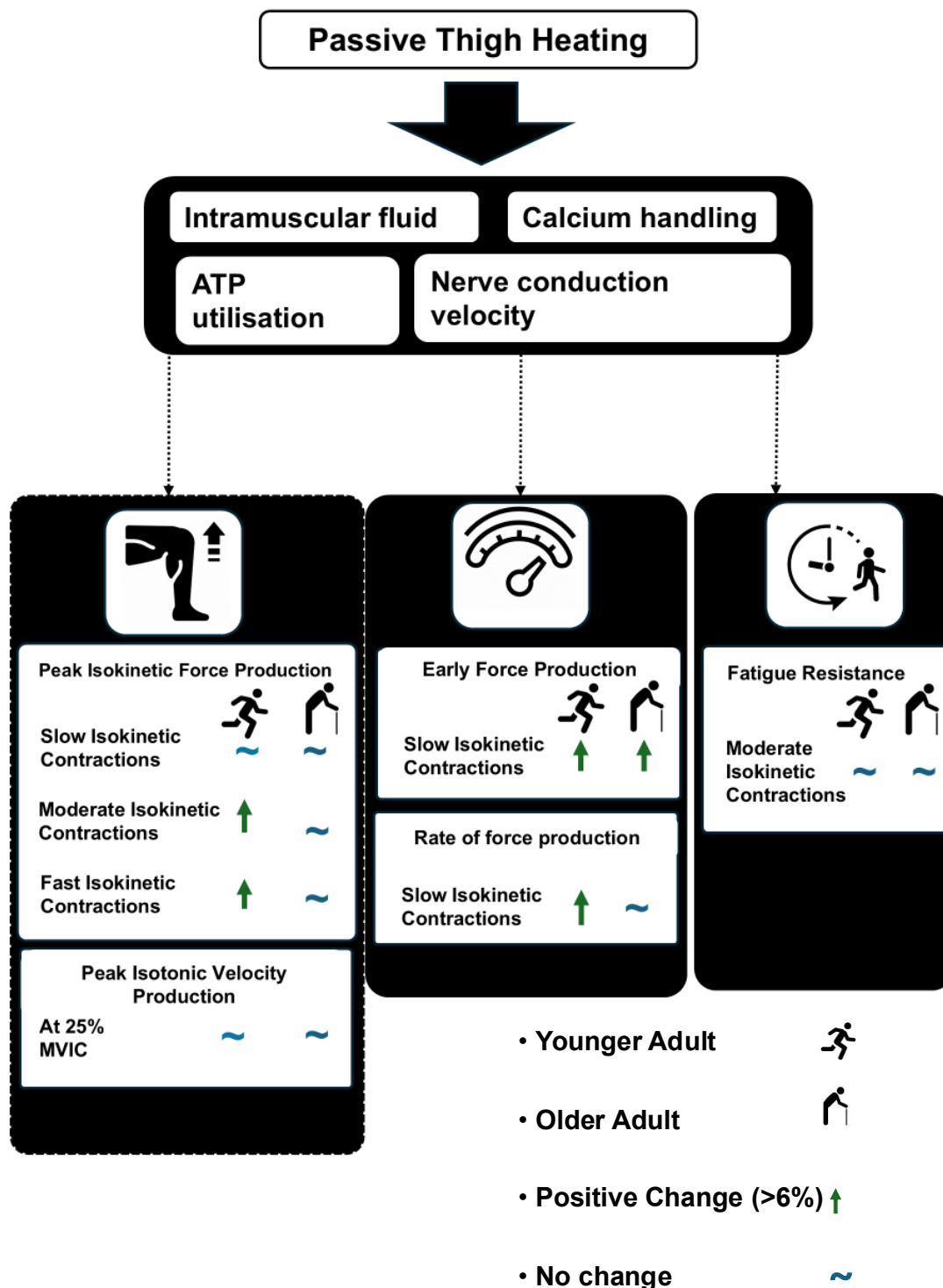


Figure 7. 1. A diagram of the performance outcomes following localised passive heating within this thesis and the suggested mechanisms

7.2 Interpretation of Findings

7.2.1 Heating responses.

This thesis aimed to manipulate muscle temperature to augment muscle function, to this end it was observed that our heating protocol reproducibly increased *vastus lateralis* temperature from baseline ($32.2 \pm 1.1^\circ\text{C}$) to 30 min ($36.8 \pm 0.7^\circ\text{C}$) with further $0.4 \pm 1.3^\circ\text{C}$ increases in the following 30 min periods. It is worthwhile to note that whilst muscle temperature was increased as was skin temperature, some prior research has suggested that skin temperature is the mediating factor in increased isokinetic muscle force production (Cheung & Sleivert, 2004); however, this thesis did not control for this factor e.g., via using heating interventions such as cooling pads or cooling environmental chambers to maintain skin temperature, and is presupposing that muscle temperature is the more important factor as it relates to the proposed mechanisms that underpin increased muscular function following passive heating (Eng et al., 2018; Kobayashi et al., 2005; Racinais et al., 2019; Rodrigues et al., 2022a). The muscle temperature increases seen within this study are consistent with other research conducted using heated garments (Chiesa et al., 2015; Gibson et al., 2023; Ihsan et al., 2020; Koch Esteves et al., 2021) $\sim 38^\circ\text{C}$, similar skin temperatures were also observed within this study when compared to prior research (Kuhlenhoelter et al., 2016) $\sim 40^\circ\text{C}$. The current study reports no ill-effects from the 90 mins of heating when exposed to water circulating the garment at 50°C .

