

**The Effects of a Menthol-Based Topical Analgesic on Delayed Onset Muscle  
Soreness-Induced Changes to Running Biomechanics and Pain Perception**

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## Dedication

*To Dr. Michael W. Holmes,*

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## Abstract

The purpose of this thesis was to evaluate the effects of a menthol-based topical analgesic on delayed onset muscle soreness (DOMS) induced changes to running biomechanics and pain perception in well-trained runners. A menthol-based topical analgesic (n=10) was compared against a placebo group (n=10) on measures of kinematics, spatio-temporal parameters, and the perception of pain. Three-Dimensional (3D) kinematics of the ankle, knee and hip as well as subjective pain (Comparative Pain Scale and Pressure Threshold) were measured during level treadmill running at baseline, 48 hours after a 30-minute DOMS-inducing downhill run, and after the application of a menthol analgesic. DOMS was induced from the downhill run as identified by our pain measures, however it had little effect on kinematic variables. Pressure threshold was significantly lower at both measurement sites for both groups and Comparative Pain Scale scores were significantly higher after inducing DOMS. There were significant interactions for condition x group, regardless of running speed; average knee and hip range of motion (ROM) during stance and swing were significantly different than baseline after inducing DOMS. The application of a menthol-based topical analgesic had no significant effect on kinematics or pain perception. Our well-trained participants may have been more well-adapted to manage DOMS-induced soreness while limiting changes to running biomechanics. Variability in gait mechanics may have also played a role in the unexpected changes between participants after inducing DOMS. Regardless of the effectiveness of the DOMS-inducing protocol, the menthol analgesic appeared to have no effect on kinematics or pain variables in well-trained runners.

Key Words: Biomechanics, Gait, Menthol, Analgesic, Delayed Onset Muscle Soreness

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## Table of Contents

Dedication	2
Abstract	3
Acknowledgements	4
List of Figures	5
List of Tables	6
List of Appendices	7
Section 1: Introduction	
1.1 Background information	10
1.2 Research gap	16
Section 2: Literature review	
2.1 Running	
2.1.1 The Anatomy of running	17
2.1.2 Foot strike patterns	22
2.1.3 Segment and joint variability in running gait	24
2.2 Delayed Onset Muscle Soreness	
2.2.1 Causation and mechanism of action	25
2.2.2 Effect on biomechanical variables	31
2.2.3 Measuring delayed onset muscles soreness	32
2.2.4 Inducing delayed onset muscle soreness	33
2.2.5 Treatment of delayed onset muscle soreness	35
2.3 Menthol	
2.3.1 Mechanism of action	36
2.3.2 Effect on biomechanical variables	37
2.3.3 Risk of injury - Exercising with DOMS and menthol	37
Section 3: Study	
3.1 Research questions	39
3.2 Hypotheses	40
3.3 Methods	41
3.3.1 Participants	

3.3.2 Instrumentation	42
3.3.3 Experimental protocol	45
3.4 Data analysis	51
3.4.1 Motion capture	51
3.4.2 Pain	53
3.5 Statistical analysis	54
Section 4: Results	
4.1 Participant demographics	56
4.2 Summary of statistics	57
4.3 Effect sizes	58
4.4 Subjective pain analysis - CPS (comparative pain scale)	59
4.5 Subjective pain analysis - pressure algometry	61
4.6 Spatio-temporal parameters	64
4.7 Ankle kinematics - stance	66
4.8 Ankle kinematics - swing	67
4.9 Knee kinematics - stance	68
4.10 Knee kinematics - swing	70
4.11 Hip kinematics - stance	73
4.12 Hip kinematics - swing	76
Section 5: Discussion	79
Section 6: Considerations	92
Section 7: Conclusion and future directions	96
Section 8: References	99
Section 9: Appendices	110

### ***List of Figures***

Figure 1. Reflective marker setup	43
Figure 2. Motion capture volume	44
Figure 3. Study timeline	45
Figure 4. pressure algometer	47
Figure 5. Comparative Pain Scale	48
Figure 6. Motion capture labelling process	53
Figure 7. Study variables	55

### ***Results***

Figure 8. Comparative Pain Scale	60
Figure 9. Pressure threshold - gastrocnemius-achilles	62
Figure 10. Pressure threshold - vastus medialis obliques	63
Figure 11. Stride rate	64
Figure 12. Stride length	65
Figure 13. Ankle kinematics during stance	66
Figure 14. Ankle kinematics during swing	67
Figure 15. Knee average kinematics during stance	68
Figure 16. Knee peak flexion / extension during stance	69
Figure 17. Knee average kinematics during swing	70
Figure 18. Knee peak flexion during swing	71
Figure 19. Knee peak extension during swing	72
Figure 20. Hip average kinematics during stance	73
Figure 21. Hip peak flexion during stance	74
Figure 22. Hip peak extension during stance	75
Figure 23. Hip average kinematics during swing	76
Figure 24. Hip peak flexion during swing	77
Figure 25. Hip peak extension during swing	78

### *List of Tables*

Table 1. Normalized gait cycle	<b>19</b>
Table 2. Muscles and actions in sagittal plane running	<b>21</b>
Table 3. Intervention-based group demographics	<b>56</b>
Table 4. p-values for all main effects and interactions for each variable	<b>57</b>
Table 5. effect sizes for all main effects and interactions for each variable	<b>58</b>



### *List of Appendices*

8.1 Informed consent	110
8.2 Speed conversions	118
8.3 Pain journal	119
8.4 Individual CPS (comparative pain scale) scores	120
8.5 Gait event selection	121
8.6 Joint angle directions	122

## Section 1: Introduction

### 1.1 Background information

It is estimated that 56% of recreational runners and 90% of experienced runners will sustain an injury directly related to running each year (Van Gent et al., 2007). Running related injuries can range from low-grade muscle strains and tendon sprains to more severe fractures, joint damage, tears, and ruptures (Van Gent et al., 2007). For the recreational runner, an injury can result in the loss of motivation or the desire to exercise, and a loss of progress made through exercise towards living a healthier lifestyle. For the elite runner, the same outcomes can occur, however, more severe injuries can lead to the loss of income, years of hard work leading up to a major event, or the shortening of a running career. Running related injuries can be the result of a single running related incident (acute) or the accumulation of trauma (overuse) over years of running, without proper recovery.

Exercise Induced Muscle Damage (EIMD) is considered a mild type I muscle strain incurred through participating in a level of exercise that is unaccustomed in intensity or duration, relative to an individual (Gulick & Kimura, 1996; Cheung, Hume, & Maxwell, 2003). EIMD can lead to a phenomenon known as Delayed Onset Muscle Soreness (DOMS), where peak symptoms occur approximately 24-48 hours after the damage inducing exercise (Gulick & Kimura, 1996). Symptoms of DOMS are greatest during eccentric-biased exercise such as downhill running, where the musculoskeletal structures of the lower limbs are exposed to greater power absorption (Chen et al.,

2007a). The various symptoms of DOMS can lead to several negative consequences for an individual who continues to exercise namely: pain in the affected musculature with a potentially disproportionate increase in perceived dysfunction, decreased running economy, strength loss, reduced joint range of motion, increased ground reaction forces, amongst others (Chen et al., 2009; Dutto & Braun, 2004; McHugh et al., 1999; Connolly, Sayers, & McHugh, 2003).

In running, DOMS can lead to biomechanical adaptations to gait, potentially caused by the symptoms of DOMS and the perception of pain. This can lead to reduced range of motion through the lower limbs and a subconscious or conscious attempt to avoid loading the affected musculature to avoid further injury (Tsatalas et al. 2013). It has not been determined whether movement variability in gait is the result, or, cause of injury but there exists an operating range of variability that people move through and remain uninjured (Hamill et al., 2012). It's therefore important to note that too little or too much variability could be the result, or cause of injury (Hamill et al., 2012). Changes to movement variability and limb coordination as a result of injury has been speculated to be a risk factor for increased chance of further injury (Dutto & Braun, 2004; Hamill et al., 2012; Tsatalas et al., 2013). If DOMS is not properly managed, the muscles affected may not fully recover between exercise sessions, leading to fatigue and the use of secondary tissues to compensate for changes in gait mechanics brought on by DOMS. This compensation could lead to changes in joint loading and performance (Dutto & Braun, 2004). There is currently a lack of longitudinal studies to evaluate the effects of consistently training with DOMS on risk of injury or any implications it may have. However, given the noted changes in running biomechanics while running with

DOMS (Tsatalas et al. 2013; Chen et al., 2009; Dutto & Braun, 2004; Paschalis et al., 2007; Paquette et al., 2017), deviations from an individual's normal pattern would be cause for concern - where changes in variability have been associated with overuse injury (Hamill et al., 2012). Treatment options for DOMS have been widely researched. While some treatment options, such as massage and cryotherapy has produced mixed results, options such as light exercise have proven effective in reducing pain (Cheung, Hume, & Maxwell, 2003). In recent years, topical analgesics and counterirritants have been studied as a potential treatment option (Page & Alexander, 2017). Such treatments are designed to produce a sensory input to the nervous system that blocks the source of pain by direct application over affected areas (Page & Alexander, 2017). Topical analgesics typically produce a hot or cold effect - these effects are commonly produced by over the counter (OTC) medicinal compounds such as Capsaicin, Camphor, Methyl Salicylate, and Menthol (Martin et al., 2004).

Menthol is derived from the *Mentha* genus of plants which can provide a cooling, analgesic effect when applied topically (Yosipovitch et al., 1996). Menthol works through the activation of transient receptor potential - melastatin 8 (TRP-M8) ion channel, which detects cold stimuli in sensory neurons (Page & Alexander, 2017). The stimulation of TRP-M8 ion channels leads to a counterirritant effect which has the potential to block pain signals when applied to an area where pain is perceived (Liu et al., 2013). Page & Alexander (2017) discuss menthol-based products as a form of cryotherapy and have been studied extensively for the relief of pain caused by various musculoskeletal ailments. Menthol has been shown to have various effects including, decreased nerve conduction velocity, sensation, skin temperature, tissue metabolism,

and increased arteriolar vasoconstriction, superficial vasodilation, and pain threshold (Page & Alexander, 2017). Menthol can also reduce pain and restore or increase biomechanical and physiological variables in several studies looking at management of various chronic and acute musculoskeletal injuries (Zhang et al., 2008; Johar et al., 2012; Bishop et al., 2011; Sundstrup et al., 2014). Due to its pain-relieving properties, menthol is utilized in many products targeted at the relief of mild musculoskeletal pain sources such as aches, sprains, strains, and arthritis (Page & Alexander, 2017; Liu et al., 2013). These products may be combined with other active or inactive ingredients designed to further aid in ease of application or concomitantly reduce pain. There are a number of topical analgesic products that contain menthol as an active ingredient, typically containing anywhere from 2% to upwards of 16% in extra-strength products. To date, questions still remain about optimal dosage amount, concentration and time of application.

The effect of menthol on DOMS has been investigated, in which DOMS was induced in the elbow flexor musculature and a menthol-based topical analgesic was applied to mitigate the effects of DOMS (Johar et al., 2012). Johar et al., (2012) compared the effect of a menthol-based topical analgesic (Biofreeze®; 3.5% menthol), to ice on biomechanical and pain variables. Pain was significantly lower in the menthol group compared to the ice condition on a 100mm visual analogue scale (VAS), where a 63.1% lower pain score was reported. The menthol group also had an increased tetanic force contraction of 116.9%, compared to the ice group (Johar et al., 2012). While an increase in tetanic force production and a much lower pain score are important

practical findings, the use of menthol under dynamic conditions such as running has not yet been explored.

If DOMS can lead to a reduced range of motion (Chen et al., 2009; Braun & Dutto, 2004; Tsatalas et al., 2013), and a reduction in pain and increased ROM has been found with menthol in other work (Johar et al., 2012), then it is reasonable to suggest that a menthol treatment for DOMS induced by running may be an effective intervention. If the application of a menthol treatment restores known DOMS-induced changes to running kinematics to baseline levels, this could allow runners to continue training, despite experiencing mild musculoskeletal soreness. Menthol's ability to reduce pain caused by mild musculoskeletal pain sources (Bishop et al., 2011; 2012; Field et al., 2013; Greenstein et al., 2013; Johar et al., 2012; Sundstrup et al., 2014; Topp et al., 2013; Zhang et al., 2008) could aid in mitigating the potentially damaging effects of DOMS on biomechanical variables in running (Tsatalas et al., 2013; Chen et al., 2009; Dutto & Braun, 2004; Paschalis et al., 2007; Paquette et al., 2017).

In addition to the ability of menthol to cause a greater decrease in pain than ice, there are several risks associated with the direct application of ice (Page & Alexander, 2017). Ice has also been found to cause a decrease in muscle strength whereas a menthol-based topical analgesic did not affect muscle strength in a study comparing placebo, a menthol-based topical analgesic (3.5% menthol) and ice (Topp et al., 2011). While there are cryotherapy modalities that can be affixed to the body, they can be cumbersome and an individual may prefer to not walk around managing a cooling sleeve or wrap. Menthol-based topical analgesics have various application methods (sprays, patches, gels, roll-ons) and little to no risk of harmful side effects

(Page & Alexander, 2017). These factors warrant further investigation into the use of menthol-based topical analgesics as a means of reducing pain in mild musculoskeletal injuries and a better understanding of their influence on biomechanical and/or physiological measures during running needs further attention.

## 1.2 Research gap

There is a large body of research investigating the effects of DOMS on variables of running performance and health (Dutto & Braun, 2004; Braun & Dutto, 2003; Chen et al., 2007a; 2007b; 2009; Eston et al., 1995; McHugh et al., 1999; Tsatalas et al., 2010 Tsatalas et al., 2013; Twist & Eston, 2005; Christina et al., 2001; Dover et al., 2012; Eston et al., 1996; Eston et al., 2000; Mizrahi et al., 2000; Morio et al., 2012; Paschalis et al., 2005; Satkunskiene et al., 2015; Paquette et al., 2017). These studies have investigated the effects of DOMS on gait, both in walking and running, and at various speeds. Research into the effects of menthol on DOMS is limited (Johar et al., 2012), and has focused on isometric contractions, rather than dynamic exercise. The effects of menthol on DOMS-induced changes to running biomechanics and pain have not yet been investigated. This study is the first to investigate the effects of a menthol-based topical analgesic on DOMS-induced changes to running biomechanics. Deviations from an individual's preferred running kinematics can change joint loading and, if uncorrected, the cumulative effects may contribute to tissue damage. Menthol-based topical analgesics can be used as one intervention to mitigate discomfort from DOMS, however, the corresponding effect on running biomechanics remains unclear and is a focus of this thesis.



## **Section 2: Literature review**

### **2.1 Running**

#### **2.1.1 The Anatomy of Running**

Running as a form of exercise is one of the most common types of physical activity globally, being relatively inexpensive and boasting many health benefits (Lee et al., 2017). While there is significant variability between individuals running biomechanics, running involves the coordination of movements between all segments within the kinetic chain (Almeida, Davis, & Lopes, 2015; Dugan & Bhat; 2005). There are two main phases of gait in running, the stance and swing phase. Transitioning between the two main actions within the stance phase of the running gait cycle; braking and propulsion, as well as efficiently coordinating the segments of the opposite lower limb as it moves through the swing phases repeatedly occurs through synchronized movement of the entire musculoskeletal system (Nicola & Jewison, 2012). The many bones, joints, and muscles of the lower limb must work in unison in order to properly propel an individual, with increasing force as they shift from walking to running to sprinting (Novachek, 1998). The predominant phases of running are stance and swing, with a specific-to-running subphase, “double-float” occurring at the beginning and prior to the termination of swing in the same stride (Novachek, 1998).

The stance phase is initiated when one foot makes contact (initial contact / foot strike) with the ground. The individual then moves through weight-acceptance onwards to mid-stance where the foot is flat on the ground, under the centre of mass - by this point in the stance phase, the “braking” action of absorbing and stabilizing the body

has been achieved and the individual shifts into the “propulsion” action where the heel begins to lift as the individual pushes off the forefoot propelling the body forward (Novachek, 1998; Dugan & Bhat, 2005).

The swing phases is initiated once the individual has reached the terminal portion of propulsion, known as the toe-off subphase. Once toe-off has been completed, the limb is considered in the swing phase, while the one limb has just completed the stance phase, the other has still yet to complete the swing phase; this ultimately results in the running-specific subphase “double-float” where neither foot are in contact with the ground, truly differentiating running from walking (Novachek, 1998; Dugan & Bhat, 2005). As the limb moves through the swing phase, the limb will shift between initial and terminal swing to a “mid-swing” subphase, identified when potential and kinetic energy peak (Novachek, 1998). It can also simply be identified as the mid-point of the swing phase. As the limb moves towards terminal swing, a second “double-float” phase occurs, this time the limbs are positioned opposite the first occurrence. As the foot contacts the ground, the following foot strike (subsequent foot contact) ends one gait cycle (Novachek, 1998). Table 1 has a brief overview of the phases and subphases of running gait.

**Table 1.** Normalized Gait Cycle: this table includes the phases of a single running stride with the percentage of time each requires from start to finish.

Running Gait Cycles						
	Stance (~40%)			Swing (~60%)		
Float Phase:				***		***
Phase:	<i>Initial Contact</i>	<i>Mid-Stance</i>	<i>Toe-Off</i>	<i>Initial Swing</i>	<i>Mid Swing</i>	<i>Terminal Swing</i>
Percent Stride (Normalized to 100%):	0-10%	10-30%	30-40%	40-60%	60-80%	80-100%

The musculoskeletal requirements of running are extensive and require coordinated movement of the entire musculoskeletal structure, however the predominant focus of this study is on the lower limb and is the focus of this literature review.

The skeletal structure of the lower limb includes the longest bone in the body, being the femur, as well as one of the most intricate segments of human bone structure in the entire body, the foot (Lieberman et al., 2010). In combination with the bones of the lower leg (fibula and tibia) as well as the pelvic structure and patella, create the “platform” for which the muscles of the lower limbs act in order to propel the body during running (Dugan & Bhat, 2005). The foot is the contact point between the running surface and the rest of the musculoskeletal system. It can be aided and affected by the rest of the musculoskeletal system involved in running gait and requires a great deal of coordination in order to maintain a healthy state (Dugan & Bhat, 2005). The aforementioned skeletal structure joins to form the major joints of the lower limb being the hip (acetabulofemoral), knee (tibiofemoral), and ankle (talocrural) joints. These bones and joints are controlled by a complex network of muscles, tendons, ligaments, and fascia that ultimately work together to propel and stabilize the lower limbs

(Lieberman et al., 2010; Dugan & Bhat, 2005). The muscles required for absorption (braking) and propulsion include the hip flexors and extensors, knee flexors and extensors, and ankle plantarflexors and dorsiflexors (Novachek, 1998). The abductors/adductors, internal/external rotators of the hip, as well as the evertor/invertor musculature of the ankle aid in stabilizing their respective segments and joints as well as the knee to assist forward propulsion (Nicola & Jewison, 2012).

Structures of the lower limb worthy of mention for their role in gait are the achilles tendon and plantar fascia. The achilles tendon and the plantar fascia work together during stance to reduce impact forces and store elastic energy and aid in propelling an individual from stance to the swing phase (Nicola & Jewison, 2012). This “energy-return” system is enhanced during a FFS due to the greater forces the foot and ankle are exposed to, which also puts them at a greater risk for injury (Novachek, 1998; Nicola & Jewison, 2012; Almeida, Davis, & Lopes, 2015; Paquette, Zhang, & Baumgartner, 2012).

While the activity of running is seemingly straight forward, the coordination required to run efficiently is rather complex. The joints of the lower limb work in unison during running gait in order to complete two opposing movements at the same time. While the one limb is moving through stance, the other moves through the swing phase and largely different demands are placed on the joints during each phase. The ankle, knee, and hip joints work together in both phases with the main goal of limiting ground reaction forces transmitted through the body and producing forward movement as efficiently as possible. Depending on foot strike pattern, ankle, knee, and hip actions change resulting in a shift in the loads placed on the musculoskeletal structure

(Almeida, Davis, & Lopes, 2015). A FFS/MFS results in a more plantarflexed ankle and more flexed knee compared to a RFS at initial contact while running (Almeida, Davis, & Lopes, 2015). Foot strike pattern has not been identified to have any effect on hip ROM while running however the hip plays a large role in stabilizing the knee and providing a great deal of propulsion through hip extension during toe off (Almeida, Davis, & Lopes, 2015; Riley et al., 2008).

Our study design revolves around sagittal plane kinematics and thus the focus of this literature review is centered on the musculature that move the joints of the lower limb in the sagittal plane. The primary musculature for these movements has been identified in a table below to simplify reading. The musculature, their actions, and role in running gait have been identified below from two reviews on the biomechanics of running (Novachek, 1998; Dugan & Bhat, 2005).

**Table 2.** muscles and actions of sagittal plane movements during running

Ankle	Plantarflexion	Gastrocnemius (medial/lateral), soleus
	Dorsiflexion	tibialis anterior
Knee	Flexion	hamstrings (biceps femoris, semitendinosus, semimembranosus)
	Extension	Quadriceps (vastus medialis/lateralis/intermedius), rectus femoris
Hip	Flexion	iliopsoas, tensor fascia latae
	Extension	gluteus maximus/minimus/medius, hamstrings

During the stance phase, much of the work being completed by the lower limbs is eccentric in nature with the ankle, knee, and to a lesser extent, the hip working in unison to stabilize the foot under the body. During early stance, the ankle moves into dorsiflexion and the knee flexes in order to reduce vertical impact forces, the hip moves

into extension as the foot shifts under the body towards midstance. As the individual moves through mid stance, the hip continues to extend, and the knee and ankle shift to extension and plantarflexion, respectively. As the individual reaches toe off, the hip and limb lifts from the ground and enters the swing phase - the hip and knee transition from extension to flexion, collectively. The ankle has no load to bear or force to produce during swing (Novachek, 1998), and simply shifts from plantarflexion into dorsiflexion in an attempt to clear the ground and prepare for the subsequent foot strike at the appropriate position for the intended foot strike position. While moving through swing, the hip and knee continue to move through flexion until positioned to receive the mass of the individual and provide impact absorption on the following foot strike.

### **2.1.2 Foot Strike patterns**

The invention of the modern running shoe has led to a shift in the way that runners strike the ground when they run (Davis, Rice, & Wearing, 2017). Before the modern running shoe with its large cushioned heel, the impact forces caused by making contact with the ground heel-first would have sent overwhelming forces through the bones and joints - runners made contact with the ground using the middle or front of the foot in order to use the ankle joint to increase shock absorption and reduce the ground reaction forces experienced (Lieberman et al., 2010). This has resulted in three foot strike patterns an individual may use; heel (or rearfoot - RFS), midfoot (MFS), or forefoot strike (FFS), where the part of the foot that contacts the ground first defines the foot strike pattern (Lieberman et al., 2010). The implications foot strike pattern have on kinematic and kinetic parameters of gait is significant, as the segment of foot

that contacts the ground first changes, so do the demands placed on the musculoskeletal system creating foot strike-specific risks for injury on an individual (Lieberman, 2010; Davis, Rice, & Wearing, 2017; Almeida, Davis, & Lopes, 2015). An understanding of how foot strike pattern affects kinematic and kinetic parameters of gait is important for any researcher, clinician, coach, or individual interested in the implications for performance and injury and will be touched on briefly in this review. Midfoot and forefoot strike patterns are commonly grouped together as there are very few if any differences in the kinematic and kinetic parameters between the two (Boyer, Rooney, & Derrick, 2014). Surprisingly, there are entire reviews on the biomechanics and analysis of running gait that have no mention of foot strike patterns between runners, the assumption that all runners have identical foot strike patterns is a large oversight for any one interested in the performance and injury implications of foot strike patterns on gait (Dugan & Bhat, 2005).

The RFS is the most common modern-day foot strike pattern seen in distance runners with many estimates of 75 to 95% of runners using this method (Novachek, 1998; Lieberman, 2010; Davis, Rice, & Wearing, 2017). The remaining individuals use either a MFS or FFS, This results in a more plantarflexed foot at contact with more compliance through weight acceptance as the ankle and foot work to slow and stabilize the individual as they move through stance (Lieberman, 2010; Davis, Rice, & Wearing, 2017) whereas a RFS in contrast lands with a more extended knee and dorsiflexed foot, resulting in greater ground reaction forces experienced and greater deceleration demands placed on the knee extensors to slow and stabilize the individual (Davis, Rice, & Wearing, 2017). These findings suggest that a RFS-runner is exposed to higher

magnitude and rates of change in vertical impact forces results in significantly higher risk and rates of MSK injuries, where a MFS/FFS, with greater loads placed on the foot and ankle result in greater injuries to the foot and ankle (Almeida, Davis, Lopes).

### **2.1.3 Segment and joint variability in running gait**

Variability has been identified as predictor of injury in a running population (Hamill, Palmer, & van Emmerik, 2012). Coordinative variability is the variability of the interaction between segments or joints - low coordinative variability, as well as excessively high coordinative variability has been identified as the unhealthy or pathological state in running gait, where healthy runners tend to be in a state of higher coordinative variability (Hamill, Palmer, & van Emmerik, 2012). Whether or not being in a state of low variability is the result of, or cause of injury has not been identified but the ability to adapt during a dynamic movement such as running to changes is considered to be healthy and functional (Hamill, Palmer & van Emmerik, 2012). Hamill, van Emmerik, and Heidersheit (1999) hypothesized that lower variability of segment and joint couplings in an injured population indicated repeatable joint actions within a very narrow range - suggesting reduced range of motion in gait could be responsible for injury or injury could result in the reduction in variability and result in the individual shifting to a pathological or unhealthy state. Being in this state of low variability, suggests not only current injury but may suggest there could be further stress on soft tissues of the MSK (Hamill, van Emmerik, & Heidersheit, 1999).



## **2.2 Delayed Onset Muscle Soreness**

### **2.2.1 Causation and Mechanisms of Action**

The pain caused by DOMS is a result of the stimulation of group III and IV sensory neurons in the muscles (Armstrong, 1984; Connolly, Sayers, & McHugh, 2003). These sensory neurons are believed to be activated by the mechanisms of the tissue fluid theory, wherein intramuscular osmotic pressure is increased in the affected area, sensitizing the sensory nerves. Both Armstrong (1984) and Smith (1991) have suggested that an inflammatory response stimulates group III and IV sensory neurons causing pain. The muscular pain associated specifically with DOMS occurs at approximately 24-48 hours after the exercise induced muscle damage that resulted in DOMS and symptoms associated with DOMS can last anywhere from 5-10 days after the initial activity, depending on a multitude of variables (Armstrong, 1984; Cheung, Hume, & Maxwell, 2003). Symptoms of DOMS typically peak at 48 hours after the EIMD has occurred (Cheung, Hume, & Maxwell, 2003).

The mechanisms of DOMS have been well studied, however a single conclusion on what causes DOMS has not been definitively reached (Cheung, Hume, & Maxwell, 2003). Cheung, Hume, & Maxwell, (2003) conducted a review of DOMS literature which included the six most probable mechanisms of DOMS and proposed a potential timeline of events causing DOMS. These six potential mechanisms are the lactic acid, muscle spasm, connective tissue damage, muscle damage, inflammation (tissue fluid), and enzyme efflux theories.

1. Lactic acid: This theory has largely been disproven by a malalignment with the DOMS timeline of pain severity (Schwane et al., 1983), wherein DOMS intensity peaks at 24-48 hours, lactic acid accumulation peaks and returns to resting within 1 hour of exercise completion. The theory suggested that the production and circulation of a toxic metabolic waste localized to the affected musculature would result in pain from DOMS (Cleak & Eston, 1992).
2. Muscle Spasm: This theory proposed by de Vries (1961) suggests that a cycle occurs in the days following EIMD wherein a decrease of blood flow to an exercising muscle would lead to the release pain substances, if enough of the substances were released, pain receptors in the affected muscle would be stimulated and cause muscle spasms, resulting in further ischemia, furthering the cycle. This theory has been contested as a resulting increase in EMG activity - “muscle spasm” (Talag, 1973; Abraham, 1977; Newham, Jones, & Edwards, 1983), and the relation between increased EMG activity and soreness perception is lacking or non-existent (Bobbert, Hollander, & Huijing, 1986).
3. Connective Tissue Damage: The excretion of amino acids hydroxyproline (HP) and hydroxylysine (HL) through urine are indications of collagen degradation from overuse and / or strain damage (Stauber, 1989; Abraham, 1977). These amino acids have been measured in relation to DOMS related soreness but have provided mixed or insignificant results (Abraham, 1977; Gissal & Hall, 1983, Horswill et al., 1988). More recent research has indicated that the timelines of peak HP and HL concentrations correlate to peak DOMS

soreness, approximately 48 hours after exercise (Sydney-Smith & Quigley, 1992). A majority of connective tissue damage initially occurs near the myotendinous junction (MTJ) where the oblique arrangement of fibers has less resistance to force than strict muscle or tendon fibers (Noonan & Garrett, 1992; Tidball, 1973). A high concentration of muscle nerve endings in the MTJ explains the initial concentration of localized distal tenderness in an affected muscle (Newham et al., 1982).

4. Muscle Damage: While this theory relates to the initial EIMD that leads to DOMS, muscle damage occurs due to a disruption of the sarcomere at the z-lines (Hough, 1902). The high forces produced by eccentric muscle action overload the tissue and damage the sarcomere structure (Friden, Sjöström, & Ekblom, 1983; Friden & Lieber, 1992). Nociceptors within the muscle fiber connective tissues and throughout the muscle to the myotendinous junction are stimulated causing EIMD pain (Cheung, Hume, & Maxwell, 2003). Increased plasma Creatine Kinase (CK) is indicative of an increase in tissue permeability in the muscle (Cleak & Eston, 1992), which suggests damage to the structural components of the muscle (Z-lines, Sarcolemma) as CK permeates into the interstitial fluid (Cheung, Hume, & Maxwell, 2003). There is a large body of evidence that suggests there is a mismatch in timelines between CK peak concentrations and DOMS, which suggests that muscle damage is only part of the mechanism behind DOMS (Newham, Jones, & Edwards, 1983; Newham, Jones, & Edwards, 1986; Walsh et al., 2001; Evans

et al., 1986; Clarkson et al., 1986; Clarkson et al., 1987; Clarkson & Ebbeling, 1988).

5. Inflammation (Tissue Fluid Theory): Due to a disparity of timelines between inflammatory cell filtration and peak DOMS, and a more aligned timeline between peak oedema and DOMS, researchers have leaned more towards this mechanism being the “Tissue Fluid Theory” over the Inflammation Theory (Gulick & Kimura, 1996). The increased osmotic pressure within the muscle due to oedema leads to the sensitization of group IV sensory neurons which cause a pain sensation. While the oedema appears to be more closely correlated than the inflammatory response in causing pain, some research has suggested that the inflammatory response produces macrophage accumulation at the site of injury which causes the stimulation of group III and IV sensory nerves on a similar time course to the peak severity of DOMS (Armstrong, 1984; Smith, 1991). Following EIMD, the structural damage incurred leads to an inflammatory response that releases proteolytic enzymes within the muscle that breakdown lipid and protein structures leading to further breakdown of damaged muscle fibers and connective tissue (Cheung, Hume, & Maxwell, 2003). The release of proteolytic enzymes and a localized accumulation of inflammatory mediators leads to the attraction of monocytes and neutrophils. Monocytes mature into macrophages that accumulate in the area of the injury, potentially causing pain.

6. Enzyme Efflux: Gulick and Kimura (1996) proposed the enzyme efflux theory, in which calcium typically stored in the sarcoplasmic reticulum (SR) accumulates in damaged muscles after the sarcolemmal damage negates the ability for calcium to reuptake. This accumulation of calcium causes Adenosine Triphosphate (ATP) production to slow, which is also required for calcium reuptake into functioning SR. ATP production is caused due to calcium slowing cellular respiration to occur at the mitochondrial level (Cheung, Hume, & Maxwell, 2003), the accumulated calcium leads to a localized inflammatory response. This inflammatory response produces substances which further degrade structural proteins (z-lines) in the muscle and cause a chemical stimulation of sensory nerve receptors, causing pain (Armstrong, 1984; 1990).

No one single theory has been proven to solely cause DOMS - research has steered away from some of these theories as major contributors as the timelines of events of the mechanism and DOMS do not meet, however it has begun to define a timeline of events as they occur and how they might correlate to DOMS (Cheung, Hume, & Maxwell, 2003). Connolly et al., (2003) notes that the pain is a direct result of the damage to sarcomeres that worsens after an inflammatory response to the damage in a cyclical fashion.

Cheung, Hume, and Maxwell (2003) created an integration of the models proposed by Armstrong (1984; 1990), Smith (1991), and Smith and Jackson (1990) that details how damage is incurred and how the current theories interact to cause pain perception.

1. Connective tissue and muscle damage theory: eccentric muscle activity during exercise produces high tensile forces that damage the myotendinous junction of a muscle and the surrounding connective tissue and muscle fibers.
2. Enzyme efflux theory: damaged sarcolemma results in higher concentrations of calcium in the sarcolemma that inhibits cellular respiration. ATP production is reduced by the presence of calcium and homeostasis is lost within the muscle. Enzymes activated by the presence of the calcium lead to the destruction of sarcomere z-lines, troponin, and tropomyosin (further damaging of muscle fibers)
3. Inflammation theory: Within hours of exercise completion, an inflammatory response occurs within the damaged area resulting in an increase in circulating neutrophils.
4. Inflammatory theory: Macrophage, mast cells and histamine production are activated and / or increased in the damaged area
5. Inflammatory theory: Monocytes/macrophages increase over the course of the initial 48 hours and lead to notable increase in pain, caused by the stimulation of group III and IV nerve endings by the macrophage production of prostaglandin (PGE<sub>2</sub>)
6. Inflammation theory: The inflammatory response, along with increased localized oedema (tissue fluid theory) and increased local temperature may activate nociceptors within the muscle fibers and the myotendinous junction, further increasing pain sensation.

Currently, the above review revolves heavily on the inflammatory response to initiate the sensation of pain caused by DOMS, however, Cheung, Hume and Maxwell (2003) also note that this theory remains controversial in the literature. What is definitive is that there is a phenomenon in which eccentric exercise leads to DOMS - the sensitizing of group III and IV nerve endings which, depending on severity, leads to a host of symptoms that can lead to pain perception and altered movement patterns resulting in secondary injuries.

### **2.2.2. Effect on Biomechanical Variables**

Previous research has suggested that DOMS can lead to significant changes to running mechanics including reductions in knee and ankle ranges of motion throughout the gait cycle (and importantly during stance), such that an increase in vertical leg stiffness leads to greater ground reaction forces impacting lower limb joints as well as throughout the spine (Dutto & Braun, 2004). The knee has been identified as a major variable in DOMS-induced running by Dutto and Braun (2004) - the musculature of the knee which is a heavily involved joint in running, typically experiences greater effects of DOMS. During initial contact leading into mid-stance, the knee extensors are working to stabilize the knee and work eccentrically as the knee flexes slightly while the lower limb passes under the body and moves towards the terminal stance phase. This eccentric load is exaggerated during downhill running and under fatigue when intensity or duration is greater than one's capacity (Eston, Mickleborough, & Baltzopoulos, 1995). Once EIMD is induced in the knee extensors, their ability to stabilize the knee and

control the stance phase of motion are reduced, this factor as well as increased knee stiffness, the avoidance of pain, and reduction of force production can lead to increased chances of secondary running related injuries or further damage to already affected structures (Dutto & Braun, 2004; Tsatalas et al., 2013). These greater impact forces can lead to increased rates of joint disorder and further, more severe injury if an individual continually runs at normal intensities with EIMD-induced changes to gait (Dutto & Braun, 2004). While DOMS is not permanent damage, the damage sustained while running with altered mechanics could very easily be. Due to the reduction in shock attenuation caused by DOMS, a cumulative increased joint stress can lead to premature wearing of joint structures (Dutto & Braun, 2004), assuming sufficient recovery is not met. Muscle recruitment patterns may also be altered by DOMS - affected musculature may not activate in the typical pattern during locomotion and may lead to the loss of joint stability, exposing the joint to a greater risk of acute injury (Tsatalas et al., 2013).

### **2.2.2 Measuring Delayed Onset Muscle Soreness**

There are many effective measures for identifying and quantifying DOMS (Connolly, Sayers, & McHugh, 2003). The primary methods for identifying and quantifying DOMS are maximum voluntary isometric contraction strength (MVC's), peak torque, Creatine Kinase activity (CK), myoglobin concentration (Mb), tenderness, pressure threshold, ROM, and self-reported perceived muscle soreness through various pain scales.



The simplest, quickest, and yet still meaningful tools to measure DOMS include pressure threshold and perceived muscle soreness. While bias can devalue self reported measures, pressure threshold - the minimal pressure (force) required in order to induce pain (Fischer, 1987) has been used extensively in research to assess pain and can produce the necessary reproducibility and validity required to measure DOMS accurately enough, concurrently with self-report measures to conclude DOMS is induced in the targeted musculature (Connolly, Sayers, & McHugh, 2003).

Connolly, Sayers, and McHugh (2003), include a table in a review of DOMS treatment literature outlining the damage information, cost, difficult of measure, and reliability of nine different indices of muscle damage. Of the nine reviewed, muscle biopsy provides the highest reliability and is the best indicator of DOMS however, strength, pain, and tenderness all warrant medium-high reliability with low cost and measurement difficulty. While both pain and tenderness have high subjectivity, they still provide some insight into the effectiveness of a DOMS-inducing protocol.

### **2.2.3 Inducing Delayed Onset Muscle Soreness**

In order to effectively research DOMS, DOMS must be induced in a laboratory setting in order to control extraneous variables not otherwise controlled if it were self-induced. Depending on the muscle(s) targeted for research, various protocols are used in order to induce DOMS (Tsatalas et al., 2013). Previous research aiming to measure the effects DOMS has used an eccentric-biased or purely eccentric exercise, normally targeting the elbow flexors or extensors/flexors of the knee (Johar et al., 2012; Tsatalas

et al., 2013), such as using isolated eccentric-biased single joint exercises, isokinetic dynamometry, cycle ergometry, high force eccentric exercise, or downhill running.

Isolation protocols, such as exclusively targeting the knee extensors in inducing DOMS, are effective in finding exact answers from an intervention such as whether a treatment like anti-inflammatories have any effect on any of the various DOMS symptoms (Tsatalas et al., 2013). While isolation protocols can be useful for specific questions, using an isolation protocol on a gait-based study lacks real world application. Exercise that leads to DOMS (downhill running, marathon racing) won't result in localized soreness to one muscle, especially with such wide variability in running mechanics. Thus, making conclusions based on isolation-based protocols and the ensuing interaction between localized DOMS-induced musculature and gait-based outcomes lacks application to real world athletes. While dynamic protocols, such as downhill running or box-stepping lack the targeted specificity of an isolation protocol, they can provide more significant information for the end-user of the findings (athletes, exercisers). Downhill running has been shown to induce significant DOMS in the knee extensor and plantarflexor musculature due to the increased eccentric loading placed on both muscle groups and are the primary focus of our study design (DOMS measurement, intervention application: Chen et al., 2007a; 2007b; 2009; Dutto & Braun, 2004).

#### 2.2.4 Treatment of Delayed Onset Muscle Soreness

There has been a wide array of treatment options investigated to mitigate the effects of DOMS, however, many of these options have proven to be ineffective or inconclusive. Some of the more well researched treatment options as per the review by Cheung, Hume, & Maxwell, (2003) include cryotherapy, stretching, anti-inflammatory drugs, ultrasound, electrical current techniques, homeopathy, massage, compression, hyperbaric oxygen, and exercise. Of these therapies, the most effective option seems to be exercise, although it's noted that the benefits only last for the duration of exercise and studies have produced mixed results. It's necessary to point out that in all of these studies, various DOMS-inducing protocols are used and variability of administration (time, type, dose, etc.) of therapeutic interventions exist and could have led to inconsistency in results. The review by Connolly et al., (2003) suggests that many of the current treatment options for DOMS, that have been researched, are at best inconclusive in their findings, however note that further, well-controlled research should be conducted to provide more accurate insights into the effects of many treatment options.