Tissue temperatures above 42°C potentiate protein degradation and may result in cell damage as well as declines in contractile function (Baracos et al., 1984; Essig et al., 1985; Ranatunga, 1984). Prolonged exposure (10+ min) to heat that results in increases in skin temperature of over 42°C are likely to result in tissue damage (Dewhirst et al., 2003), for example burn injuries have been reported in cases where skin temperature $< 43^\circ\text{C}$ (Robins et al., 1989). This concern needs to be considered, especially, when applied in clinical or older adult populations as older adult skin is more susceptible to burns due to age-related changes that impact skin structure and function. These changes include thinning skin, decreased sensation, and impaired judgment and coordination, making them more vulnerable to heat related damage (Jeschke et al., 2015). In the context of muscle temperature manipulation/control, a relevant consideration is the inclusion of 10 submaximal warm up repetitions of the knee extension exercise. Whilst important within research to ensure safety of the participant, this activity increases muscle temperature and may not be truly representative of the resting state of the muscle prior to any exercise or daily living tasks. In addition to the warm up repetitions, this thesis used a methodology whereby 16 maximal knee extensions happened every 30 mins,

this had an observable effect on muscle temperature in the control, normothermic, limb also not giving a true comparison between heated and cold muscle. This may have hidden some of the true difference between limbs as both saw temperature increases from baseline. Future work should aim to test a normothermic and heated limb whilst controlling for muscle temperature, this may involve temperature manipulation or a longer time between familiarisation and testing.

7.2.2 Systemic physiological responses

The localised heating methodology employed within this thesis did not meaningfully impact any of the physiological measures i.e, changes in mean arterial blood pressure were < 5 mmHg in all groups and > 4 mmHg in older adults and heart rate saw changes < 5 b.min⁻¹. This lack of change in blood pressure is especially important for older adults, as lower blood pressure increases the risk of falls and syncope (fainting) events due to a limited blood flow to the brain (Röthlisberger et al., 2023). Previous work involving whole body heating has reported a decreased blood pressure resulting from vasodilation (Pizzey et al., 2021) whilst reports of passive heat exposure reducing blood pressure can vary (Romero et al., 2022; Rowell et al., 1969), individual responses of -20mmHg have been observed in response to passive heat stress (Ganio et al., 2011). Some older adults experience disorientation or syncopal episodes when standing from seated. This condition, known as orthostatic hypotension, is characterised by a significant drop in blood pressure upon standing, resulting in decreased cerebral perfusion and potential loss of consciousness (Dani et al., 2021). If a passive heating methodology decreases blood pressure by >6 mmHg (Freeman et al., 2011), this condition would only be exacerbated and could not be recommended for older adults to aid in daily living tasks due to the risk of fall injury.

7.2.3 Peak force production

This thesis aimed to expand on the prior work that saw increases in force production during dynamic exercise (Faulkner et al., 2013) and test applicability to low repetition maximal effort contractions. This thesis also aimed to confirm velocity dependent benefits of passive heating (Sargeant, 1987) and observe the force production during early contraction to test if benefits observed within isometric contractions were also applicable to dynamic isokinetic contractions (Mornas et al., 2022; Rodrigues et al., 2021). Within this thesis a significant increase in muscle force production in moderate and fast isokinetic contractions was observed in young healthy adults after 30 mins of heating, this result is novel with all prior research to date reporting that isokinetic force production following passive heating does not increase (Cheung & Sleivert, 2004; Stanley et al., 1994). A crucial difference being that the present thesis intentionally maintained systemic normothermia with both aforementioned studies using 42°C hot water

immersion which induced substantial systemic hyperthermia i.e., core temperature $\approx 39.5^{\circ}\text{C}$. High core temperatures have been suggested to decrease neural drive and ameliorate ergogenic gains that may be occurring locally at the muscle (Rodrigues et al., 2022a; Thomas et al., 2006). The magnitude of the change that was observed in the moderate and fast isokinetic contractions, 8% and 10% respectively. Within this thesis >75% of the population saw peak torque increases (8% and 10% during moderate and fast isokinetic contractions) above the calculated minimal detectable change and can confidently be described as meaningful change above random variance. These results are similar in magnitude to the benefits observed in sprint cycling studies ((Sargeant, 1987) 10%) ((Faulkner et al., 2013) 11%). Interestingly the increases in muscular force presented in this study (+10 N.m, 8 - 10%) provides similar benefits to widely used performance aids such as caffeine (+5 N.m, 3%) (Grgic et al., 2022) and L-arginine (+4.2%) (Zart et al., 2023) and may be able to be applied simultaneously for maximal performance benefits; however, future work would need to be conducted to confirm this. Traditional active warm ups consisting of submaximal and maximal contraction have been shown to provide no significant benefits to peak torque production or total work done (Júnior, 2013.; Park et al., 2018). Cycling ergometry based warm ups have been shown to also not improve peak torque production, however, increases in the magnitude of EMG signal have been observed, suggesting a change in the mechanical properties of the muscle (Altamirano et al., 2012). In the research conducted for this thesis there was no correlation between the rate of change scale responses and the increases in peak force production in moderate or fast isokinetic contractions or in increases of RFD or EFP.

7.2.4 Early force production

In addition to the peak force production benefits observed in the faster contractile speed, increases in RFD and EFP were observed in the slow contraction speed in healthy younger and older adults. The older adults saw greater enhancements than the younger adults, +20% and +8% increase in early force production during slow isokinetic contractions respectively. When older and younger adult data was combined rate of force development in HEAT from baseline saw an increase of 29% and a difference from CONT of 16% from 30 min. These findings agree with prior work examining passive heating have observed rate of force production improvements of +26% at 50 ms (Rodrigues et al., 2021) and +48% at 100 ms (Mornas et al., 2022) in younger adults. The current work may have been underpowered to observe the 3 way interaction in RFD but given a similar pattern in younger and older adults was observed whereby the heated limb saw increases from baseline whilst the control did not it is assumed that no age specific differences occurred. Enhancements in early force production and rate of force development were observed in the slowest isokinetic contractile

speed only, with previous research that demonstrated ergogenic benefits of passive heating utilising isometric contractions to observe this phenomenon (Mornas et al., 2022; Rodrigues et al., 2021). Taken together it may be that only the slowest contractions are beneficiaries in response to the intervention.

7.2.5 Mechanistic insights

The current mechanistic theories speculate that the benefits to peak and early force production may be explained by heating increasing attachment and detachment rates within cross bridge formation. Elevated muscle temperatures enhance the rates at which cross-bridges attach to and detach from actin filaments, heating increases the rate of calcium ion release and reuptake within muscle cells as Ca^{2+} binding to troponin C facilitates cross-bridge formation, faster calcium kinetics contribute to more rapid contraction and relaxation cycles. (Rodrigues et al., 2022a). Research should attempt to investigate calcium concentrations within muscle fibres using ion-selective electrode potentiometry or indicator dyes (Baylor & Hollingworth, 2011; Lambolley et al., 2015). Enhancements in nerve conduction velocity may also play a role in the increase in early force production, a higher nerve conduction velocity enables faster transmission of motor signals, leading to quicker muscle activation and contraction (Kiernan et al., 2001; Todnem et al., 1989). Muscular gearing through the increase of intramuscular fluid, optimising muscle pennation angle and creating a stiffer medium for force to more efficiently pass through may also contribute to increased early force development (Eng et al., 2018; Sleboda et al., 2019). To investigate this mechanism 3D ultrasound should be employed to create a better understanding of muscle structure before and after passive heat exposure (Sahrmann et al., 2024). These mechanisms have been suggested to be the cause of the improvements observed in muscular function, both increases in peak power and force as well as increases in RFD (Rodrigues et al., 2022a), therefore, the increases in peak torque in moderate and fast contractile speeds may be attributed to the increased rate at which muscle fibres can be activated and the increased mobilisation time allows for the increase in cross bridge formations, increasing peak force as the muscle moves through its range of motion. Increases in peak muscle force production may not be observed within slower contractions as there is already plenty of time for full cross bridge formation; however, in slower contractions the effect of increased cross bridge formation may be observed during the very early stages of contraction before maximal activation occurs, EFP and RFD. The evidence from the experimental studies within this thesis suggest that the primary mechanism behind the increased peak force production in moderate and fast isokinetic contractions is different than the primary mechanism behind the increases in RFD and EFP, this is evidenced by the healthy

older adults seeing no enhancement in peak force production but large gains in RFD and EFP, whilst the healthy younger adults saw increases in both.

This thesis did not investigate the mechanisms underpinning muscle function following passive heating, for each proposed mechanism there is an explanation explained by normal ageing which may blunt the effectiveness of heating. The hypothesis that increased intramuscular fluid enhances muscle stiffness and optimizes muscle fibre alignment for exercise (Eng et al., 2018) may not fully apply to older adults. This demographic often exhibits higher intramuscular fat deposits (Pinel et al., 2021) which are more compressible than muscle tissue, potentially preventing the muscle from achieving the theorized optimal deformation. In this study, older participants had a 10% higher body fat percentage. Although intramuscular fat wasn't directly measured, this suggests a greater absolute fat mass. Considering that aging is associated with fat redistribution, leading to visceral fat infiltrating skeletal muscle (Li et al., 2022), it's likely that these individuals had increased fat deposits within muscle tissue. Aging also impairs the Sarco/Endoplasmic Reticulum Calcium ATPase (SERCA) pump, slowing calcium release into working muscles (Miljkovic et al., 2015) and hindering subsequent calcium reuptake (Carmeli et al., 2002; Hunter et al., 1999). In young, healthy adults, skeletal muscle hyperthermia stimulates the SERCA pump (Davies & Young, 1983), enhancing calcium reuptake and reducing muscle half-relaxation time (Rodrigues et al., 2022a). However, since the SERCA pump is susceptible to various age-related muscle pathologies (Xu & Van Remmen, 2021), its responsiveness to passive heating may be diminished in older adults. Furthermore, aging often leads to increased variability in motor neuron function (Welsh et al., 2007) and a decrease in nerve conduction velocity (Rivner et al., 2001).

Peak isotonic contraction velocity saw no improvement within this study, despite being the fastest contraction type within the study- with contractile speeds frequently being $>400^{\circ}/s$. This may be explained as passive heating having peak benefit within an optimal contractile velocity range rather than faster velocities simply benefiting more. It is however possible that isotonic contractions do not benefit in the same way isokinetic contractions do, further research is required to test this.

7.2.6 Timecourse of heating benefit and decay of the heating effect

The application of localised passive heating for 30 mins was enough to observe significant increases in peak isokinetic force production (+7%) and increased RFD (+13%) and EFP (+9%) in younger adults; however, older adults required 60 mins of heating before observing EFP (+21%) enhancements. It is important therefore to further explore the differences in kinetic response between younger and older adult responses to heating so that optimal

programming may be prescribed to each population. This thesis also tested if heated benefits would remain 30 min post cessation of heating. Thirty mins following heating withdrawal the increases in peak isokinetic torque enhancements during moderate and fast contractions and increases in early force production during slow contractions were maintained in younger adults, whilst the early force production enhancements in older adults were lost. The need to maintain muscle temperature after a warm up, has been established previously, noting a 10% decrease in peak power when muscle temperature was not maintained 30 min post warm up (Faulkner et al., 2013). The authors suggest that the maintenance of heat was important due to a heightened rate of ATP turnover resulting from the increased muscle temperature. This is supported by the elevated blood lactate levels observed after the maximal sprint under the HEAT condition. The results from this thesis suggest that heat maintenance may be especially necessary for older adults.