Johar et al., (2012) compared the effects of ice and a menthol-based topical analgesic (Biofreeze®, 3.5% menthol) on variables of muscle activity and pain in DOMS-induced elbow flexors and found significant positive results (increased tetanic contraction, decreased pain) for its use. While Johar et al., (2012) utilized an isolation protocol for inducing pain (isolated eccentric elbow extension), this approach yields real world results in an upper limb focused study as DOMS localized in the elbow flexors

is a common occurrence in exercisers. The use of isometric and evoked contractions in the elbow flexors doesn't translate well to the dynamic movement of running therefore investigating the effects of a menthol-based topical analgesic on generalized lower limb DOMS on running biomechanics may provide different results than previous work.

## **2.3 Menthol**

### **2.3.1 Mechanism of Action**

A systematic review by Page and Alexander (2017), reviewed research outlining the mechanism of action for menthol and its overall clinical efficacy in treating musculoskeletal pain. In the systematic review, the mechanism of menthol pain reduction was identified as a form of cryotherapy. The specific thermal receptor for cold sensation is identified as the Transient Receptor Protein - Melastatin 8 (TRP-M8). TRP-M8 receptors are sensitized when exposed to temperatures ranging from 30°C and 8°C, as well as menthol, the active ingredient in Biofreeze. Menthol sensitizes TRP-M8 receptors and creates a "cold" sensation from sensory neurons in the skin. Due to menthol activating the same receptors as ice, it has much the same effect as ice, without potentially harmful effects of overexposure to ice, such as frostnip/bite and muscular stiffness. Both pain and temperature sensations ascend the spinal cord towards the thalamus along the spino-thalamic tract. It is believed that, in mild forms of musculoskeletal pain, menthol, along with its local effects, overrides the pain signal

being sent to the thalamus and the “cold” sensation is perceived over the pain - This is also known as the gate control theory, proposed by Melzac and Wall (1965).

### **2.3.2 Effect on Biomechanical Variables**

The effect of menthol on DOMS-induced changes to biomechanical variables has been investigated, but not under the dynamic requirements of running. Research by Topp et al., (2017) suggests that menthol had no significant change in ROM over a placebo group when applied to individuals with mechanical neck pain prior to cervical manual manipulation. While this study showed no benefit of menthol application for cervical spine ROM, the use of menthol for mechanical neck pain is a very different mechanisms of pain than for use with muscles experiencing DOMS. The research conducted by Johar et al., (2012) suggests menthol had a significant positive treatment effect on DOMS, unfortunately, changes in ROM (such as a joint position sense test) were not assessed.

### **2.3.3. Risk of Injury - Exercising with DOMS and Menthol**

Exercising with DOMS can be an unpleasant experience and may lead to exercise avoidance in novice exercisers and/or potential performance decrements to experienced exercisers and athletes. Novice and expert athletes experiencing soreness while attempting to exercise may seek out pain relief from a variety of sources. In studies where a topical analgesic was used to block the pain associated with DOMS and

participants were asked to exercise 48 hours after the induction of DOMS (Johar et al., 2012), or perform movements after the application of menthol in participants with chronic pain, no further injury was reported (Page & Alexander, 2017; Sundstrup et al., 2014; Topp et al., 2017; Zhang et al., 2008). Based on the current literature, there are no concerns that light to moderate exercise with DOMS under proper supervision is likely to cause further injury to the already affected musculature. Despite this, it should be noted that no studies have looked at the long term, cumulative effect of running with DOMS. This review has identified that DOMS can change an individual's typical/preferred running kinematics. A longitudinal study is needed to identify the likelihood that running consistently with DOMS may lead to an increased injury risk, or performance decrement. Furthermore, using a downhill running protocol to induce DOMS was found to be an effective means of inducing DOMS and no major injuries are noted in any of these studies by way of the downhill running protocol (Chen et al., 2007a; 2007b; 2009; Dutto & Braun, 2003; Dutto & Braun, 2004; Eston et al., 1996; Eston et al., 2000; Eston et al., 1995).

## Section 3: Study

### 3.1 Research Questions

1. Does a topically applied menthol-based analgesic affect delayed onset muscle soreness induced changes to running kinematics?
2. Does a topically applied menthol-based analgesic affect delayed onset muscle soreness induced changes to spatio-temporal parameters?
3. Does a topically applied menthol-based analgesic affect delayed onset muscle soreness induced changes to perception of pain?

## 3.2 Hypotheses

There will be a decreased ROM in running kinematics associated with DOMS and the application of menthol will return ROM to baseline (Dutto & Braun, 2004; Tsatalas et al., 2013; 2013; Chen et al., 2007a; 2009). Specifically, the application of menthol will result in an increase in ankle dorsiflexion during stance (and consequentially an increased ROM throughout gait cycle), and an increase in knee ROM during stance and swing phases (compared to the DOMS trial).

Where a reduced ROM caused by DOMS typically causes a shift in the stride length - frequency paradigm (a negative correlation) (Braun & Dutto, 2003), the application of menthol will increase stride length, thereby reducing stride frequency. These interactions may be produced in part or whole by the physiological reaction and reduction of pain perception from the mechanisms of menthol.



### 3.3 Methods

#### 3.3.1 Participants

Twenty participants (12 Females, 8 Males) volunteered for this study. Inclusion criteria were a minimum running experience of 20 kilometres per week averaged over the last 6 months, with no lower body injuries over the last 12 months. The twenty participants were randomly selected to be placed in a Placebo (PLA; n=10) or a Menthol (Biofreeze®, 3.5% menthol; BIO; n=10) gel group.

Participants first read an informed consent (Appendix 8.1), and once questions were answered and clarifications were made, the document was signed. Additionally, participants had the choice to sign a consent to have photographs and video taken (beyond motion capture for document or presentation purposes). Prior to testing, the participant was informed they must bring the same pair of running shoes to both days in order to minimize any extraneous effect different shoe structure can have on running mechanics (Nigg et al., 2006). Further, participants had the protocol verbally communicated and had the opportunity to ask any outstanding questions prior to each interaction. Participants were familiarized with the lab space and the treadmill used for all analysis, as well as the downhill running protocol. The study was approved by the Brock University Research Ethics Board (REB #17-125) as per the standards of declaration of Helsinki.

### 3.3.2 Instrumentation

Following informed consent and baseline measures (age, height, weight, average weekly mileage, baseline pain analyses - pressure threshold, subjective analysis), participants were instrumented for three-dimensional motion capture. Three-dimensional kinematics were collected using a 10-camera Vicon motion capture system (Vicon, Oxford, UK) sampled at 330Hz. Each participant was instrumented with passive reflective markers (Figure 1) that were placed on anatomical landmarks identifying the foot, shank, thigh, pelvis, and torso segments independent of each other. Specifically, individual markers were placed on the 2<sup>nd</sup> and 5<sup>th</sup> distal metatarsal heads, calcaneus, medial and lateral malleoli, medial and lateral femoral epicondyles, greater trochanters, superior iliac crests, acromion processes, posterior and anterior superior iliac spines. Custom made rigid bodies, consisting of four reflective markers each were placed on the torso (posterior), trunk (posterior), thigh (lateral), and shank (lateral) segments. Assuming a fixed spatial relationship of the rigid bodies to the calibration markers, the calibration markers were removed following a static calibration trial. All markers were attached to the participants skin or tight-fitting clothing using double sided tape. All necessary precautions were taken to mitigate as much motion artifact from clothing or skin.



**Figure 1.** The reflective markers on a subject used to identify anatomical landmarks and segments. Torso markers are not pictured here.

The laboratory global axis system was defined as X (anterior/posterior), Y (medial/lateral) and Z (vertical). Following standard calibration procedures, participants were asked to step onto the treadmill for a static and motion calibration. Static calibration was used to identify limb segments and to define local coordinates systems from the global (laboratory coordinate system). Once the static calibration was collected a dynamic calibration was performed. The dynamic, or motion calibration required the subject to move through a series of movements that would be expected during recording to ensure the cameras can capture the markers moving through the required space. Each participant performed the following movements 3 times on each leg: hip flexion / extension, hip circumduction, knee flexion / extension, and ankle circumduction while standing on the treadmill. Motion calibration was also used for later refinement of functional joint axes in Visual3D (C-motion Inc., Germantown, MD, USA) - see data analysis section.

Following the calibration, calibration markers were removed, and the participant was given time to warm up on the treadmill with tracking markers in-place, so they could become accustomed to running under experimental conditions (Figure 2).



**Figure 2.** The lab capture volume including the suspended railing system and motion capture cameras mounted in optimized positions.

### 3.3.3 Experimental Protocol

Participants visited the lab for 2 experimental sessions, separated by approximately 48 hours (Figure 3). On day 1, participants performed a baseline (BASE) data collection session, followed by a DOMS-inducing protocol (DOMS). On day 2, participants performed the same baseline session, followed by an intervention (INT) session (either placebo or intervention, depending on the group). This is summarized in figure 3 and more details on the data collection for each sessions is broken down below.

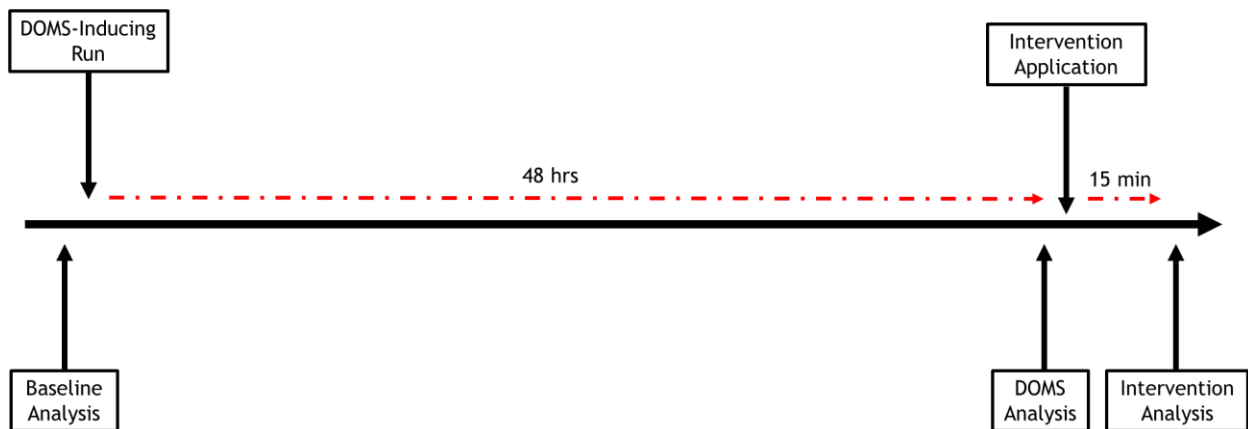


Figure 3. A timeline of study protocol.

#### Day 1: Baseline Analysis

At the start of each session, pressure threshold was measured at two sites bilaterally (four sites total) using a pressure algometer with a 0.785cm<sup>2</sup> pressure applicator (Wagner Model FDIX Force One Pressure Gauge, Wagner Instruments,

Greenwich, CT, Figure 4). All indentations were taken by the principal investigator in order to increase reliability and standard instructions were given to participants to indicate the moment they felt pain as the investigator increased pressure at approximately 1kg/s as per the clinical practice pressure threshold guideline outlined by Fischer (1986). The four sites include: vastus medialis oblique (VMO) and the myotendinous junction (MTJ) of the gastrocnemius-achilles complex, bilaterally as these sites are known to be affected by downhill running (Eston, Mickleborough, & Baltzopolous, 1995).












Each site was measured five times: two initial measures to accustom each participant to the protocol then the last three samples were averaged at each site. Participants were instructed to sit upright, ankles, knees, and hips at 90° and remain relaxed during measurements. Pressure threshold measurements were recorded in newtons (N). Pressure threshold analysis occurred directly prior to the participant initiating each running analysis. The measurements were taken in a sequenced order, one measure at each site from left VMO, right VMO, left GA-MTJ, right GA-MTJ, repeated five times in a cyclical fashion.



**Figure 4.** The Pressure Gauge used for this study, a Wagner Instruments FDIX Force One pressure algometer (Wagner Instruments, Greenwich, CT)

Once participants were adequately warmed up, a baseline running analysis was completed. Participants ran at 2.5, 3.0, and 3.5m/s (Appendix 8.2) as running gait parameters are speed dependent (Tsatalas et al., 2013). The participant was given the first minute to obtain the first running speed which was completed in random-order for each participant (maintained order for each analysis). Three 15 second samples were recorded during each running speed; these samples were taken at 30 seconds, 90 seconds, and 150 seconds. Barring no recording issues, the second sample was then used for all analysis. While running at each speed, a subjective pain analysis scale - the comparative pain scale (CPS; Figure 5) was affixed to the treadmill for each participant to report their pain level during running motion capture.

### COMPARATIVE PAIN SCALE CHART (Pain Assessment Tool)

										
0 Pain Free	1 Very Mild	2 Discomforting	3 Tolerable	4 Distressing	5 Very Distressing	6 Intense	7 Very Intense	8 Utterly Horrible	9 Excruciating Unbearable	10 Unimaginable Unspeakable
<b>No Pain</b>	<b>Minor Pain</b>			<b>Moderate Pain</b>			<b>Severe Pain</b>			
Feeling perfectly normal	Nagging, annoying, but doesn't interfere with most daily living activities. Patient able to adapt to pain psychologically and with medication or devices such as cushions.			Interferes significantly with daily living activities. Requires lifestyle changes but patient remains independent. Patient unable to adapt pain.			Disabling; unable to perform daily living activities. Unable to engage in normal activities. Patient is disabled and unable to function independently.			

**Figure 5.** Comparative Pain Scale used to identify the amount of pain experienced during running analyses. Participants had each description read to them and were asked to report their pain level. Half scores (ie. 3.5) were accepted.

#### Day 1: DOMS-Inducing Protocol

Once the baseline running analysis was completed, reflective markers were removed and participants were given time to prepare themselves (redress if preferred, drink water, bathroom, etc.) for the DOMS-inducing protocol. Once participants were ready, they were brought to a second treadmill prepared specifically for the DOMS-inducing protocol. This protocol simulated a downhill run, with the treadmill positioned on a decline slope. The treadmill was set on a platform placed under the rear legs of the treadmill to create a decline angle of  $-10^\circ$ . Participants were given time to familiarize themselves with the sensation of being on the treadmill as it was a novel situation for all participants. Once ready, participants were instructed to run on the treadmill for 30-minutes at roughly tempo pace or  $\sim 85\%$  of their heart rate maximum. Once completed, the participant was asked to remain in the lab until their heart rate returned to  $<100\text{BPM}$ . Average downhill run speed was  $246.5 \pm 34.1\text{m/min}$ .



### *Day 2: DOMS Analysis*

48-hours after DOMS-inducing protocol, participants returned to the lab, for a protocol similar to the baseline collection. This again included marker placement and calibration, removal of calibration markers, and measurement of pressure thresholds at the same sites under the same conditions. The running protocol was identical to the day 1 baseline collection, including specific randomized order of speeds.

### *Day 2: Intervention - Menthol / Placebo Analysis*

After the day 2 DOMS analysis, participants rested in a chair while the menthol-based topical analgesic, Biofreeze (3.5% menthol) or a placebo gel (created by Biofreeze®, product in same container with no menthol) was applied over the knee extensor and plantar flexor musculature, pending no open wounds as per the guidelines provided by Biofreeze. The recommended dosage of Biofreeze (3.5% menthol) is 1mL/200cm<sup>2</sup> of application area. Gel was applied to the maximum accessible area of the knee extensors and plantarflexor musculature as they are the musculature primarily affected by a DOMS-inducing downhill run (Dutto & Braun, 2004). Both menthol and placebo gels were applied in the same quantities. Gels were applied to the anterior thigh at approximately 4.5% of total body surface area (800cm<sup>2</sup>), equal to 4ml of gel. Gels were applied to the posterior shank at approximately 3.5% of total body surface area (600cm<sup>2</sup>), equal to 3ml of gel. A single application of a menthol-based topical analgesic has previously been shown to significantly reduce pain perception (Johar et al., 2012) and was considered adequate for our study design.

After application, participants were instructed to remain in a seated and upright position for 15 minutes. While the time for maximum effect of menthol varies per individual, 15-minutes allow for the sensation of cold to be perceived and provided a window of opportunity for us to complete the study while cold sensation was still being reported (times based on Yosipovitch et al., 1996). After the 15 minutes, pressure threshold was measured under the same conditions as previously described. Next, participants completed the final 10-minute running analysis under the same conditions as the two previous analyses.

#### *Day 1-5: Pain Journaling*

In order to ensure that participants were experiencing DOMS and that symptoms had subsided after the study; a pain journal was kept over a 5-day period from initial visit until 96 hours post. A Baseline measure was used to identify their base level of pain but also to ensure that there was no predisposing injury that might affect the outcome of participation. Other important timepoints outside of each kinematic analysis was the two days following participation. Participants were asked to report their pain level on the CPS scale used during the study on the morning and night of the two days following to ensure that they recovered from the DOMS-inducing protocol adequately. If a participant reported a “0” before the end of the outlined timepoints, they were not contacted further. See Appendix 8.3 for a detailed outline of each timepoint for the pain journal.

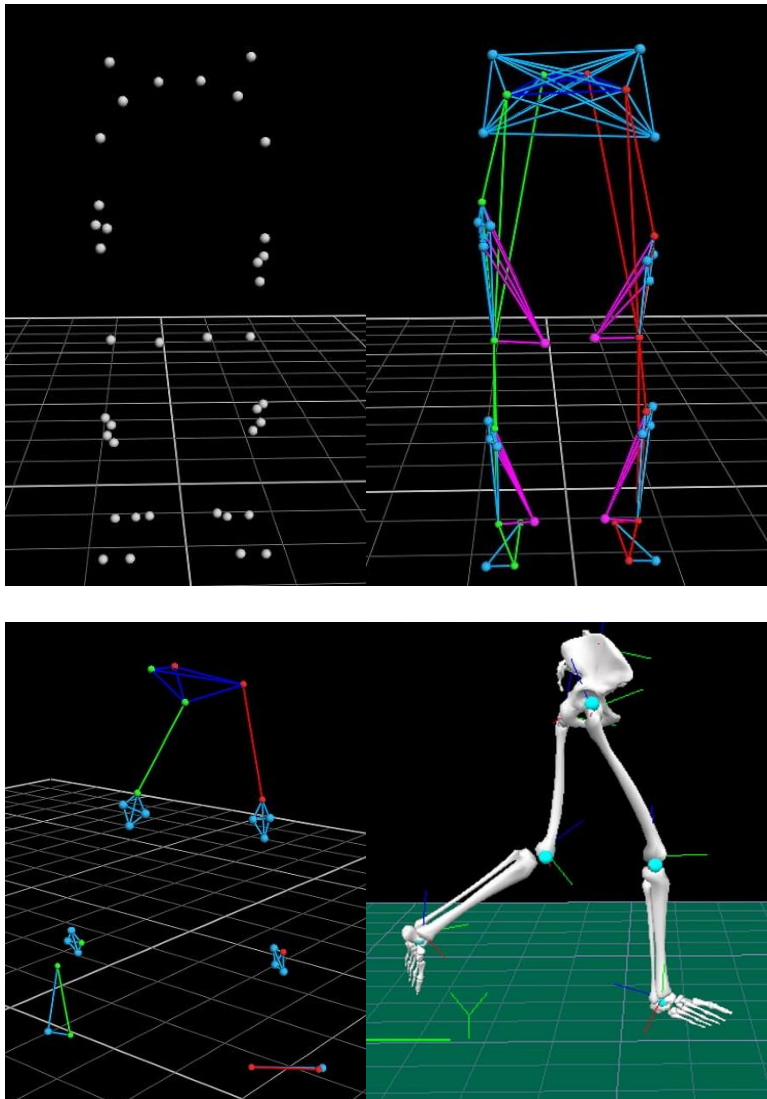
## 3.4 Data Analysis

### 3.4.1 Motion Capture

The second recording from the three collected during each run was used for further analysis. From this trial, 10 total strides were selected to be analyzed. All participants were right-leg dominant, therefore, the right leg data was chosen for the final kinematic analysis. Marker data were labelled using Vicon Nexus 2.7.1 and files were exported as .c3d files for use in Visual3D (C-motion Inc., Germantown, MD, USA) (Figure 6). Once in Visual3D, kinematic data were filtered using a 2<sup>nd</sup> order dual-pass Butterworth filter with a 10Hz cut-off. The movement calibration file was used to create functional joint centers at the hip, knee, and ankle which more closely identifies each participants' axes of rotation at each joint. Next, experimental trials were labelled for specific gait events (identifying each stance and swing phase by identifying 10 consecutive foot strikes and toe-offs). Each gait event (identified in Visual 3D) for each trial was visually confirmed and identified by the following: each foot strike (depending on heel or forefoot initial contact) was identified by the point at which the closest marker of contact ceased downward projection along the Z-axis (vertical axis). Each toe-off was identified by the frame before the rapid upward acceleration of the toe marker in the Z-axis. A figure demonstrating the accuracy of gait event selection has been included in the appendices (Appendix 8.5). Each foot contact was used to normalize gait cycle events to a percent of the cycle (%) with the first foot contact being initial contact at 0% of total gait cycle and the following foot contact being

terminal contact at 100% of gait cycle. Phases of gait, stance and swing, were analyzed separately as the requirements of the limb during each phase (loaded, unloaded) are significantly different (Novachek, TF., 1998). For each phase, joint angle data was derived, including ankle, knee and hip angles. The ankle was defined as the foot relative to the shank, the knee as the shank relative to the thigh and the hip as the thigh relative to the pelvis. 3D rotations were calculated using a X-Y-Z rotation sequence (X - flexion/extension). At each phase, minimum, maximum and average joint angles were determined. Stride Rate (SR; Hz) and Stride Length (SL; m) were calculated as spatio-temporal measures. SR and SL were measured during each running analysis congruent with kinematic analysis, during each speed and each condition. SR was calculated as a product of foot strike and was recorded as the time for one stride (s). SL was calculated using SR in the following equation and was recorded as distance in meters (m).

$$\text{Stride Rate (s/stride)} \times \text{Speed(m/s)} = \text{Stride Length (m)}$$



**Figure 6.** The entire labelling process up to functional trials used for analysis in Visual3D (C-motion Inc., Germantown, MD, USA). Left to Right: Initial upload of a calibration trial, labelled calibration trial, labelled movement trial, Visual3D processed trial with functional joints calibrations.