7.3 Limitations

While the findings of this thesis highlight the potential benefits of passive heating for improving muscle function, there are several limitations that warrant consideration. Firstly, whilst isokinetic dynamometry is considered the gold standard for muscle function assessment it is important to state that the results derived from dynamometry does not truly reflect how a muscle will function during physical activities or daily living tasks. Additionally, this thesis has a limited generalisability of findings across different populations. While healthy younger and older adults were included, variations in training status, sex differences, and potential clinical populations were not examined. The current research did not specifically assess the level of sarcopenia within the older adult population. The participants were all able to locomote independently and many were regular participants in moderate intensity exercise, suggesting their classification of sarcopenia to be non-sarcopenic (Cruz-Jentoft et al., 2010), therefore the findings may not be applicable to those with sarcopenia or severe sarcopenia. The lack of sarcopenia control could have affected the homogeneity of the older adult group. The surface EMG measures within the experimental studies were not time-synched to the isokinetic dynamometer limiting the analysis available, however, this had no impact on the ability to measure peak outputs. There was also no control for EMG signal cross talk, recorded electrical signal from nearby synergistic muscle, although as this study was interested in holistic muscle function rather than isolated vastus lateralis function, the testing all occurred on the same day further limiting the effect of cross talk as this would have been measured at all timepoints. The testing within this study, whereby muscle function was assessed every 30 mins resulted in increased muscle temperature in the control limb and was therefore not truly representative of how 90 mins of heating at rest may affect muscle function. Moreover, the optimal heating duration and decay period require further clarification. The current research

suggests that older adults may need longer exposure to achieve similar benefits as younger adults, but more detailed time-course studies could establish precise recommendations. Finally, while heating was shown to enhance perceived readiness for exercise, the psychological effect may have been influenced by the emphasis that was placed on the heated leg as it was wrapped in the heated garment, further research may look to standardise or use a slightly warmed leg as a control to investigate this phenomenon.

7.4 Future Research directions and application of passive heating

The use of passive heating as a long term tool has several speculative benefits. This field of study still requires a considerable number of questions to be answered; mechanistic work should be undertaken to ascertain the main drivers of improved muscle function following passive heating. Further research investigating how long term use of passive heating as a tool prior to exercise affects muscular hypertrophy and muscular strength gains. Finally, research investigating how passive pre-heating affects different exercise types, including multi joint exercise and balance-based tasks. The effect on real world tasks or daily living tasks would be a key next step in identifying the usage and benefit that passive preheating may have as a tool for older adults who struggle due to a lack of muscular power. As previously mentioned in 7.2.5 Mechanistic insights future research is needed to understand the physiological mechanisms that underpin muscle function enhancement, whilst recent research has speculated that calcium handling, nerve conduction velocity and muscle structure may be the main driving factors (Rodrigues et al., 2022); there is little observational data within humans during exercise to confirm that these mechanisms are improving muscle function after passive heating.

The experimental studies within this thesis propose a novel finding that passive heating increases the perception of readiness for exercise and decrease the perceived exertion following exercise. The psychological aspects of why older adults are not engaging with exercise and physical activity have been well documented: worries about physical ability, bodily pain or fear of bodily pain prevent many older adults from partaking in physical activity (Clark, 1999; Costello et al., 2011). Subjective perceptions of how an intervention makes a participant feel is often overlooked in physiological research. Whilst perception may not be reality, it is the individual's reality; if an intervention increases perceptions of readiness for exercise and decreases negative feelings of fatigue, even if nothing changes physiologically, those feelings are still positively influencing that individual as it relates to physical activity. Pre-exercise rituals including mental warm ups have shown efficacy in improving perceptions of readiness for sport (Van Raalte et al., 2019), along with the aforementioned physiological benefits to heating muscle the analgesic properties of heating (Chabal et al., 2020) are

expected to create a sensation of preparedness for exercise in younger and older adults. The potential psychological benefits, combined with the potential physiological improvements, highlight the multifaceted role of passive heating as an exercise tool.

Improvements in acute perceptions of readiness for exercise can positively influence exercise participation and performance in both younger and older adults. For younger individuals, enhanced readiness may boost training intensity, consistency, and long-term fitness gains. In older adults, better perceived readiness can reduce psychological barriers to activity, increase adherence to exercise programs, and support functional independence. Across age groups, fostering a positive mindset before exercise may help optimise physical and mental benefits and promote a more enjoyable and sustainable active lifestyle. The integration of passive heating protocols into daily routines, such as during periods of sedentary behaviour, meals or transportation, presents a practical and accessible strategy to enhance the benefits of resistance exercise. Utilising heating methods prior to training has been shown to provide a range of physiological and psychological advantages, which, although minor in isolation, collectively contribute to improved exercise outcomes. This strategy offers a simple yet effective means to optimise muscle function, enhance training adaptations, and improve overall exercise adherence. Incorporating heating as part of a pre exercise ritual may reduce physical activity avoidance and reduce perceptions of failure (Hobson et al., 2017). The psychological benefits of passive heating remain largely speculative and further work should be undertaken to fully assess how heating affects, mood, motivation and perceived fatigue in a single acute session whilst exercise adherence, mental health and enjoyment of exercise should be observed after a longer period of using passive heating as a pre exercise tool.

Further research is warranted to ascertain whether acute exposure to passive heating confers measurable performance benefits during resistance training sessions. Although the present thesis has identified increases in peak force production following passive heat exposure, the extent to which these findings translate to improvements in dynamic, task-specific resistance exercise remains uncertain. By contrast, caffeine, a well-documented ergogenic aid, has demonstrated not only enhancements in isokinetic force production (Grgic & Pickering, 2019), but also clear translatability to resistance training contexts. Empirical evidence supports its efficacy in augmenting one-repetition maximum strength, prolonging time under tension, and increasing power output during resistance exercise (Filip-Stachnik et al., 2021; Grgic & Mikulic, 2021). This distinction underscores the necessity for further investigation into the applied effects of passive heating protocols on resistance training performance.

Passive heating which results in an acute increase in muscle function can potentially lead to more effective training sessions, with higher peak forces achieved there is a potential for greater training adaptations. Over time, higher-quality workouts may contribute to greater long-term gains in muscle strength and size. Resistance exercise stimulates hypertrophy through a complex combination of molecular, immune, metabolic, and endocrine signalling pathways (Schoenfeld, 2013); similarly, exposure to heat stress can trigger a variety of beneficial molecular responses. Heat stress has been shown to activate the Akt/mTOR signalling pathway (Kakigi et al., 2011), a key regulator of muscle protein synthesis and hypertrophy. The exposure to heat stress increases heat stress protein (HSP) expression (Gibson et al., 2014, 2015, 2023), particularly HSP72, may facilitate the activation of the mTOR pathway, promoting muscle growth (Naito et al., 2012). Whilst mTOR increases the drive to anabolism just as importantly heat stress may decrease the catabolic drive within the body, which may be especially pertinent for older adults, HSPs can mitigate muscle atrophy by inhibiting pathways that lead to muscle degradation. Increased expression of HSPs has been associated with down regulating the expression of muscle specific E3 ubiquitin ligases involved in protein degradation (Fennel et al., 2022). Aerobic capacity, increased insulin sensitivity and skeletal muscle capillarisation have also been reported to benefit from heating (Wagenmakers et al., 2019).

The use of localised heating has been suggested to improve static and dynamic balance in younger adults, however, the efficacy of this effect in older adults has yet to be investigated, this could have implications for the development of a beneficial tool to help address a common problem faced by many older adults. While further research is needed to fully understand the long-term implications of heat exposure in resistance training, the current evidence suggests that passive heating serves as a valuable complementary tool in strength and power physical activities.

8 Conclusion

The findings presented in this thesis demonstrate that localised passive heating can serve as an effective ergogenic aid for improving muscle function, particularly in younger adults. The experimental studies provide novel evidence that heating enhances peak isokinetic force production in moderate and fast contractions while also improving early force production and rate of force development in both younger and older adults. These benefits appear to be independent of systemic physiological changes, suggesting that local heating directly influences muscle contractile properties. While older adults did not experience improvements in peak force production, they exhibited notable gains in early force production, highlighting the potential for passive heating to support this critical aspect of neuromuscular function in aging populations. Muscular power has been identified as a key component of everyday living tasks, the observed improvements in early force production indicate an increase in muscular power generation which may improve quality of life through improved ability to complete tasks that are associated with independent living. The observed psychological benefits, including increased perceptions of readiness for exercise and reduced perceived exertion, further suggest that passive heating could play a valuable role in developing self-efficacy during tasks of daily living and encouraging physical activity, particularly in populations that may be hesitant to engage in movement due to discomfort.

Despite these promising findings, several questions remain regarding the long term effects, underlying mechanisms, and optimal application of passive heating. Practical considerations, such as the most effective heating duration and the best strategies for integrating passive heating into daily routines or training programs, should be explored. While passive heating presents a simple, non-invasive tool for improving muscle function, further research is necessary to fully harness its potential and optimize its application in both athletic and rehabilitative settings.

9 References

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10 Appendices

Appendix A. Custom Matlab code for bandpass filtering and rectifying surface EMG data

```
% Define the file path
file_path = 'E:\PhD\Delsys Folder\4.xlsx';

% Read the Excel file
[num_data, ~, ~] = xlsread(file_path);

% Extract EMG data from column B
EMG = num_data(:, 2); % Assuming column B contains EMG data

% Parameters for bandpass filter
fs_bandpass = 2150; % Sampling frequency for bandpass filter
fcut_bandpass = [6 500]; % Bandpass cutoff frequencies
order_bandpass = 4; % Filter order for bandpass filter

% Apply bandpass filter to EMG data
MFilt = bandpass(EMG, fcut_bandpass, fs_bandpass);

% Compute mean of the filtered signal
MfiltMean = mean(MFilt);

% Subtract mean from filtered signal
Mfiltoffset = MFilt - MfiltMean;

% Calculate absolute values
Mfiltoffsetrec = abs(Mfiltoffset);

% Parameters for lowpass filter
fs_lowpass = 2250; % Sampling frequency for lowpass filter
fc_lowpass = 10; % Lowpass cutoff frequency
order_lowpass = 2; % Filter order for lowpass filter

% Design lowpass filter
[b_lowpass, a_lowpass] = butter(order_lowpass, fc_lowpass/(fs_lowpass/2), 'low');

% Apply lowpass filter to the signal
filtered_signal = filtfilt(b_lowpass, a_lowpass, Mfiltoffsetrec);

% Find peak value
peak_value = max(filtered_signal);

% Display peak value
disp(['Peak value: ', num2str(peak_value)]);
% Plot the filtered signal
plot(filtered_signal);
hold on;
% Mark the peak value
plot(find(filtered_signal == peak_value), peak_value, 'ro', 'MarkerSize', 10);
hold off;
xlabel('Sample');
ylabel('Filtered Signal');
title('Filtered Signal with Peak Value');
legend('Filtered Signal', ['Peak Value: ', num2str(peak_value)]);
```

Appendix B- Ethical approval for data collection for chapter 4



College of Health, Medicine and Life Sciences Research Ethics Committee (DLS)
Brunel University London
Kingston Lane
Uxbridge
UB8 3PH
United Kingdom
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24 May 2024

LETTER OF APPROVAL

APPROVAL HAS BEEN GRANTED FOR THIS STUDY TO BE CARRIED OUT BETWEEN 02/10/2023 AND 08/10/2024

Applicant (s): Mr Desmond Denny

Project Title: Effect of Local hyperthermia on upper leg muscular function in healthy older adults

Reference: 44577-A-May/2024- 50884-1

Dear Mr Desmond Denny

The Research Ethics Committee has considered the above application recently submitted by you to increase the upper age range from 80 to 90.

The Chair, acting under delegated authority has agreed that there is no objection on ethical grounds to the proposed study. Approval is given on the understanding that the conditions of approval set out below are followed:

- **The agreed protocol must be followed. Any changes to the protocol will require prior approval from the Committee by way of an application for an amendment.**

Please note that:

- Research Participant Information Sheets and (where relevant) flyers, posters, and consent forms should include a clear statement that research ethics approval has been obtained from the relevant Research Ethics Committee.
- The Research Participant Information Sheets should include a clear statement that queries should be directed, in the first instance, to the Supervisor (where relevant), or the researcher. Complaints, on the other hand, should be directed, in the first instance, to the Chair of the relevant Research Ethics Committee.
- Approval to proceed with the study is granted subject to any conditions that may appear above.
- The Research Ethics Committee reserves the right to sample and review documentation, including raw data, relevant to the study.
- If your project has been approved to run for a duration longer than 12 months, you will be required to submit an annual progress report to the Research Ethics Committee. You will be contacted about submission of this report before it becomes due.
- You may not undertake any research activity if you are not a registered student of Brunel University or if you cease to become registered, including abeyance or temporary withdrawal. As a deregistered student you would not be insured to undertake research activity. Research activity includes the recruitment of participants, undertaking consent procedures and collection of data. Breach of this requirement constitutes research misconduct and is a disciplinary offence.