### 3.4.2 Pain

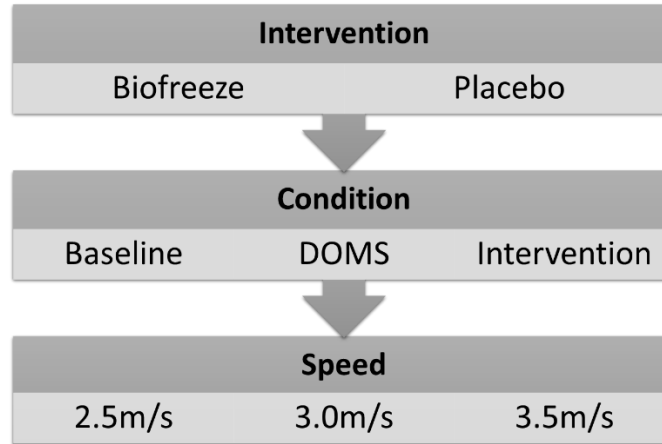
CPS scores were measured during each running analysis at each speed (2.5, 3.0, 3.5 mps) and under each experimental condition (BASE, DOMS, INT). Pressure thresholds were measured at 3 time points - directly prior to each experimental condition.

Measurements from each site were averaged over the last 3 of 5 trials to allow participants to acclimate to the testing procedure and provide more reliable scores (Nussbaum & Downes, 1998).

### 3.4.3 Statistical Analysis

A Mixed-Design Three Way 2 x (Intervention) x 3 (Condition) x3 (Speed) Analysis of Variance (Repeated Measures with a Between Subject Factor) was used to determine the effect of Biofreeze versus the placebo gel on all kinematic variables, spatio-temporal parameters as well as comparative pain scale.

A Mixed Design Two Way 2 (Intervention) x 3 (condition) ANOVA was used to determine the effect of menthol versus a placebo gel on pressure threshold. All statistics were completed with IBM SPSS (V 25 IBM Corp., Armonk, N.Y., USA). Significance was set to  $p < .05$ . Significant main effect and interactions were evaluated using pair-wise comparisons and a Bonferroni correction. An overview of the dependent and independent variables is outlined in Figure 7. Values are expressed as Mean  $\pm$  SD and all error bars in figures are expressed as standard error (SE). Tables including p-values and effect sizes have been included in the results (Tables 4 and 5, respectively).



**Figure 7.** An outline of the study variables, where participants were randomly selected for the Menthol or Placebo condition, tested at Baseline and 48 hours later for DOMS and Intervention with the same randomized order of speeds for each participant.

## Section 4: Results

### 4.1 Participant Demographics

Twenty participants were randomized into either a menthol-based topical analgesic (BIO; n=10) or placebo gel group (PLA; n=10). Both groups had equal gender splits (6 Females, 4 Males). Demographics and weekly average mileage breakdown are included in Table 3. P-values and effect sizes from statistical testing (V 25 IBM Corp., Armonk, N.Y., USA) are included in Table 5 and 6 in the following pages.

**Table 3.** Intervention-Based Group Demographics

		Age (years)	Height (m)	Weight (kg)	Weekly Mileage (km/week)
<b>Menthol</b>	Male (n=4)	22.25±0.97	1.82±0.07	76.92±10.61	45.0±37.2
	Female (n=6)	22.67±2.16	1.70±0.03	59.20±5.03	42.5±24.2
	<i>Group (n=10)</i>	22.5±1.72	1.75±0.08	66.29±11.63	43.5±28.1
<b>Placebo</b>	Male (n=4)	23.0±2.45	1.75±0.07	66.74±5.99	42.5±18.5
	Female (n=6)	21.17±2.14	1.66±0.04	57.74±5.77	39.2±19.9
	<i>Group (n=10)</i>	21.5±2.17	1.69±0.07	61.34±7.21	40.5±18.3



## 4.2 Summary of Statistics

**Table 4.** p-values for all main effects and interactions for each variable

Variable	Condition	Condition*Group	C*S*G
Average Ankle Stance	0.242	0.518	0.611
Average Ankle Swing	0.176	0.209	0.091
Average Hip Stance	0.552	0.001 *	0.227
Average Hip Swing	0.201	0 *	0.777
Average Knee Stance	0.172	0.01 *	0.695
Average Knee Swing	0.119	0.042 *	0.343
Peak Ankle Dorsiflexion Stance	0.102	0.591	0.923
Peak Ankle Plantarflexion Stance	0.454	0.686	0.316
Peak Ankle Dorsiflexion Swing	0.272	0.077	0.579
Peak Ankle Plantarflexion Swing	0.007 *	0.556	0.223
Peak Hip Flexion Stance	0.624	0.074	0.646
Peak Hip Extension Stance	0.562	0.00 *	0.359
Peak Hip Flexion Swing	0.698	0.001 *	0.425
Peak Hip Extension Swing	0.164	0.001 *	0.989
Peak Knee Flexion Stance	0.433	0.099	0.945
Peak Knee Extension Stance	0.575	0.098	0.343
Peak Knee Flexion Swing	0.796	0.367	0.136
Peak Knee Extension Swing	0.015 *	0.034 *	0.818
Comparative Pain Scale	0.00 *	0.69	0.157
Pressure Algometry (L-VM0)	0.00 *	0.637	NA
Pressure Algometry (R-VM0)	0.00 *	0.872	NA
Pressure Algometry (L-GAS)	0.016 *	0.768	NA
Pressure Algometry (R-GAS)	0.00 *	0.14	NA
Stride Rate (Hz)	0.798	0.419	0.759
Stride Length (m)	0.875	0.417	0.870

### 4.3 Effect Sizes

**Table 5.** Effect sizes (Partial Eta Squared) for all main effects and interactions for each variable

Variable	Condition	Condition*Group	C*S*G
Average Ankle Stance	0.076	0.036	0.033
Average Ankle Swing	0.092	0.083	0.104
Average Hip Stance	0.033	0.343	0.075
Average Hip Swing	0.085	0.389	0.024
Average Knee Stance	0.093	0.227	0.030
Average Knee Swing	0.112	0.162	0.060
Peak Ankle Dorsiflexion Stance	0.119	0.029	0.012
Peak Ankle Plantarflexion Stance	0.043	0.021	0.063
Peak Ankle Dorsiflexion Swing	0.070	0.133	0.039
Peak Ankle Plantarflexion Swing	0.240	0.032	0.075
Peak Hip Flexion Stance	0.054	0.264	0.145
Peak Hip Extension Stance	0.032	0.448	0.058
Peak Hip Flexion Swing	0.020	0.344	0.052
Peak Hip Extension Swing	0.063	0.357	0.013
Peak Knee Flexion Stance	0.045	0.121	0.010
Peak Knee Extension Stance	0.030	0.121	0.060
Peak Knee Flexion Swing	0.013	0.054	0.092
Peak Knee Extension Swing	0.095	0.340	0.004
Comparative Pain Scale	0.710	0.020	0.087
Pressure Algometry (L-VM0)	0.425	0.025	NA
Pressure Algometry (R-VM0)	0.385	0.008	NA
Pressure Algometry (L-GAS)	0.205	0.015	NA
Pressure Algometry (R-GAS)	0.393	0.104	NA
Stride Rate (Hz)	0.012	0.047	0.025
Stride Length (m)	0.007	0.047	0.017

#### 4.4 Subjective Pain Analysis - Comparative Pain Scale

CPS was assessed during each running speed and condition to determine subjective soreness, specific to the musculature of the lower limbs. There was a significant main effect for condition ( $p < 0.05$ ). Pairwise comparisons revealed a significant increase in CPS scores after the DOMS-inducing protocol. There were no significant interactions for CPS (figure 8). Irrespective of speed, the BIO group had a  $3.03 \pm 0.32$  increase on the CPS scale from the Baseline run analysis (BASE) to the DOMS-Induced run analysis (DOMS) and a  $0.9 \pm 0.21$  decrease after the application of Menthol during the Intervention run analysis (INT) (figure 8). The PLA group had a  $2.52 \pm 0.3$  increase from BASE to DOMS and a  $0.53 \pm 0.25$  decrease after the application of the Placebo gel during INT.

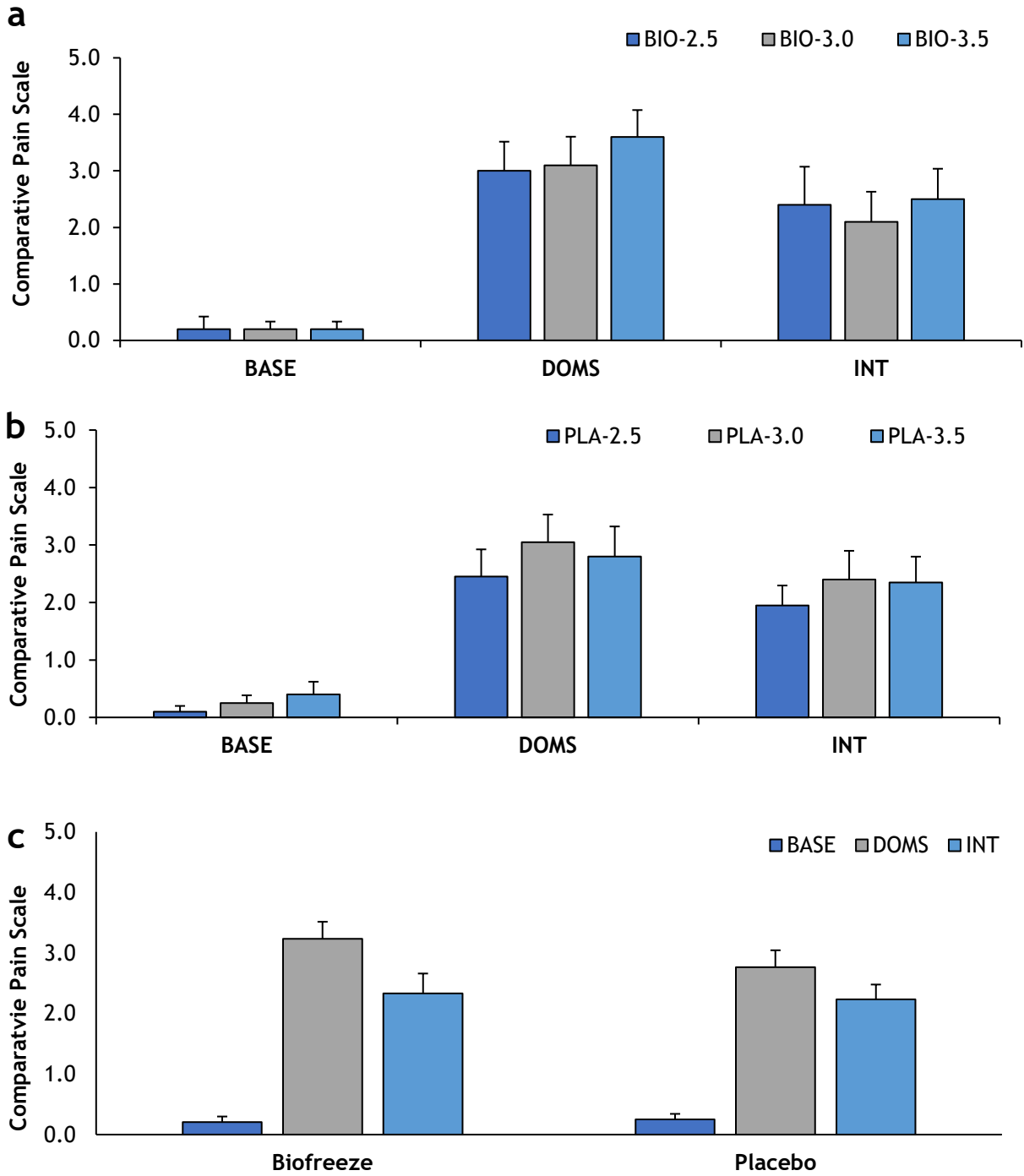


Figure 8. Comparative Pain Scale scores for Menthol (a) Placebo (b) and Condition x Group (c)

## 4.5 Subjective Pain Analysis - Pressure Algometry

### *Gastrocnemius-Achilles Complex*

There was a significant main effect of condition for both left and right GA-MTJ's ( $p < .05$ ). Both left and right GA-MTJ's showed a significant decrease in pressure threshold after the DOMS-inducing protocol and increase after the intervention application (Figure 9) There was no significant interaction of Group x Condition for either GA-MTJ. The left GA-MTJ for the Menthol group (BIO-L) had a  $12.56 \pm 20.71$  N decrease in pressure threshold from BASE to DOMS and an  $11.21 \pm 19.86$  N increase in pressure threshold from DOMS to INT. The right GA-MTJ for the Menthol group (BIO-R) had a  $21.24 \pm 20.71$  N decrease in pressure threshold from BASE to DOMS and a  $14.2 \pm 12.72$  N increase from DOMS to INT. The left GA-MTJ for the Placebo group (PLA-L) had a  $12.6 \pm 23.17$  N decrease from BASE to DOMS and a  $5.8 \pm 7.05$  N increase from DOMS to INT. PLA-R had a  $10.42 \pm 16.91$  N decrease from BASE to DOMS and a  $2 \pm 8.47$  N increase from DOMS to INT (Figure 9).

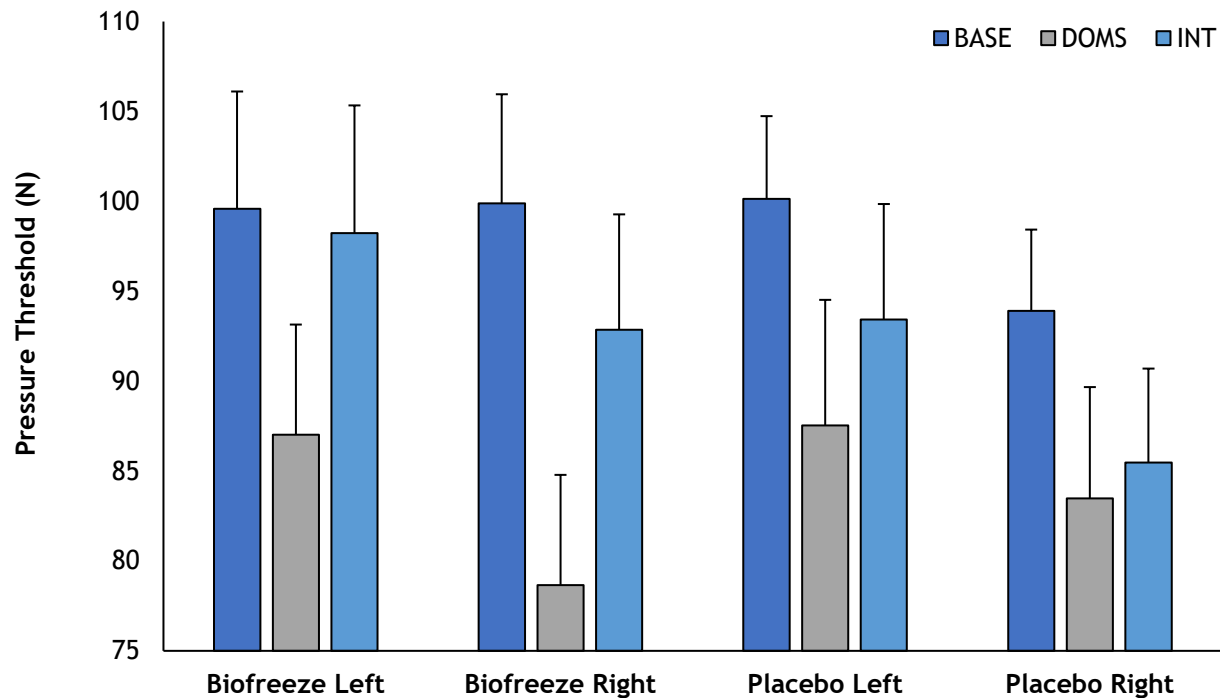


Figure 9. Pressure threshold (N) for the gastrocnemius-achilles myotendinous junction site.

### *Vastus Medialis Oblique*

There was a significant main effect of condition for both left and right VMO's ( $p < .05$ ). Both left and right VMO's showed a significant decrease in pressure threshold after the DOMS-inducing protocol and increase after the intervention application (Figure 10). There was no significant interaction of Group x Condition for VMO. BIO-L had a  $17.67 \pm 16.39$  N decrease in pressure threshold from BASE to DOMS and a  $10.45 \pm 14.76$  N increase from DOMS to INT. BIO-R had a  $18.17 \pm 18.45$  N decrease from BASE to DOMS and a  $8.32 \pm 20.54$  N increase from DOMS to INT. PLA-L had a  $13.39 \pm 14.97$  N decrease from BASE to DOMS and a  $4.95 \pm 6.45$  N increase from DOMS to INT. PLA-R had a  $15.5 \pm 21.43$  N decrease from BASE to DOMS and a  $4.7 \pm 8.54$  N increase from DOMS to INT (Figure 10).