Professor Louise Mansfield

Chair of the College of Health, Medicine and Life Sciences Research Ethics Committee (DLS)

Brunel University London

Appendix C Ethical approval for data collection for chapters 5 & 6



University Research Ethics Committee
Brunel University London
Kingston Lane
Uxbridge
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3 November 2023

LETTER OF APPROVAL

APPROVAL HAS BEEN GRANTED FOR THIS STUDY TO BE CARRIED OUT BETWEEN 05/09/2022 AND 01/09/2023 01/09/2023 01/09/2023
01/09/2023 01/09/2023

Applicant (s): Mr Desmond Denny

Project Title: Examining the reliability of changes in upper leg muscle function via dynamometry in response to upper leg hyperthermia verses normothermia

Reference: 37843-A-Sep2023- 47312-1

Dear Mr Desmond Denny

The Research Ethics Committee has considered the above application recently submitted by you.

The Chair, acting under delegated authority has agreed that there is no objection on ethical grounds to the proposed study. Approval is given on the understanding that the conditions of approval set out below are followed:

- **The agreed protocol must be followed. Any changes to the protocol will require prior approval from the Committee by way of an application for an amendment.**
- **Please ensure that you monitor and adhere to all up-to-date local and national Government health advice for the duration of your project.**

Please note that:

- Research Participant Information Sheets and (where relevant) flyers, posters, and consent forms should include a clear statement that research ethics approval has been obtained from the relevant Research Ethics Committee.
- The Research Participant Information Sheets should include a clear statement that queries should be directed, in the first instance, to the Supervisor (where relevant), or the researcher. Complaints, on the other hand, should be directed, in the first instance, to the Chair of the relevant Research Ethics Committee.
- Approval to proceed with the study is granted subject to any conditions that may appear above.
- The Research Ethics Committee reserves the right to sample and review documentation, including raw data, relevant to the study.
- If your project has been approved to run for a duration longer than 12 months, you will be required to submit an annual progress report to the Research Ethics Committee. You will be contacted about submission of this report before it becomes due.
- You may not undertake any research activity if you are not a registered student of Brunel University or if you cease to become registered, including abeyance or temporary withdrawal. As a deregistered student you would not be insured to undertake research activity. Research activity includes the recruitment of participants, undertaking consent procedures and collection of data. Breach of this requirement constitutes research misconduct and is a disciplinary offence.

Professor Christina Victor

Chair of the University Research Ethics Committee

Brunel University London

Appendix D. Three way ANOVA SPSS output for peak force data for 20 young healthy adults for CHAPTER 4 - Experimental study 1 – Passive thigh heating improves isokinetic but not isotonic muscle function.

		Isokinetic torque at 60°/s	Isokinetic torque at 180°/s	Isokinetic torque at 300°/s	Isotonic velocity at 25% MVC
Visit	Mauchly's				
	F-value	1.789	4.178	7.588	4.740
	P-value	.197	.055	.013	.042
	η^2	.086	.180	.285	.200
	power	.246	.492	.742	.542
Time	Mauchly's	.590	<.001	.014	.047
	F	.512	1.894	2.8	.565
	P	.676	.170	.048	.641
	ES (partial η^2)	.026	.091	.128	.029
	observed power	.148	.345	.645	.143
Leg	Mauchly's				
	F	8.118	9.885	12.031	1.739
	P	.010	.005	.003	.203
	ES (partial η^2)	.299	.342	.388	.084
	observed power	.771	.847	.908	.240
Visit*Time	Mauchly's	.285	.001	.131	.285
	F	.435	3.096	2.926	.046
	P	.729	.063	.041	.987
	ES (partial η^2)	.022	.140	.133	.002
	observed power	.132	.533	.666	.058
Visit*Leg	Mauchly's				
	F	.373	.012	.803	.301
	P	.548	.913	.381	.590
	ES (partial η^2)	.019	.001	.041	.016
	observed power	.089	.051	.136	.082
Time*Leg	Mauchly's	.049	.040	.082	.037
	F	.708	4.524	7.062	.801
	P	.516	.012	<.001	.465
	ES (partial η^2)	.036	.192	.271	.040
	observed power	.169	.790	.974	.183
Visit*Leg*Time	Mauchly's	.136	.515	.127	.011
	F	.964	1.586	2.879	2.354
	P	.416	.203	.044	.105
	ES (partial η^2)	.048	.077	.132	.110
	observed power	.250	.395	.658	.464

Appendix E. 3 way ANOVA SPSS output for early force production for 20 young healthy adults for CHAPTER 4 - Experimental study 1 – Passive thigh heating improves isokinetic but not isotonic muscle function.

		Isokinetic torque at 0.18s during 60°/s	Isokinetic torque at 0.18s during 180°/s	Isokinetic torque at 0.18s during 300°/s	Isotonic velocity at 0.18s during 25% MVC
Visit	Mauchly's				
	F	2.593	3.924	.998	6.521
	P	.124	.062	.330	.019
	ES (partial η^2)	.120	.171	.050	.256
	observed power	.334	.468	.158	.678
Time	Mauchly's	.002	.007	.891	.195
	F	16.870	2.564	1.372	.228
	P	<.001	.091	.260	.877
	ES (partial η^2)	.470	.119	.067	.012
	observed power	.999	.476	.346	.090
Leg	Mauchly's				
	F	2.697	9.127	4.806	2.030
	P	.117	.007	.041	.170
	ES (partial η^2)	.124	.324	.202	.097
	observed power	.345	.817	.548	.273
Visit*Time	Mauchly's	.158	.002	.229	.434
	F	5.567	1.921	2.299	2.393
	P	.005	.165	.087	.078
	ES (partial η^2)	.227	.092	.108	.112
	observed power	.876	.356	.550	.074
Visit*Leg	Mauchly's				
	F	.235	.012	.315	.230
	P	.633	.914	.581	.637
	ES (partial η^2)	.012	.001	.016	.012
	observed power	.075	.051	.083	.074
Time*Leg	Mauchly's	.411	.030	.124	.008
	F	4.002	2.725	.637	1.081
	P	.012	.071	.594	.347
	ES (partial η^2)	.174	.125	.032	.054
	observed power	.812	.542	.175	.220
Visit*Leg*Time	Mauchly's	.740	.480	.912	.184
	F	.840	1.445	1.834	2.549
	P	.478	.239	.151	.065
	ES (partial η^2)	.042	.071	.088	.118
	observed power	.221	.363	.452	.599