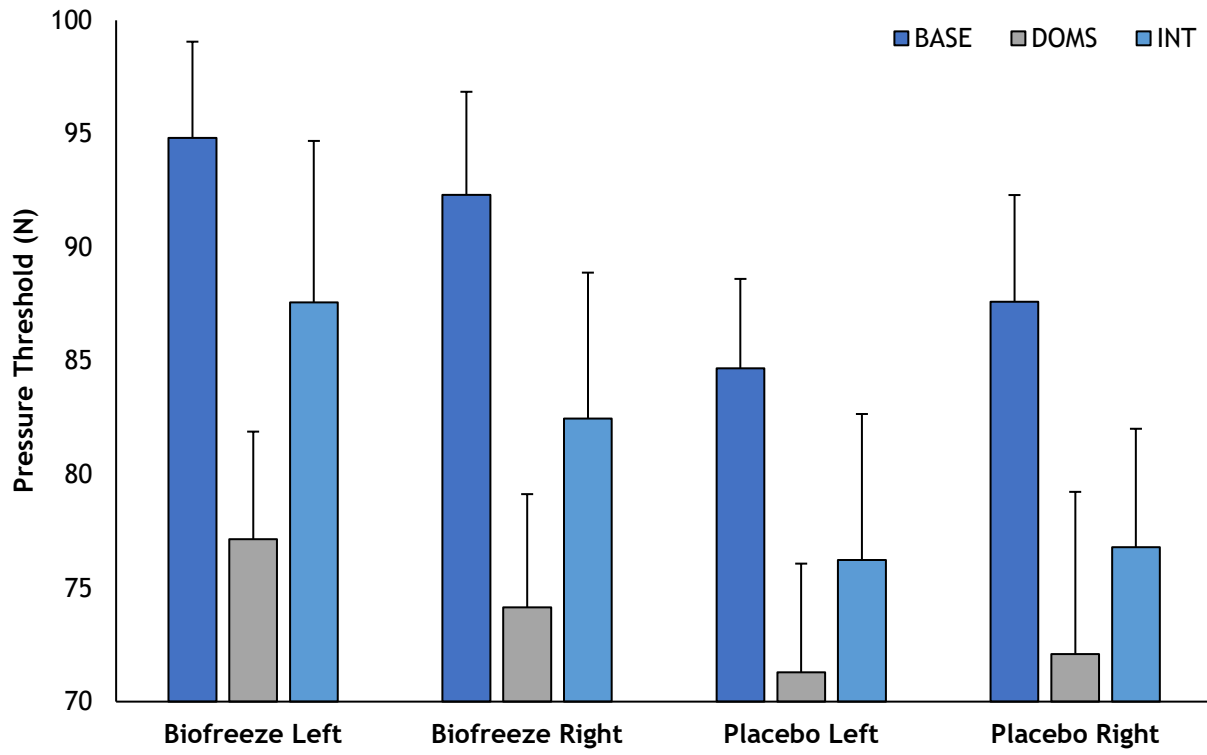
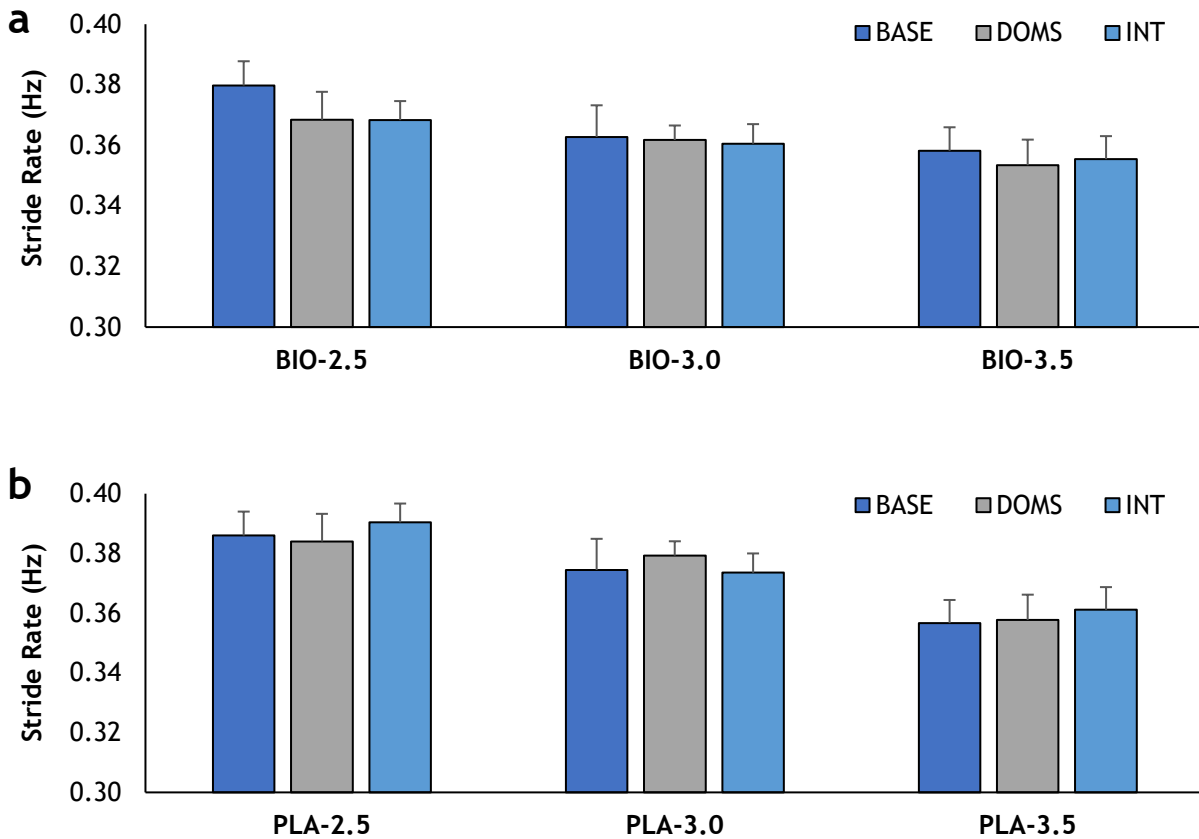


Figure 10. Pressure threshold (N) for vastus medialis obliques

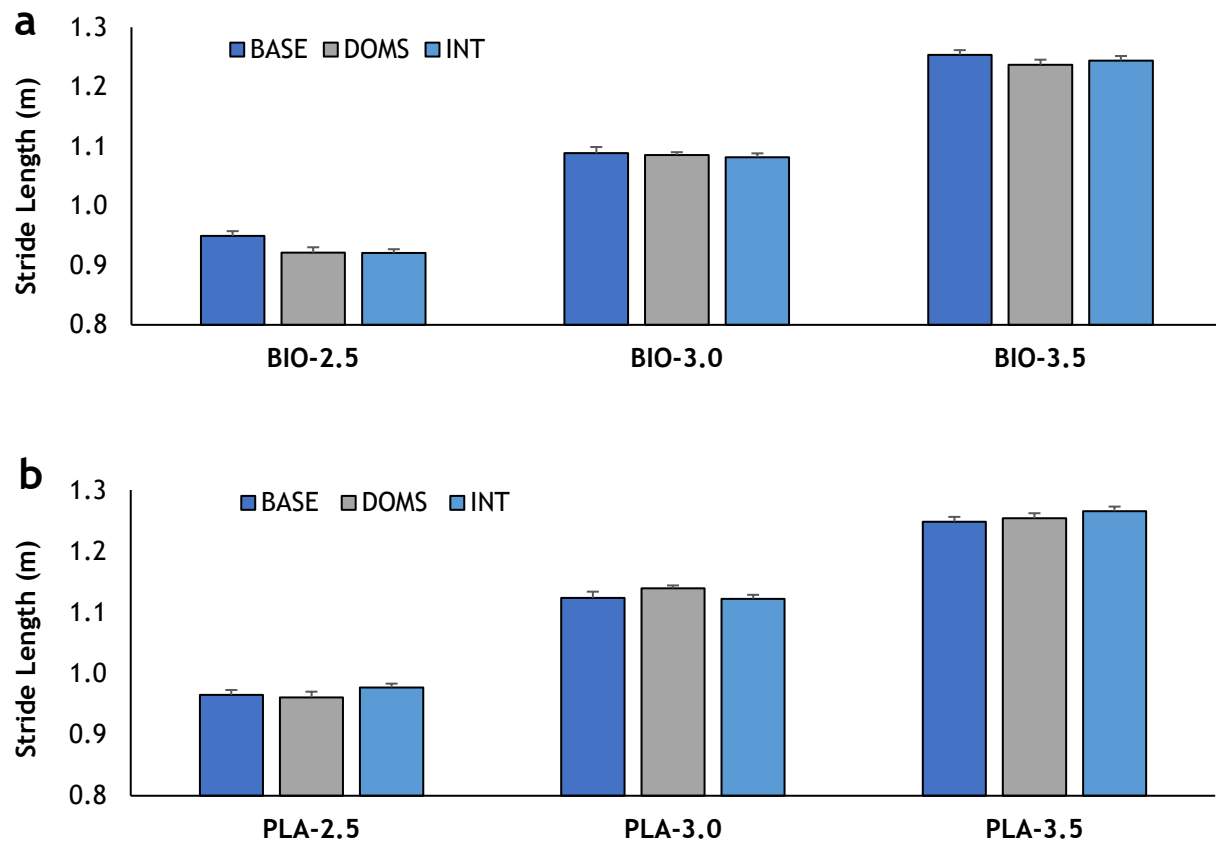
## 4.6 Spatio-Temporal Parameters

There were no significant main effects or interactions for Stride Rate (Figure 11) or stride length (Figure 12).



**Figure 11.** Stride Rate (hz) for Menthol (a) and Placebo (b) separated by condition, intervention and speed





**Figure 12.** Stride Length (m) for Menthol (a) and Placebo (b) separated by condition, intervention and speed

#### 4.7 Ankle Kinematics - Stance

There were no significant main effects or interactions for ankle kinematics during stance (Figure 13).

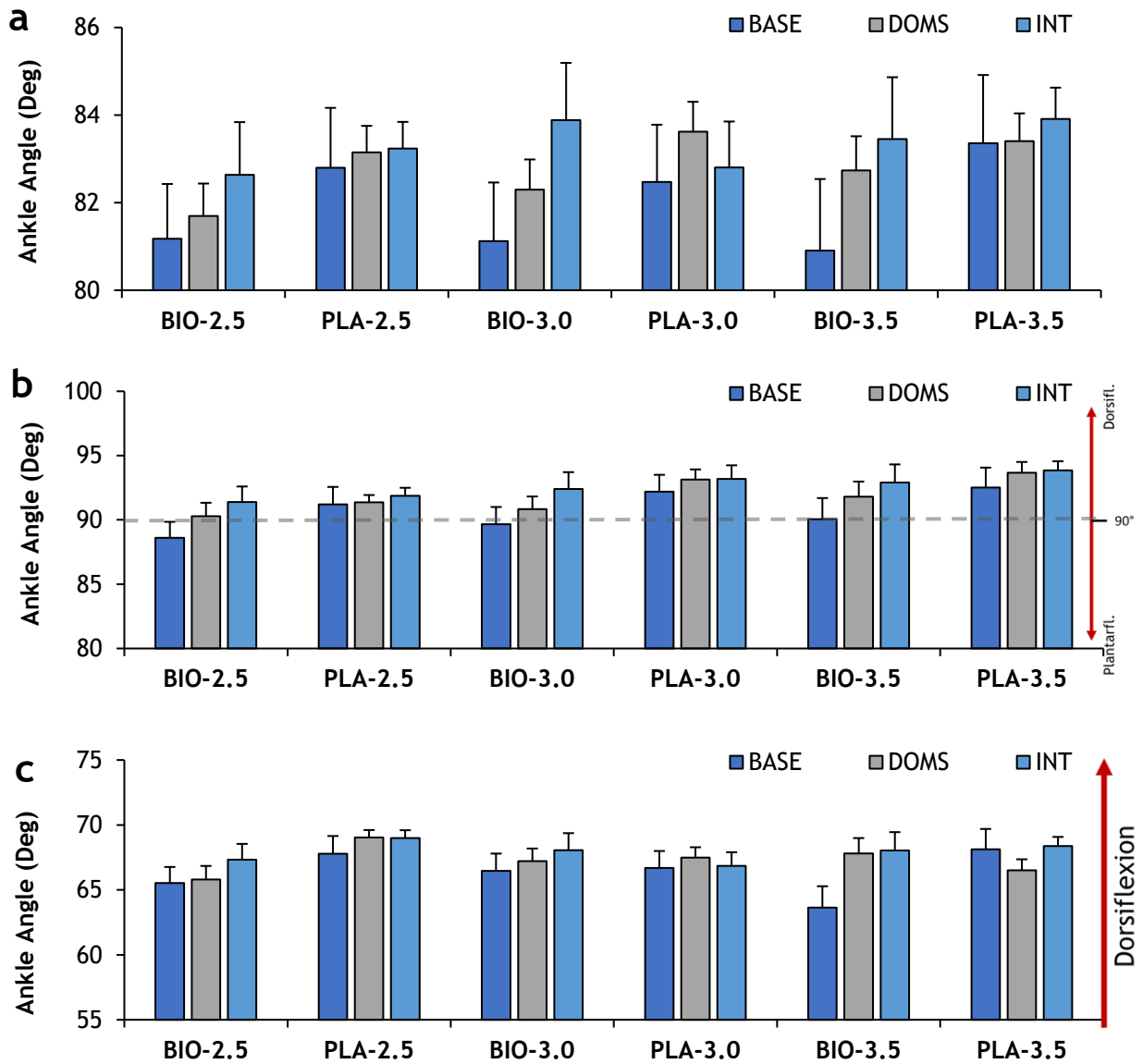


Figure 13. Average ankle ROM (a), peak dorsiflexion (b), and peak plantarflexion (c) over stance

## 4.8 Ankle Kinematics - Swing

There were no significant main effects or interactions for average ankle ROM or peak dorsiflexion during swing (Figure 14). A significant main effect for condition was found for peak plantarflexion during swing, however, pairwise comparisons revealed no significant differences.

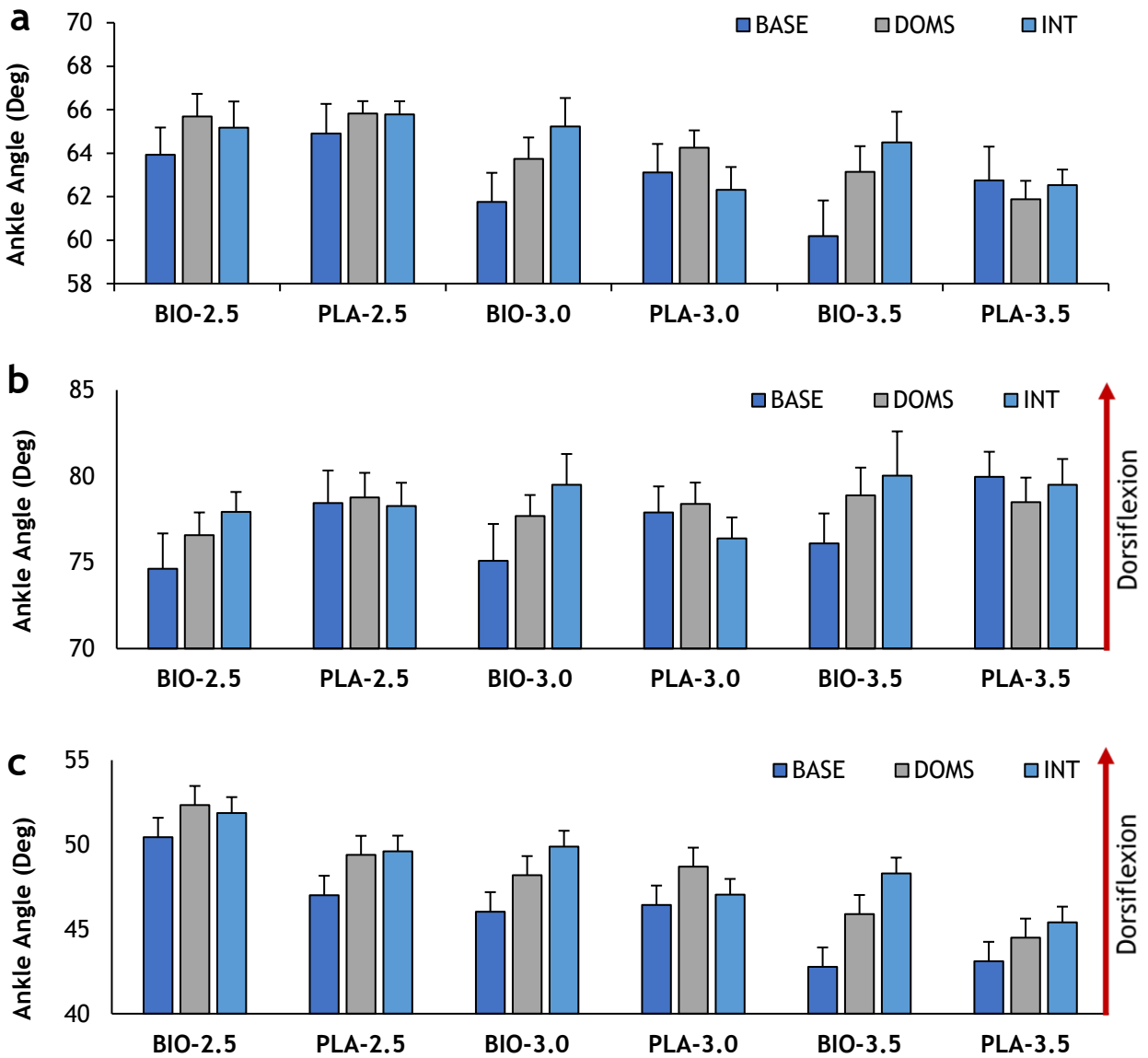


Figure 14. Average ankle ROM (a), peak dorsiflexion (b), and plantarflexion (c) over swing

## 4.9 Knee Kinematics - Stance

For this analysis,  $0^\circ$  is considered a fully extended knee (Appendix 8.6). There were no significant main effects for average knee ROM during stance. There was a significant Condition x Group interaction for average knee ROM during stance ( $p < .05$ ). The BIO group had a  $2.47 \pm 0.44^\circ$  reduction in knee ROM during stance from BASE to DOMS and a  $1.35 \pm 0.93^\circ$  increase from DOMS to INT. The PLA group had a  $1.10 \pm 0.10^\circ$  increase from BASE to DOMS and a  $1.58 \pm 1.63^\circ$  from DOMS to INT (Figure 15).

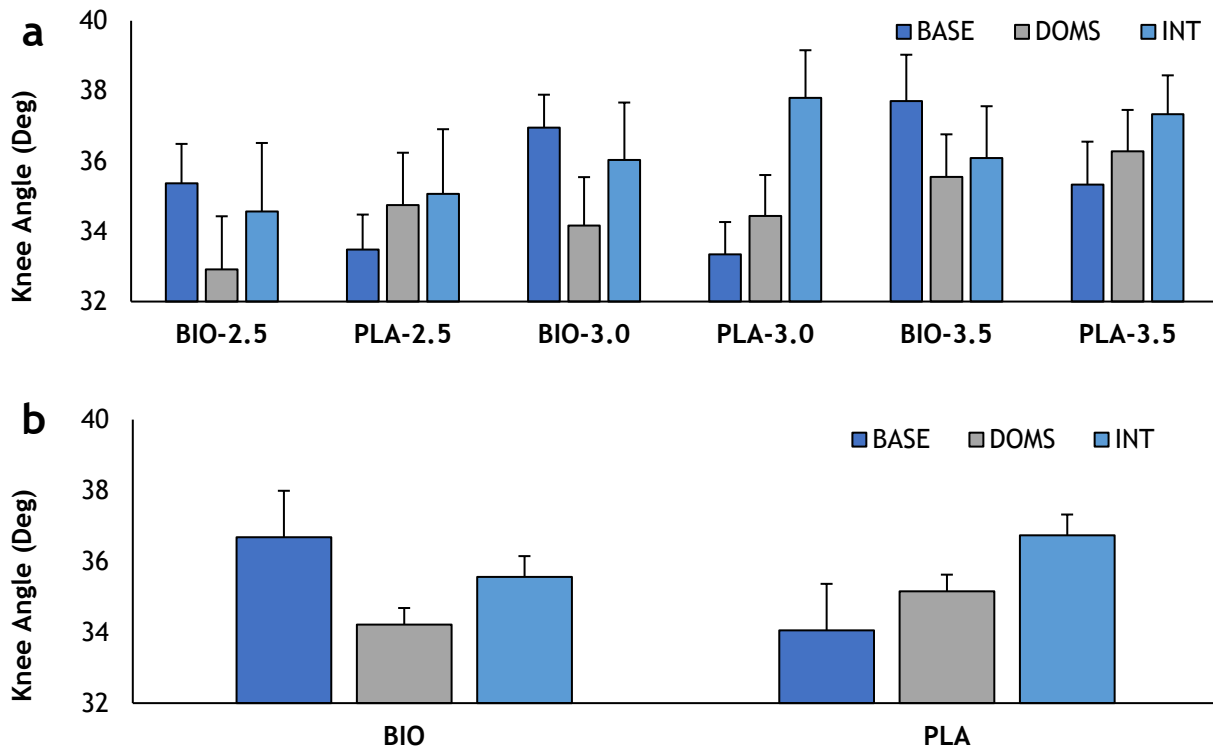


Figure 15. Average knee ROM (a); speed-averaged (b) during stance

There were no significant main effects or interactions for peak knee flexion or extension during stance (Figure 16).

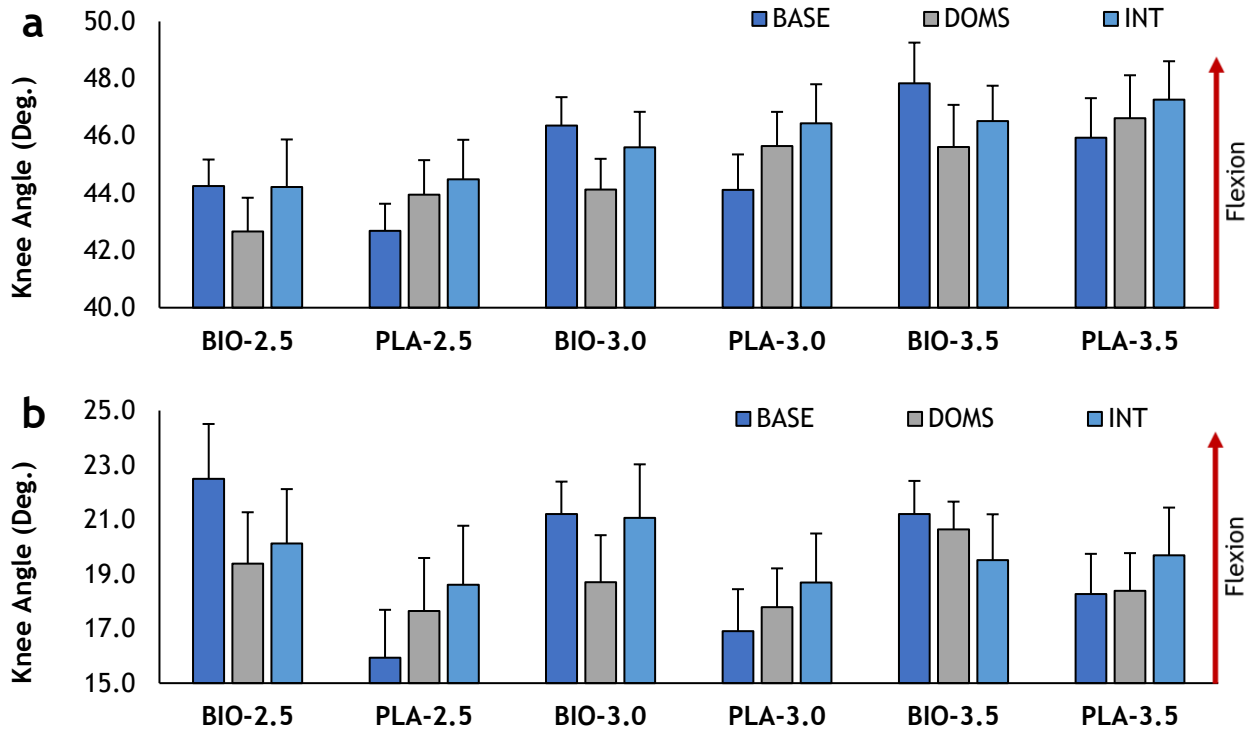


Figure 16. Peak knee flexion (a) and extension (b) during stance

#### 4.10 Knee Kinematics - Swing

There were no significant main effects for average knee ROM during swing. There was a significant interaction of Condition x Group ( $p < .05$ ). The BIO group had a  $1.49 \pm 0.99^\circ$  decrease in knee ROM during swing from BASE to DOMS and a  $1.73 \pm 1.09^\circ$  increase from DOMS to INT. The PLA group had a  $2.11 \pm 1.01^\circ$  increase from BASE to DOMS and a  $0.46 \pm 1.41^\circ$  increase from DOMS to INT (Figure 17).

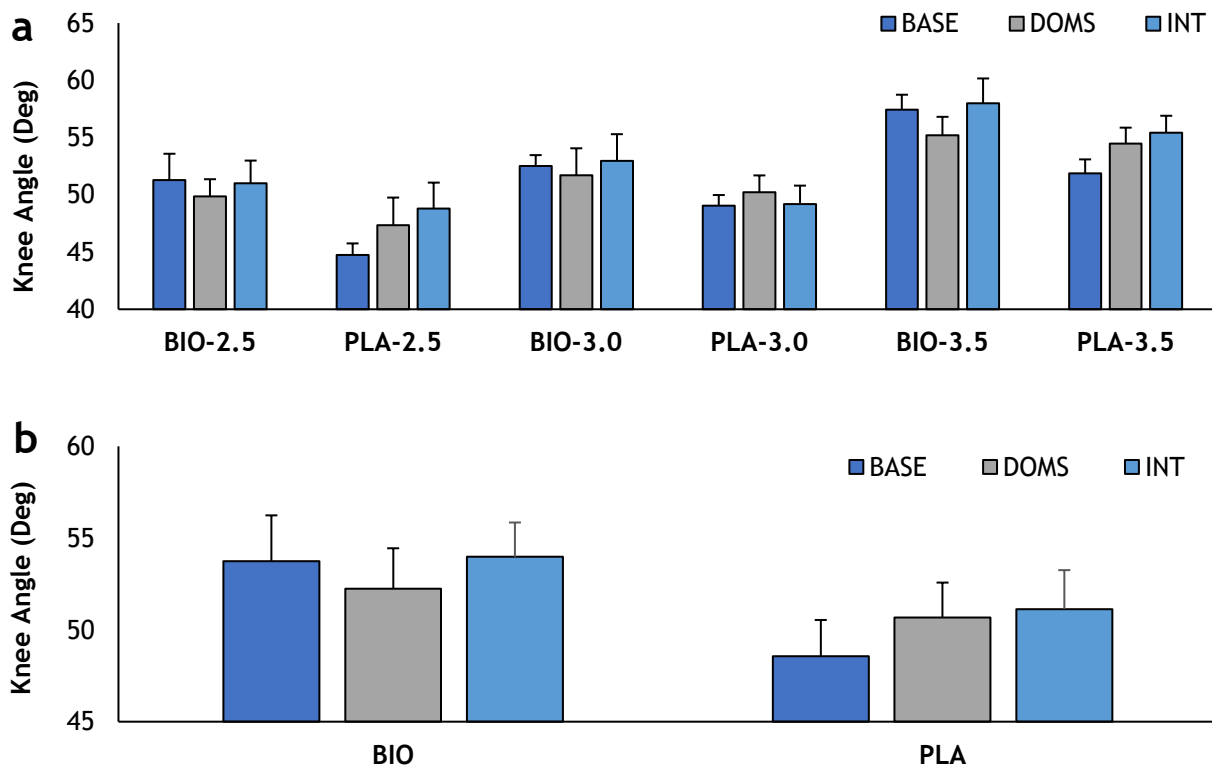


Figure 17. Knee average ROM (a); speed-averaged (b) during swing

There were no significant main effects or interactions for peak knee flexion during swing (Figure 18).

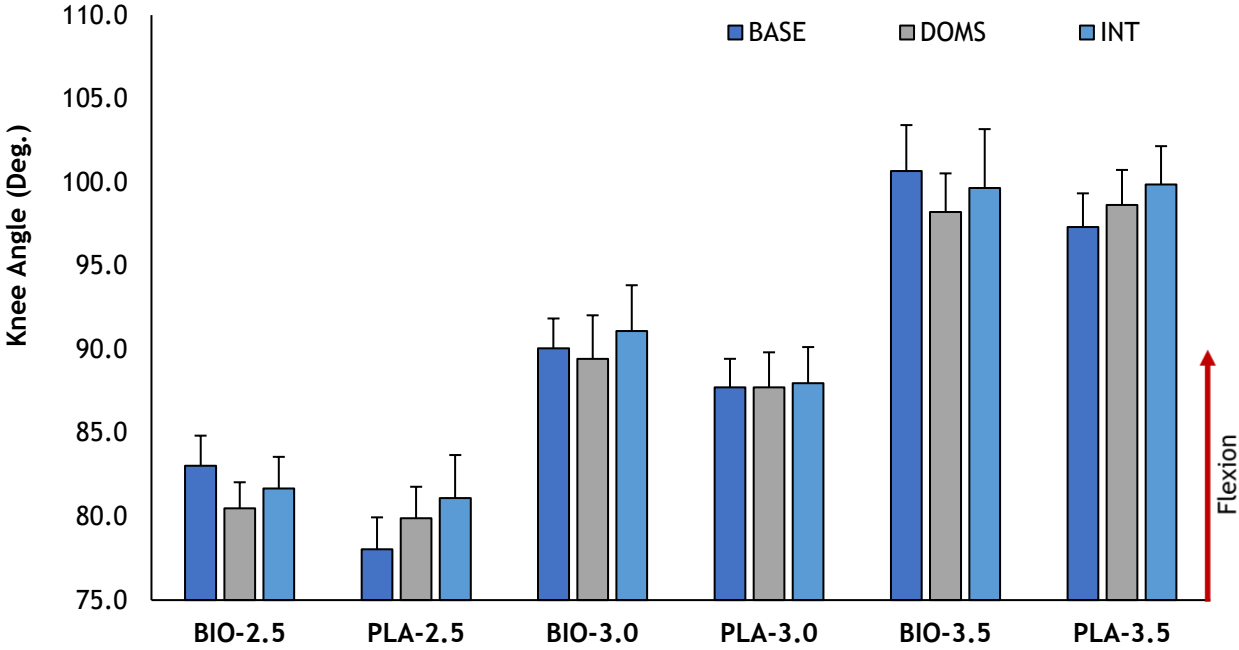


Figure 18. Peak knee flexion during swing

There was a significant main effect of Condition for peak knee extension during swing ( $p < .05$ ). Pairwise comparisons revealed no significant differences. There was a significant interaction of Condition x Group for peak knee extension during swing ( $p < .05$ ). Irrespective of speed, the BIO group had a  $1.18 \pm 1.04^\circ$  increase (more extended) in peak knee extension during swing from BASE to DOMS and a  $1.81 \pm 1.16^\circ$  decrease (more flexed) from DOMS to INT. The PLA group had a  $2.28 \pm 0.78^\circ$  decrease from BASE to DOMS and a further decrease of  $1.47 \pm 0.16^\circ$  from DOMS to INT (Figure 19).

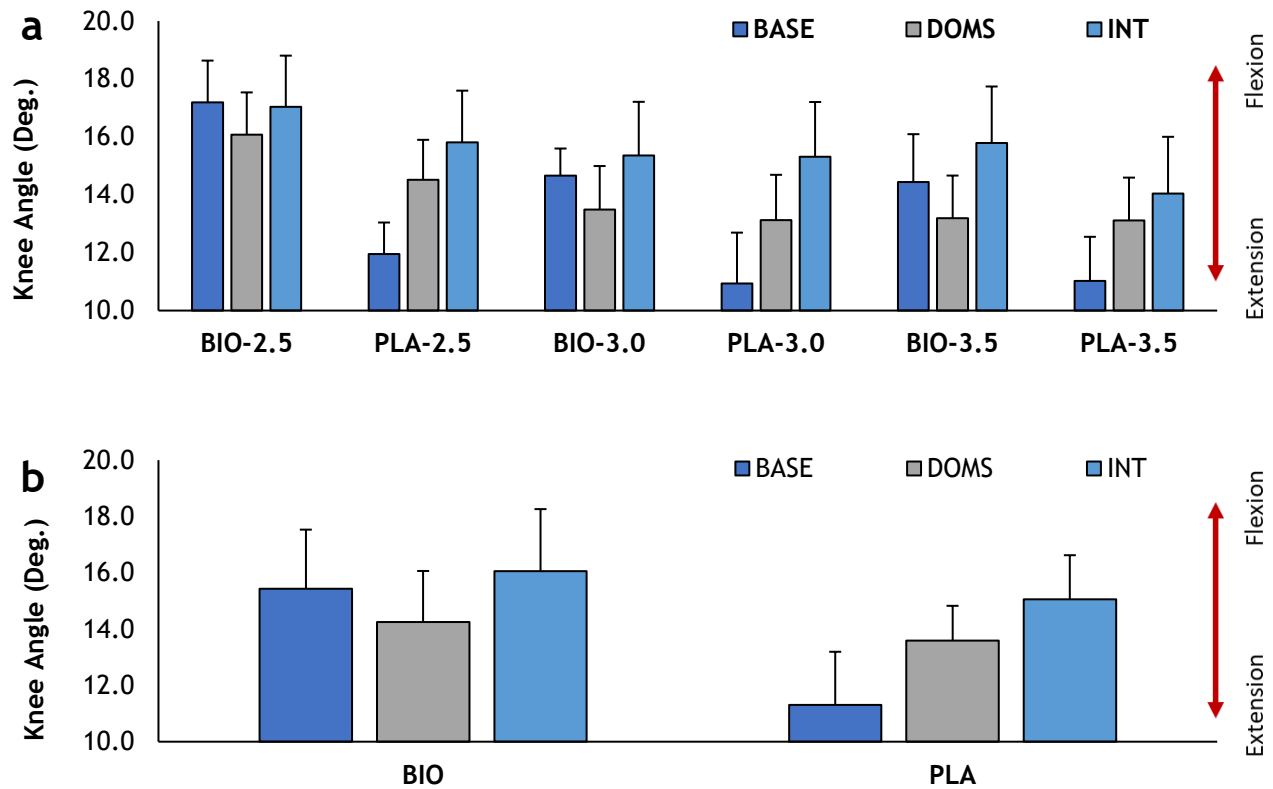


Figure 19. Peak knee extension (a); speed-averaged (b) during swing



#### 4.11 Hip Kinematics - Stance

There were no significant main effects for average hip ROM during stance. There was a significant interaction of Condition x Group ( $p < .05$ ). The BIO group had a  $3.21 \pm 0.38^\circ$  reduction in hip ROM during stance from BASE to DOMS and a  $0.95 \pm 1.35^\circ$  increase from DOMS to INT. The PLA group had a  $4.38 \pm 1.22^\circ$  increase from BASE to DOMS and a  $0.03 \pm 0.88^\circ$  decrease from DOMS to INT (Figure 20).

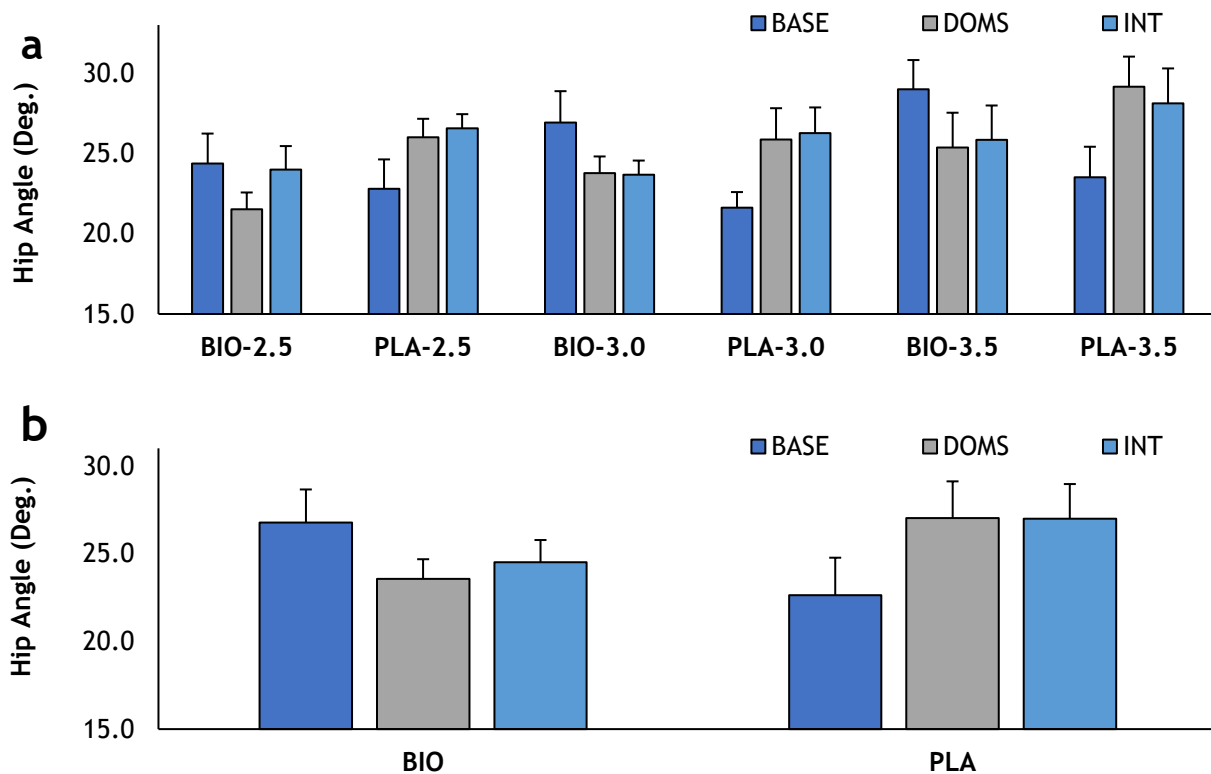


Figure 20. Hip average ROM (a); speed-averaged (b) during stance

For this analysis, 0° is considered a neutral hip position (Appendix 8.6). There were no significant main effects or interactions for peak hip flexion during stance (Figure 21).

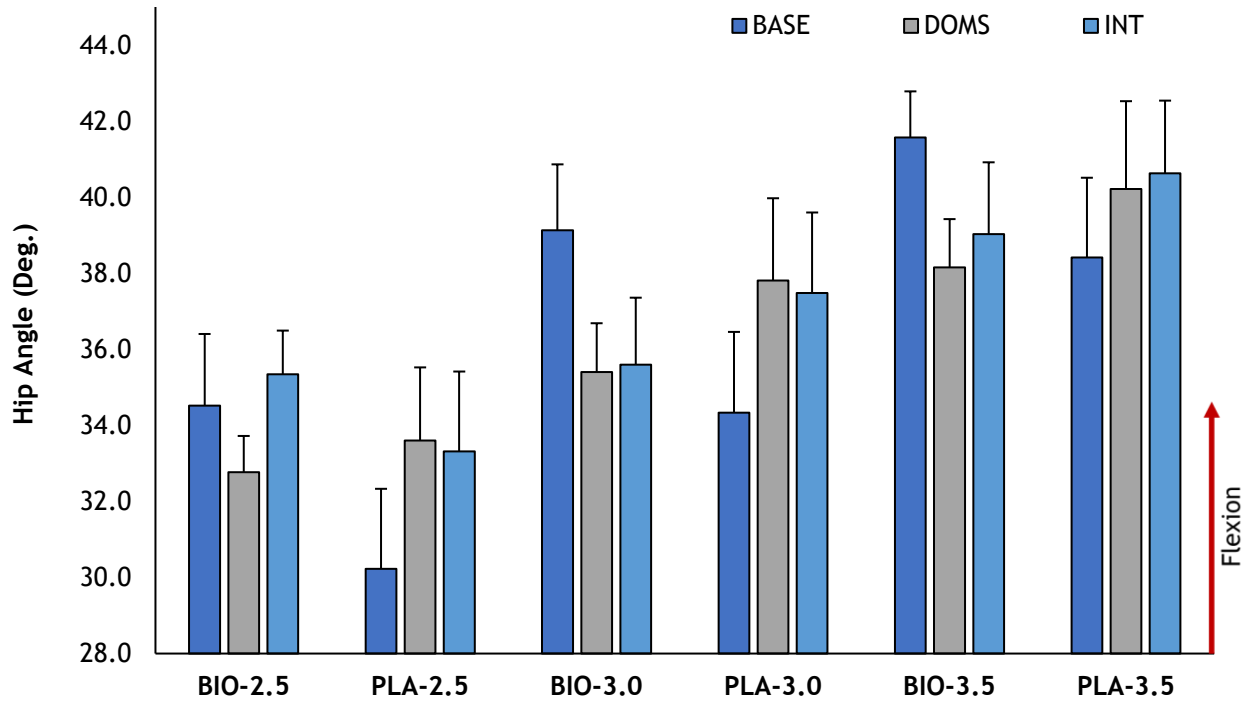


Figure 21. Peak hip flexion during stance

There were no significant main effects for peak hip extension during stance. There was a significant interaction of Condition x Group ( $p < .05$ ) for peak hip extension during stance. For Condition x Group, the BIO group had a  $3.74 \pm 0.13^\circ$  increase (more extended) in peak hip extension during stance from BASE to DOMS and had no change ( $0.04 \pm 1.23^\circ$ ) from DOMS to INT. The PLA group had a  $5.54 \pm 0.83^\circ$  decrease (less extended) from BASE to DOMS and had no change ( $.06 \pm 1.00^\circ$ ) from DOMS to INT (Figure 22).