Appendix F. 2 way ANOVA SPSS output for physiological responses and thermal sensation for 20 young healthy adults for CHAPTER 4 - Experimental study 1 – Passive thigh heating improves isokinetic but not isotonic muscle function.

		Heart Rate	SBP	DBP	MAP	Tympanic	T. Sensation
Visit	Mauchly's						
	F	.309	.241	4.689	2.756	3.065	2.883
	P	.585	.629	.043	.113	.096	.106
	ES (partial η^2)	.289	.013	.198	.127	.139	.132
	observed power	.083	.075	.538	.351	.383	.364
Time	Mauchly's	.002	.160	.929	.207	<.001	<.001
	F	7.706	6.685	5.102	8.723	10.812	48.592
	P	.003	<.001	.003	<.001	<.001	<.001
	ES (partial η^2)	.389	.260	.212	.315	.363	.719
	observed power	.903	.966	.902	.992	.973	1
Visit*Time	Mauchly's	.080	.417	.639	.241	.001	.047
	F	.287	.334	.815	.650	.594	2.240
	P	.835	.801	.491	.586	.539	.117
	ES (partial η^2)	.015	.017	.041	.033	.030	.105
	observed power	.102	.111	.215	.178	.136	.440

Appendix G. 3 way ANOVA SPSS output for muscle and skin temperature for 20 young healthy adults for CHAPTER 4 - Experimental study 1 – Passive thigh heating improves isokinetic but not isotonic muscle function.

		T _{mu}	T _{skin}	RPE
Visit	Mauchly's			
	F	.099	1.137	0
	P	<.001	.322	1
	ES (partial η^2)	.011	.140	0
	observed power	.059	.153	.5
Time	Mauchly's	.009	.017	<.001
	F	79.496	312.971	23.985
	P	<.001	<.001	<.001
	ES (partial η^2)	.898	.978	.558
	observed power	1	1	1
Leg	Mauchly's			
	F	44.516	300.401	14.951
	P	<.001	<.001	.001
	ES (partial η^2)	.832	.977	.440
	observed power	1	1	.956
Visit*Time	Mauchly's	.523	.233	.231
	F	5.289	.840	2.370
	P	.005	.487	.080
	ES (partial η^2)	.37	.107	.111
	observed power	.889	.201	.565
Visit*Leg	Mauchly's			
	F	1.628	.267	.147
	P	.234	.621	.706
	ES (partial η^2)	.153	.037	.008
	observed power	.208	.073	.065
Time*Leg	Mauchly's	.56	.024	.061
	F	51.244	181.431	1.533
	P	<.001	<.001	.216
	ES (partial η^2)	.851	.963	.075
	observed power	1	1	.383
Visit*Leg*Time	Mauchly's	.098	.646	.002
	F	.040	.856	.509
	P	.989	.479	.621
	ES (partial η^2)	.004	.109	.026
	observed power	.056	.204	.131

Appendix H. 3 way ANOVA SPSS output for peak force data for 22 young healthy adults and 16 older healthy adults for CHAPTER 5 - Experimental study 2 – Passive thigh heating improves peak force production in younger adults and early isokinetic force production in younger and older adults.

		Isokinetic torque at 60°/s	Isokinetic torque at 180°/s	Isokinetic torque at 300°/s	Isotonic velocity at 25% MVC
Condition	Mauchly's				
	F-value	13.293	6.675	5.862	0.025
	P-value	<.001	0.014	0.021	0.874
	η^2	0.27	0.156	0.140	0.001
	power	0.944	0.710	0.654	0.053
Condition * Group	Mauchly's				
	F	0.272	1.729	3.946	2.572
	P	0.605	0.197	0.055	0.117
	ES (partial η^2)	0.007	0.046	0.099	0.065
	observed power	0.08	0.249	0.489	0.346
Time	Mauchly's	0.015	<0.001	<0.001	0.009
	F	1.59	5.771	2.793	0.987
	P	0.18	0.004	0.064	0.401
	ES (partial η^2)	0.042	0.138	0.072	0.026
	observed power	0.481	0.874	0.550	0.262
Time * Group	Mauchly's				
	F	0.395	0.441	0.297	1.805
	P	0.812	0.800	0.880	0.131
	ES (partial η^2)	0.011	0.011	0.008	0.047
	observed power	0.139	0.144	0.115	0.540
Condition * Time	Mauchly's	0.003	<0.001	0.030	0.332
	F	0.125	1.185	3.541	1.690
	P	0.973	0.320	0.015	0.155
	ES (partial η^2)	0.003	0.032	0.090	0.044
	observed power	0.072	0.365	0.858	0.509
Condition * Time * Group	Mauchly's				
	F	0.979	3.084	1.611	0.435
	P	0.406	0.018	0.175	0.783
	ES (partial η^2)	0.026	0.79	0.043	0.012

	observed power	0.261	0.800	0.487	0.150
--	-------------------	-------	-------	-------	-------

Appendix I. 3 way ANOVA SPSS output for early force production for 22 young healthy adults and 16 older healthy adults for CHAPTER 5 - Experimental study 2 – Passive thigh heating improves peak force production in younger adults and early isokinetic force production in younger and older adults.