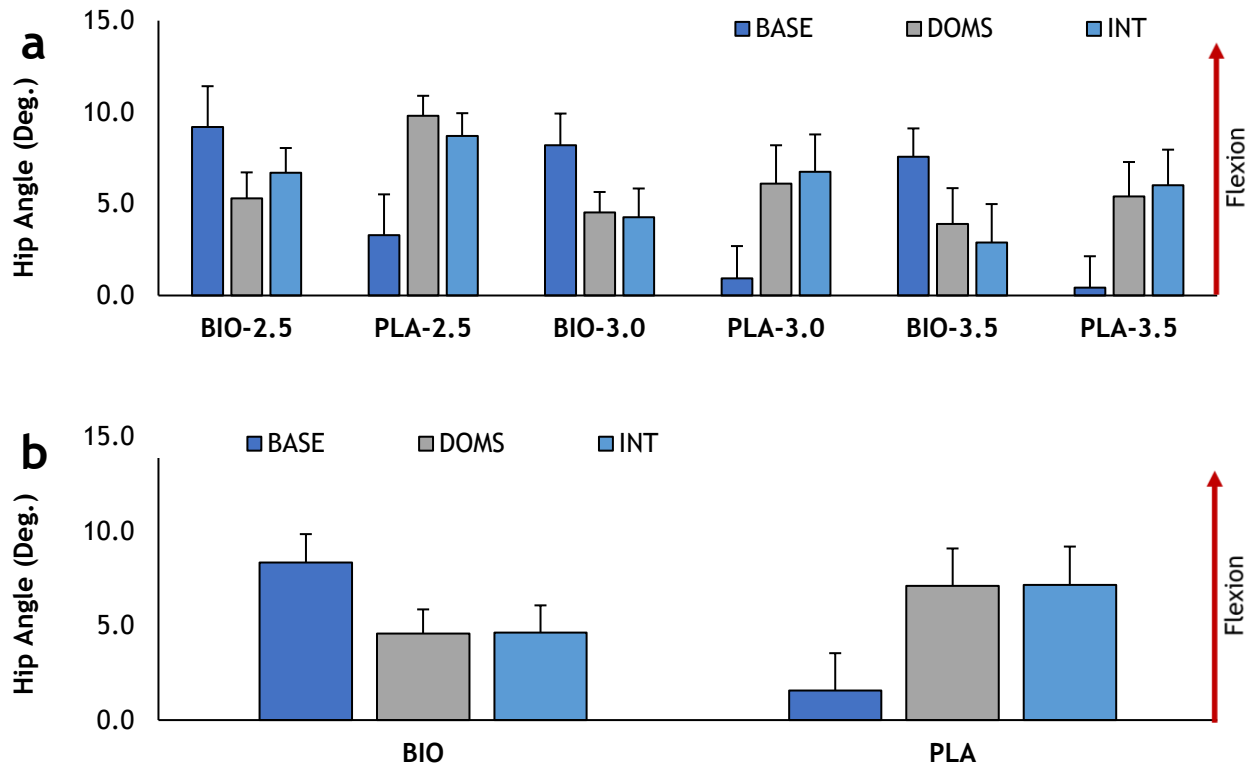


Figure 22. Peak hip extension (a); speed-averaged (b) during stance

#### 4.12 Hip Kinematics - Swing

There were no significant main effects for average hip ROM during swing. There was a significant interaction of Condition x Group ( $p < .05$ ). The BIO group had a  $2.87 \pm 0.32^\circ$  reduction in hip ROM during swing from BASE to DOMS and a  $0.48 \pm 0.56^\circ$  increase from DOMS to INT. The PLA group had a  $5.66 \pm 0.56^\circ$  increase from BASE to DOMS and a  $0.25 \pm 0.08^\circ$  increase from DOMS to INT (Figure 23).

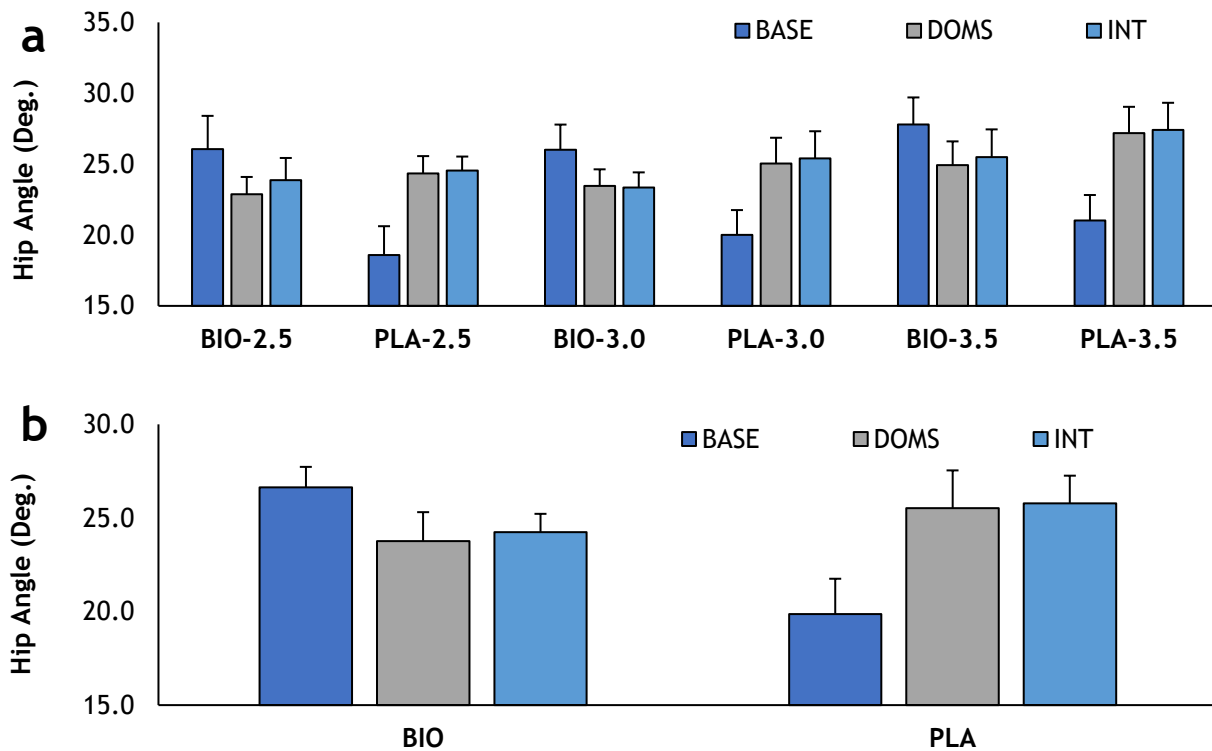


Figure 23. Hip average ROM (a); speed-averaged (b) during swing

There were no significant main effects for peak hip flexion during swing. There was a significant interaction of Condition x Group for peak hip flexion during swing ( $p < .05$ ). The BIO group had a  $4.07 \pm 0.88^\circ$  decrease in peak hip flexion during swing from BASE to DOMS and a  $0.71 \pm 1.89^\circ$  from DOMS to INT. The PLA group had a  $6.16 \pm 0.83^\circ$  increase from BASE to DOMS and a  $1.29 \pm 1.94^\circ$  decrease from DOMS to INT (Figure 24).

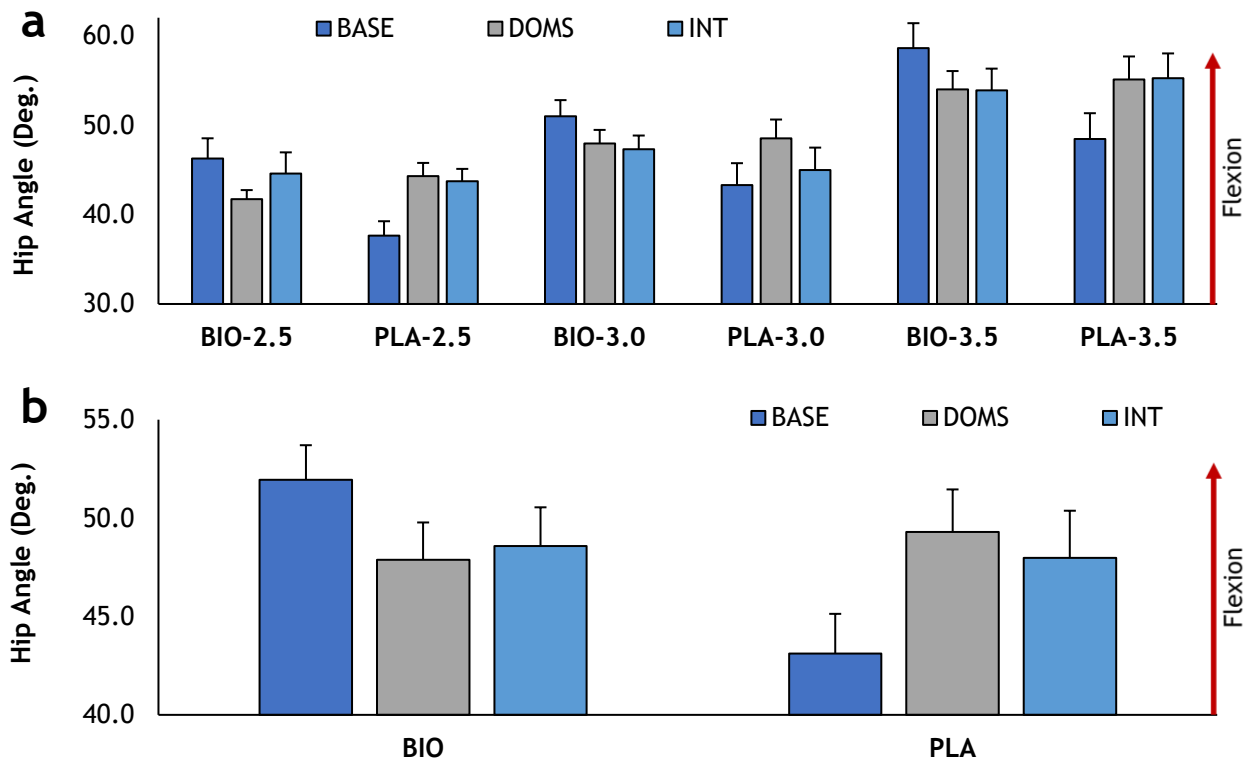


Figure 24. Peak hip flexion (a); speed-averaged (b) during swing

There were no significant main effects for peak hip extension during swing. There was a significant interaction of Condition x Group ( $p < .05$ ). The BIO group had a  $1.03 \pm 0.38^\circ$  increase in peak hip extension (more extended) during swing from BASE to DOMS and a  $0.53 \pm 0.42^\circ$  decrease (more flexed) from DOMS to INT. The PLA group had a  $5.27 \pm 0.32^\circ$  decrease from BASE to DOMS and a further decrease of  $0.59 \pm 0.18^\circ$  from DOMS to INT (Figure 25).

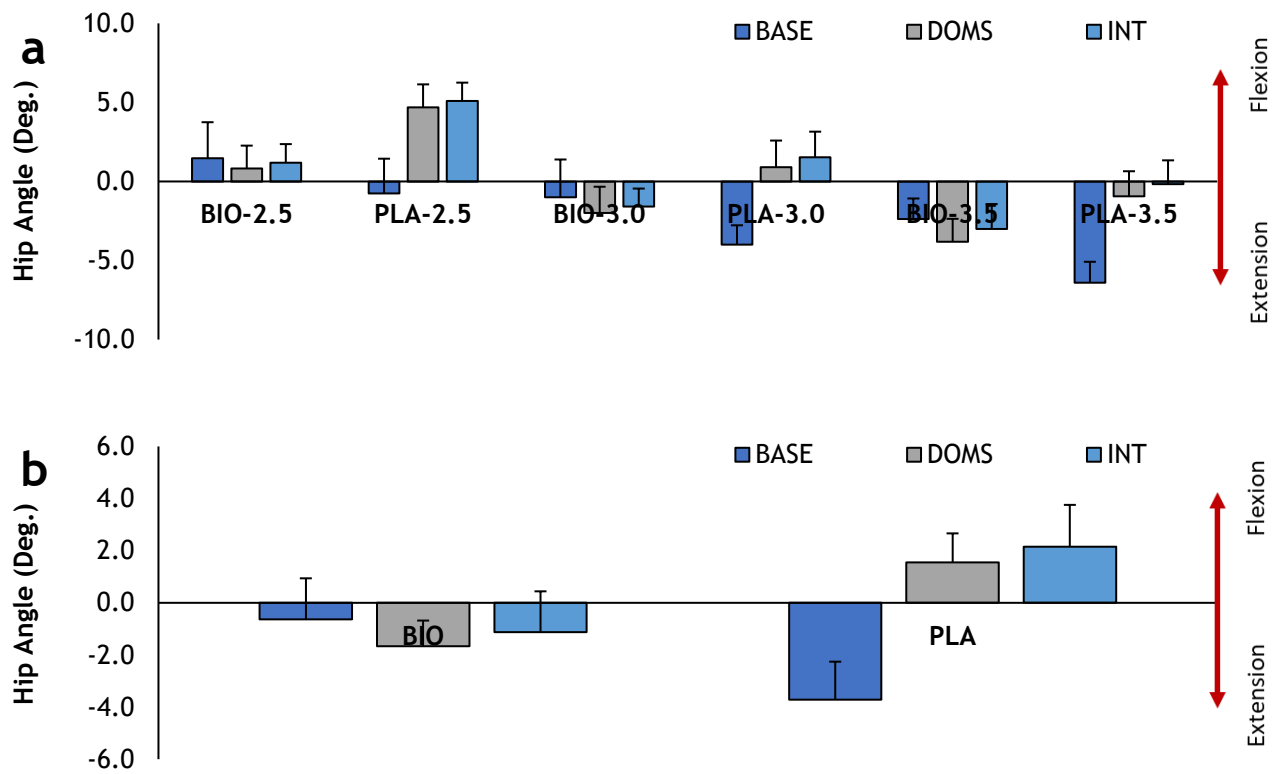


Figure 25. Peak hip extension (a); speed-averaged (b) during swing

## Section 5: Discussion

### 5.1 Overall Trends and Themes

This study assessed the effects of a menthol-based topical analgesic on DOMS-induced changes to running biomechanics and pain perception. Previous work has demonstrated that DOMS can lead to potentially undesirable changes in running mechanics (Tsatalas et al. 2013; Chen et al., 2009; Dutto & Braun, 2004; Paschalis et al., 2007; Paquette et al., 2017). This thesis sought to evaluate if a menthol-based intervention could restore running mechanics to pre DOMS values, which could, in theory, lead to a reduction in overall injury risk while maintaining training intensity. Overall, the findings from this study provided mixed results on the effectiveness of a menthol-based topical analgesic on DOMS-induced changes to running biomechanics and pain perception in experienced runners. In terms of running kinematics (hip, knee and ankle angles), there were only a few statistical differences and relatively small effect sizes. However, there were some changes to pain measures and a few kinematic variables, which could potentially have clinical or practical relevance. This could suggest that the menthol-based intervention did not change (restore) running kinematics to baseline (pre DOMS) levels. It should be noted that previous running studies found significant decreases in knee ROM after a DOMS-inducing protocol (Braun & Dutto, 2004; Chen et al., 2009; Tsatalas et al., 2013). While our subjective analysis, pressure algometry and anecdotal evidence suggests that our DOMS protocol was effective, we didn't find significant changes in running kinematics like found by others (Braun & Dutto, 2004; Chen et al., 2009; Tsatalas et al., 2013). This could suggest that

our DOMS protocol did not change running kinematics to a large enough extent for the menthol-based intervention to have an effect.

Our comparative pain scale and pressure threshold variables suggest that DOMS was induced via our downhill run protocol. However, our spatio-temporal parameters (stride length, stride frequency) were not different across the baseline, DOMS and intervention conditions. In addition, there were no changes in ankle kinematics, which was surprising given that previous work found a significant decrease in ankle ROM after a DOMS-inducing downhill run (Chen et al., 2009; Dutto & Braun, 2004; Tsatalas et al., 2013). There were significant changes for many of our knee and hip variables, however, inconsistent trends and relatively small effect sizes were observed. From a descriptive perspective, many of the biomechanical and pain variables exhibited the expected trends after the DOMS-inducing protocol and the application of a menthol-based topical analgesic. For example, we often observed a decrease in ROM following DOMS and a slight increase (closer to Baseline) following the menthol intervention. This could suggest a few things: 1) that a larger sample size might be needed, 2) the DOMS protocol needs to be adapted and 3) the use of a higher dosage of menthol was needed. Our placebo group had unexpected changes, not consistent with previous work, namely slight increases in range of motion after the DOMS-inducing protocol.

#### *Subjective pain analysis: Comparative Pain Scale*

Our hypothesis for subjective pain analysis was an increase in pain followed by a decrease after the application of a menthol-based topical analgesic. Both groups



exhibited a significant increase in pain after the DOMS-inducing protocol, suggesting that DOMS was induced. A 30mm difference in VAS score (+3.0 on 11-point scale) can be considered a clinically significant increase in pain (Boonstra et al. 2007), and in our work, both groups fall within the range of a clinically significant increase in pain after the DOMS-inducing protocol (BIO:  $3.03 \pm 0.32$ ; PLA:  $2.52 \pm 0.3$ ). This could suggest that our DOMS protocol was successful in inducing a desired effect. The CPS was used for this study because it was thought that the descriptive redundancy (images and words associated to each number) to the 11-point (0-10) scale would reduce some subjectivity in pain score selection, compared to a more traditional pain analysis tool like the Visual Analogue Scale (VAS).

To our knowledge, only a few studies have induced DOMS and utilized a menthol-based analgesic to counter the effects caused by DOMS (Johar et al., 2012; Hill & Sumida 2002). Although elbow flexor musculature was selected to induce DOMS and different scales were used in order to measure self-reported soreness levels, our findings were similar to both studies in regard to the self-reported pain level after DOMS was induced. Johar et al., (2012) utilized a 10x10 supramaximal eccentric elbow flexor DOMS-inducing protocol using the non-dominant arm as their protocol. They found that after the application of Menthol, participants reported a soreness level of  $1.1 \pm 0.4$  on a VAS, where as their comparison group (application of ice), reported a soreness level of  $3.1 \pm 1.7$ , however pre-intervention scores were not reported. Hill et al., (2002) utilized a similar eccentric elbow flexor DOMS-inducing protocol using the non-dominant arm. They found a greater reduction in VAS scores after the application of a menthol/methyl salicylate gel compared to a placebo or capsaicin gel on a VAS, however they believe

the reduction in pain was likely caused by the active ingredient methyl salicylate and not solely produced by the menthol.