		Isokinetic torque at 0.18s during 60°/s	Isokinetic torque at 0.18s during 180°/s	Rate of force production at 50ms	
Condition	Mauchly's				
	F	9.715	8.513	5.126	
	P	0.004	0.006	0.030	
	ES (partial η^2)	0.217	0.196	0.131	
	observed power	0.858	0.810	0.595	
Condition*Group	Mauchly's				
	F	0.033	0.275	0.306	
	P	0.857	0.603	0.584	
	ES (partial η^2)	0.001	0.008	0.009	
	observed power	0.054	0.080	0.084	
Time	Mauchly's	<0.001	<0.001	0.004	
	F	6.307	3.040	11.101	
	P	0.001	0.039	<0.001	
	ES (partial η^2)	0.153	0.080	0.246	
	observed power	0.937	0.660	1.000	
Time*Group	Mauchly's				
	F	1.323	0.864	0.292	
	P	0.264	0.487	0.883	
	ES (partial η^2)	0.036	0.024	0.009	
	observed power	0.405	0.270	0.113	
Condition*Time	Mauchly's	0.018	<0.001	0.047	
	F	5.632	1.358	6.015	
	P	<0.001	0.252	<0.001	
	ES (partial η^2)	0.139	0.037	0.150	
	observed power	0.976	0.415	0.962	
Condition*Time*Group	Mauchly's				
	F	2.814	1.613	0.800	
	P	0.028	0.174	0.527	
	ES (partial η^2)	0.074	0.044	0.023	
	observed power	0.757	0.487	0.251	

Appendix J. 2 way ANOVA SPSS output for physiological responses and thermal sensation for 22 young healthy adults and 16 older healthy adults for CHAPTER 5 - Experimental study 2 – Passive thigh heating improves peak force production in younger adults and early isokinetic force production in younger and older adults.

		Heart Rate	MAP	Tympanic	T. Sensation
Time	Mauchly's	<0.001	0.002	<0.001	<0.001
	F	2.682	6.057	1.050	30.796
	P	0.034	<0.001	0.384	<0.001
	ES (partial η^2)	0.071	0.148	0.029	0.461
	observed power	0.589	0.984	0.180	1.000
Group*Time	Mauchly's				
	F	1.038	0.727	1.356	3.566
	P	0.390	0.575	0.252	0.008
	ES (partial η^2)	0.029	0.020	0.037	0.090
	observed power	0.321	0.230	0.415	0.861

Appendix K. 3 way ANOVA SPSS output for RPE and skin temperature for 22 young healthy adults and 16 older healthy adults for CHAPTER 5 - Experimental study 2 – Passive thigh heating improves peak force production in younger adults and early isokinetic force production in younger and older adults.

		RPE	T _{skin}
Condition	Mauchly's		
	F	12.354	79.995
	P	0.001	<0.001
	ES (partial η^2)	0.261	0.769
	observed power	0.927	1.000
Condition*Group	Mauchly's		
	F	4.909	0.847
	P	0.033	0.366
	ES (partial η^2)	0.123	0.034
	observed power	0.577	0.143
Time	Mauchly's	<0.001	<0.001
	F	17.958	30.845
	P	<0.001	<0.001
	ES (partial η^2)	0.339	0.562
	observed power	1.000	1.000
Time*Group	Mauchly's		
	F	5.449	0.423
	P	<0.001	0.531
	ES (partial η^2)	0.135	0.017
	observed power	0.972	0.097
Condition*Time	Mauchly's	0.379	<0.001
	F	0.169	24.168
	P	0.345	<0.001
	ES (partial η^2)	0.847	0.502
	observed power	0.127	1.000
Condition*Time*Group	Mauchly's		
	F	2.638	1.688
	P	0.037	0.159
	ES (partial η^2)	0.070	0.066
	observed power	0.726	0.501

Appendix L. 2 way ANOVA SPSS output for peak torque, total work done and skin temperature during a 30 repetition isokinetic fatigue task in 15 older healthy adults for CHAPTER 6 - Experimental study 3 – Localised passive heating does not improve isokinetic fatigue resistance during repeated and prolonged knee extensor exercise.

		Peak torque	Total work	Skin Temp
Condition	Mauchly's			
	F	13.460	0.264	202.185
	P	0.003	0.615	<0.001
	ES (partial η^2)	0.490	0.019	0.940
	observed power	0.926	0.077	1.000
Time	Mauchly's			
	F	4.171	2.596	246.798
	P	0.060	0.129	<0.001
	ES (partial η^2)	0.230	0.156	0.950
	observed power	0.477	0.324	1.000
Condition*Time	Mauchly's			
	F	3.112	2.736	102.002
	P	0.100	0.120	<0.001
	ES (partial η^2)	0.182	0.163	0.887
	observed power	0.376	0.338	1.000

Appendix M. 3 way ANOVA SPSS output for averaged torque during a 30 repetition isokinetic fatigue task in 15 older healthy adults for CHAPTER 6 - Experimental study 3 – Localised passive heating does not improve isokinetic fatigue resistance during repeated and prolonged knee extensor exercise.

		Averaged Torque during fatigue task
Condition	Mauchly's	
	F	3.028
	P	0.104
	ES (partial η^2)	0.178
	observed power	0.367
Time	Mauchly's	
	F	8.771
	P	0.010
	ES (partial η^2)	0.385
	observed power	0.786
Thirds	Mauchly's	<0.001
	F	26.895
	P	<0.001
	ES (partial η^2)	0.658
	observed power	.999
Condition*Time	Mauchly's	
	F	1.778
	P	0.204
	ES (partial η^2)	0.113
	observed power	0.237
Condition*Third	Mauchly's	0.026
	F	1.204
	P	0.305
	ES (partial η^2)	0.079
	observed power	0.203
Time*Thirds	Mauchly's	0.005
	F	4.351
	P	0.043
	ES (partial η^2)	0.237
	observed power	0.563
Time*Thirds*Condition	Mauchly's	0.007
	F	1.473
	P	0.248
	ES (partial η^2)	0.095
	observed power	0.232