*Subjective pain analysis: Pressure algometry*

Both groups at all four sites (left and right GA-MTJ; left and right VMO) exhibited a significant decrease in pressure threshold after the DOMS-inducing protocol. While there was a significant decrease in pressure threshold after the DOMS-inducing run, it appears as though the menthol-based topical analgesic was not effective in increasing pressure threshold. Pairwise comparisons revealed at all four sites, for both groups, that there was also a significant increase after the intervention application, suggesting perhaps that the light exercise influenced perceived soreness resulting from DOMS (Cheung, Hume, & Maxwell, 2012). Consistent with the CPS data, perhaps a higher dosage of a menthol-based topical analgesic would result in a greater increase in pressure threshold after inducing DOMS. Conflicting studies completed by Chesterton et al., (2007) and Fischer (1990) suggest a minimum clinically important difference (MCID) of 17.39 N/cm<sup>2</sup>, and 14.71 N/cm<sup>2</sup>, respectively. Due to this discrepancy, Chesterton and colleagues (2007) suggested that a MCID may be masked by error when using multiple observers and mean measurements. The right gastrocnemius-achilles myotendinous junction site for the menthol group exceeded both MCID's with the DOMS-inducing protocol but fell just short of the 14.71 N/cm<sup>2</sup> MCID after the application of Menthol. The left gastrocnemius-achilles myotendinous junction and both left and right gastrocnemius-achilles myotendinous junction for the placebo group in our work was

lower than the MCID after the DOMS-inducing protocol and had even smaller changes after intervention applications. The large standard deviation (upwards of  $\pm 20.71$  N) for some of these sites following the DOMS-inducing protocol highlights the variability and subjective nature of these measures. This could be a result of the variety of foot strike patterns exhibited by the participants (rearfoot, midfoot, forefoot) and the variability that they may produce as a result of the DOMS-inducing protocol (i.e. A forefoot striker may exhibit greater DOMS in the plantarflexors as a result of the downhill run than a heel striker due to the greater loading placed on the musculature) (Lieberman et al., 2010). The literature supports the effectiveness of a downhill run to induce global lower limb DOMS (Braun & Dutto, 2003; Dutto & Braun, 2004; Chen et al., 2007a; 2009), however the novel use of a menthol-based topical analgesic to mitigate these effects has not been studied (until now) and lacks comparison. A study by Miller et al., (2003) found that the use of a protease supplementation can aid in recovery from soreness after a DOMS-inducing downhill run, measured using an 11-point pain scale and pressure threshold measures. Combining treatment options that tend to reduce pain and do not have any physiological interactions could be a viable option for further mitigating the effects of DOMS. Hill and Sumida (2002) suggested that the reduction of pain via a methyl salicylate and menthol-based topical analgesic was likely a result of the methyl salicylate and not solely the menthol. They also note that the reduction in pain was likely a result of a counterirritant effect and not any anti-inflammatory effect, as the reduction in pain was short-lived. These conclusions should be considered when treating mild musculoskeletal pain/injury and a multi-modal approach will likely result in

greater reductions in pain and potentially faster recovery (Miller et al., 2003; Hill & Sumida, 2002).

#### *Spatio-Temporal parameters: Stride rate and length*

Stride rate and length have a speed-dependent relationship (Tsatalas et al., 2013) that was affected by a DOMS-inducing protocol in untrained participants. With DOMS, there is typically a reduced ROM at the knee throughout the gait cycle that leads to a reduction in stride length, ultimately leading to an increase in stride rate to accommodate this change in length at a constant speed (Tsatalas et al., 2013; Braun & Dutto, 2003; Dutto & Braun, 2004; Chen et al., 2007a). Although the effects of DOMS on running kinematics is well-researched in untrained populations, we are among the first to study it in trained runners and found no change to either spatio-temporal variable after a DOMS-inducing protocol and application of a topical-analgesic gel. Contrary to the work by Tsatalas et al., (2013) where a significant change to the inverse relationship between stride length and rate were seen after a DOMS inducing protocol, we found no significant changes at any of our selected speeds. Tsatalas and colleagues (2013) had an untrained population and produced a localized DOMS-inducing protocol. At their fastest speed (3.0m/s) the authors found a decrease in stride length ( $150\pm 7$  to  $144\pm 8$  cm,  $\sim -4\%$ ) and an increase in stride frequency ( $137\pm 6$  to  $147\pm 8$  steps per minute,  $\sim +6.8\%$ ), however, our work demonstrated no significant changes, even at our fastest speed of 3.5m/s ( $>1\%$  for both groups, speed-averaged). As Tsatalas et al., (2013) alluded that this may be due to the nature of the DOMS-inducing protocol and the participants selected. Our well-trained participants, relatively low running analysis

speeds and global DOMS-inducing protocol may have contributed to no differences in stride length or frequency.

### *Kinematic Analysis: Ankle kinematics*

Surprisingly, where pressure threshold showed the greatest decrease (right gastrocnemius), kinematic changes at the ankle would have been expected. Only one of our ankle kinematic variables (peak ankle plantarflexion during stance) had a significant change from DOMS to INT during stance. This was unexpected considering previous work in the area, where there were decreases in ROM at the ankle, knee, and hip during stance after a DOMS-inducing protocol (Dutto & Braun, 2004; Tsatalas et al., 2010; 2013; Chen et al., 2007a; 2009). Again, this could suggest that, despite our subjective pain measures, our DOMS inducing protocol was not sufficient in altering our participants mechanics.

Tsatalas et al. (2013) found no changes to ankle kinematics, however their DOMS-inducing protocol (5x15 max eccentric contractions, knee flexors and extensors), did not target the dorsi/plantarflexors. Chen et al., (2007a; 2009) found significant decreases in ankle ROM, however, did not define dorsi/plantarflexion peaks and did not differentiate phases of gait. We found no main effects or interactions and a small group average increase in peak dorsiflexion ( $1.14 \pm 0.35^\circ$ ) after the DOMS-inducing protocol. Based on the loading of the plantarflexors during stance (Lieberman et al., 2010) and the DOMS created in the musculature, we expected to see a change in ankle kinematics.

Peak ankle plantarflexion during stance was somewhat unexpected, where, at the fastest speed (3.5m/s), the menthol group had a  $4.15 \pm 5.96^\circ$  decrease in peak plantarflexion (less plantarflexed position) from baseline to the DOMS-induced run and no change after the application of the intervention ( $-0.23 \pm 5.83^\circ$ ). At the same speed, the Placebo group had a  $1.62 \pm 7.87^\circ$  increase in peak plantarflexion after the DOMS inducing protocol and a  $1.85 \pm 5.04^\circ$  decrease after the application of the placebo gel. Due to low muscle activity at the ankle during the swing phase (Ahn et al., 2014), no changes were expected for ankle ROM during the swing phase. Not surprisingly, there were no main effects or interactions for ROM at the ankle during swing. The work by Tsatalas et al., (2013) also found no significant changes during swing.

#### *Kinematic Analysis: Knee kinematics*

Our DOMS-inducing protocol targeted a global DOMS effect in the lower limbs, rather than an isolating protocol like others (Tsatalas et al., 2010; 2013; Paquette et al., 2017). Based on previous work with a similar protocol (Braun & Dutto, 2003; Dutto & Braun, 2004; Chen et al., 2007a; 2007b; 2009), a reduction in average knee ROM over the entire gait cycle, as well as, reduced peak flexion and extension during both primary phases of gait were expected. We hypothesized a significant Group x Condition x Speed interaction based on the work by Tsatalas et al., (2013) and found a Group x Condition interaction, where speed did not contribute to the differences between conditions. The menthol group exhibited our hypothesized results and the placebo group had a slight increase ( $1.44 \pm 0.16^\circ$ ) in ROM during stance after the DOMS-inducing protocol. Tsatalas

et al., (2013) found significant decreases in knee ROM during the entire gait cycle after a DOMS-inducing protocol; a more flexed knee at foot strike and mid-stance. A more flexed knee during weight-acceptance suggested a reduced ability of the knee musculature to control the knee during gait - these results are expected to be exacerbated at greater speeds. Tsatalas et al., (2013) notes that their findings are contradictory to Paschalis et al., (2007), where their participants had a more extended knee at foot strike and at mid-stance, however, they do note that the difference in DOMS-inducing protocols may be responsible for these differences. Paschalis et al., (2007) utilized an isolating protocol which only targeted the knee flexors (5x15 eccentric contractions using an isokinetic dynamometer). In that regard, it's expected that the differences in study design (DOMS protocol, participant fitness level) could lead to difficulties in direct comparisons with our work. Our average knee ROM during stance are consistent with the work of Dutto and Braun (2004) where there was a decrease in knee ROM during stance from  $30.4 \pm 2.0^\circ$  to  $28.1 \pm 1.4^\circ$  after a similar DOMS-inducing protocol, which suggests that a downhill run to induce DOMS is an effective method even in trained populations.

Both peak knee flexion and extension during stance were trending towards significance ( $p < 0.10$ ) however had relatively small effect sizes (both - 0.121). All of the changes in ROM were consistent between speeds, except for the Menthol group at the fastest speed (3.5m/s). In this case, the peak knee extension angle became more extended both after the DOMS protocol and after the intervention application. Paquette et al., (2017) found that knee flexion ROM decreased during stance as a result of decreased knee flexion at stance. They found no significant change in peak knee flexion

during stance although there was a decrease ( $47.0 \pm 5.1^\circ$  to  $45.8 \pm 5.4^\circ$  48-hours post DOMS-inducing protocol). At the knee, there are clear differences between our findings and previous work, even in well-trained populations where decreases were seen in all participants (Dutto & Braun, 2004). While the findings by Dutto and Braun (2004) were not statistically significant, the trend for knee ROM to decrease was consistent with other works using untrained populations (Chen et al., 2007a; 2009; Tsatalas et al., 2013). Our findings within the Placebo group may have been a result of the DOMS-inducing protocol not causing enough pain to lead to biomechanical changes within a well-trained population.

Similar to the changes found for average knee ROM during stance, average knee ROM during swing had no significant main effects for the expected Group x Condition x Speed interaction. However, there was a significant Group x Condition interaction. Our Menthol group was consistent with Dutto and Braun (2004) where they found a decrease in knee ROM during swing from  $93.5 \pm 3.6^\circ$  to  $90.9 \pm 3.6^\circ$  after a similar DOMS-inducing protocol. The unexpected results from our Placebo group may have been a result of the DOMS-inducing protocol not causing any significant decrement to ROM. Most of this change in our Placebo group appears to have occurred during a shift in peak knee extension during swing as outlined below.

Peak knee flexion during swing had no significant main effects or interactions. Our results for knee flexion during stance and swing are rather surprising given that very similar previous works on trained and untrained participants produced significant decreases in knee ROM (Dutto & Braun, 2004). Peak knee extension during swing had a significant group x condition interaction and a moderate effect size (0.340). Our data



suggests that the Menthol group moved towards a more extended peak knee position during swing while the Placebo group presented the opposite, where the knee was in a more flexed peak knee extension position (decrease in peak knee extension). Both groups had a more flexed peak knee extension position during INT. Changes to peak knee extension during swing were not hypothesized to change and so this is an interesting conclusion. Again, the conflicting directional changes in ROM for our groups may suggest the DOMS-inducing protocol had high variability in its effect on our participants.

#### *Kinematic Analysis: Hip kinematics*

There was a significant condition x group interaction for average hip ROM during stance and a moderate effect size (0.343). The menthol group experienced a decrease in ROM and the Placebo group experienced an increase in ROM after the DOMS-inducing protocol. Our data suggests the menthol group moved to a less extended position during stance and the Placebo group shifted to a more extended position after the DOMS-inducing protocol. The conflicting results after the DOMS-inducing protocol may be a result of some participants in the Menthol group experiencing greater soreness or the Placebo group not experiencing biomechanical deficits at the hip and compensating for any soreness they may have been experiencing. No significant changes at the hip were expected as the intervention gels were not applied to the primary movers of the hip and this is consistent with our findings. Chen et al., (2009) found a significant reduction in hip ROM (up to  $91 \pm 3.7\%$  of baseline at  $90\% \dot{V}O_{2max}$ ) but did not differentiate phases of

gait. Our Menthol group saw a similar decrease ( $87 \pm 1.42\%$  of baseline, speed-averaged), however our Placebo group saw a  $17 \pm 5.38\%$  increase in hip ROM during stance. Contradictory to their earlier findings, Chen et al., (2007a) found no significant change at the hip and note that this may have been due to the use of well-trained participants versus untrained participants (2007b; 2009).

For average hip ROM during swing there were no main effects but a significant interaction of condition x group with a moderate effect size (0.389). Further differences between the groups found the Menthol group decreasing ROM and the Placebo group increasing ROM after the DOMS-inducing protocol. Given the variability between participants individual biomechanics, these findings might further suggest the individual and variable response of well-trained runners to the DOMS-inducing protocol we selected. The Menthol group exhibited a decrease in average ROM during swing ( $2.87 \pm 0.32^\circ$ ) while the Placebo group exhibited an increase ( $5.66 \pm 0.56^\circ$ ) after the DOMS-inducing protocol. Both groups remained relatively unchanged after the intervention applications consistent with our hypotheses for hip ROM during stance where there would be no change due to no intervention application on the primary movers of the hip (hip flexors and extensors). The increase in the Placebo group average hip ROM appears to have occurred by a shift to greater flexion and less extension whereas the decrease the Menthol group experienced appears to have occurred by a shift to less flexion and greater extension.

Our findings for hip kinematics during swing are different than Tsatalas et al., (2013) and Chen et al., (2009). Both studies had untrained participants and Tsatalas et al., (2010) utilized an isolating protocol to induce DOMS in the knee extensors and

flexors. This may have been a factor in the differences with our Placebo group experiencing increased average ROM. Our peak hip flexion was consistent with the work of Tsatalas et al., (2013) who found at their fastest speed (3.0 m/s) peak hip flexion of  $51.3 \pm 5.7^\circ$ , where our Menthol and Placebo groups averaged  $50.90 \pm 5.07^\circ$  and  $43.3 \pm 8.83^\circ$ , respectively.

## Section 6: Considerations

Several unexpected findings were discovered in our analysis that were not consistent with previous work and this section identifies a few speculations as to how these occurred. This study's methodology was based on previous work in the area that found significant increases in self-reported perceived pain (VAS) and significant decrements to spatio-temporal parameters as well as hip, knee, and ankle ROM 48 hours after a downhill run that induced DOMS in untrained populations (Tsatalas et al., 2010; Chen et al., 2007a; 2009). Our expectation was that the downhill run utilized in previous studies with untrained participants would also elicit DOMS in a trained population (Chen et al., 2007a; 2007b; 2009; Eston, 1996). Dutto and Braun, (2004) found that the use of a similar DOMS-inducing downhill running protocol in a trained population was still successful in eliciting deviations to several biomechanical and physiological measures. In some of our measures, rather than a decrease in ROM and decrease in stride length (with a resultant increase in stride frequency), we found small, yet unexpected increases in ROM and stride length (with a resultant decrease in stride frequency). While significant DOMS was induced based on our subjective measures, the menthol gel did not elicit a significant decrease in perceived soreness. There are several considerations within our study design that could have led to these results and they are discussed below.

### *Pain threshold*

One of the common themes with much of the previous work was the use of untrained participants. The participants for this study were largely sampled from the

varsity Cross-Country and Track and Field teams at the university. Runners at this level are well acquainted with exercising while experiencing DOMS, and in some cases even more severe musculoskeletal injuries. Their ability to continue training through most instances of DOMS may have had an impact on our results. The purpose for using a trained population was due to more experienced runners having very low variability from stride-to-stride and day-to-day in kinematic and spatio-temporal parameters (Morgan et al., 1991). It was believed this low variability would lead to more notable, consistent changes if the DOMS-inducing protocol and intervention was effective. One limitation with our study design may have been the participants ability to manage their DOMS-related soreness which may have resulted in the few biomechanical deviations or alternatively an increase in ROM where participants may overcompensate on the knowledge that their gait was being monitored.

#### *Foot strike variance*

Only one study referenced in our work outlined and controlled the foot strike pattern of their participants (Paquette et al., 2017). Given that the population for this study was a group of trained runners, a percentage of forefoot and midfoot strikers not typically seen in novice or untrained populations may have been present (Larson et al., 2011). A meta-analysis by Almeida, Davis, and Lopes (2015) determined upwards of 89% of the population is a habitual rearfoot striker - our study had only 70% rearfoot strikers (determined during gait event selection). Based on an individuals' foot strike pattern, the effects of the DOMS-inducing downhill run may have produced different areas of soreness and changes to biomechanical variables as a result of different mechanics and variability in impact forces (Lieberman et al., 2010). Typically, a forefoot strike, where

much more load is incurred on the plantarflexors of the ankle at foot strike compared to a heel strike may produce much greater DOMS in the plantarflexor musculature. In future research it would be recommended to limit participant inclusion to one gait pattern or design a study to compare groups.

### *Skin-motion artifact*

One of the major drawbacks of motion capture for gait research is error in recordings caused by skin-motion artifact and general unavoidable motion artifact (ie. A reflective marker placed on an area of skin with unavoidable movement). In a validation study, Benoit and colleagues (2005) found significant error with skin-mounted reflective markers even during the stance phase of walking of up to 4.4° compared to directly implanted markers attached to bone. While we did our best during collection to mitigate any skin-motion artifact (avoiding larger areas of soft tissue, firmly affixing markers and rigid bodies to anatomical landmarks), some error may have been produced from sources of motion artifact that are beyond our control for the type of research we conducted.

### *DOMS-Inducing protocol*

Based on previous work with treadmills at varying range of decline from -10% up to -26% (Chen et al., 2009; Dutto & Braun, 2004), a -10% decline was selected as our pilot data showed that perceived soreness and DOMS had been produced by a 30-minute downhill run at -10%. The average speed for the DOMS-inducing protocol for our participants was  $4.08 \pm 0.51$  m/s, which of the studies that included the average speed of their downhill runs, was considerably faster and we believe was an equal means of

producing DOMS in a well-trained population (Chen et al., 2009 -  $3.24 \pm 0.54$ m/s; Chen et al., 2007a -  $3.3 \pm 0.50$ m/s; Chen et al., 2007b -  $3.45 \pm 0.19$ m/s). The global nature of inducing DOMS with a downhill run, variability in foot strike patterns, shoes used, and considerable variability in weekly average mileage ( $42 \pm 23.14$  km/week) may have also contributed to the unexpected results of the DOMS-inducing protocol at the ankle and hip.

### *Warm-up Effect*

It is well known that one of the few treatment options to mitigate the effects of DOMS is light exercise, albeit, the effect is concurrent and temporary (Cheung, Hume, & Maxwell, 2003). For the purpose of this study it was impossible to avoid a potential warm-up effect. Given the trained nature of our participants, the three speeds were selected in order to reduce any fatigue-based changes to kinematics and therefore were relatively light intensity. Our DOMS and intervention trials may have been affected by the DOMS-mitigating effect of light exercise and this could have played a role in some of our unexpected findings.

## Section 7: Conclusion and future directions

Our study has given insight into the use of a menthol-based topical analgesic and its effect on DOMS-induced changes to running biomechanics and pain perception. This thesis suggests that a menthol-based topical analgesic applied to the thigh and calf muscles following DOMS has little effect on running kinematics. This suggests that the menthol-based intervention did not change (restore) running kinematics to baseline (pre DOMS) levels. It should be noted that others found significant decreases in knee ROM after a DOMS-inducing protocol (Braun & Dutto, 2004; Chen et al., 2009; Tsatalas et al., 2013). This could suggest that our DOMS protocol did not change running kinematics to a large enough extent for the menthol-based intervention to have an effect. A recurring difference between our work and other literature is the participants included; most work has used untrained participants, rather than the well-trained participants in this thesis. There are clear contrasts between our measured kinematics and subjective pain scores. Our DOMS-inducing protocol induced DOMS, however, maybe it was not significant enough to cause significant change in kinematics. More standardized DOMS-inducing protocols, kinematic variables, and means of measuring perceived soreness may assist in creating more consistent findings.

All participants had a full recovery from our study by day 5 (Appendix 8.3). We cannot, therefore, infer whether a global lower-limb DOMS inducing protocol would result in biomechanical changes that could be detrimental to performance or lead to further injury in well-trained runners. Future work should consider the use of untrained populations, as the impact of DOMS on running biomechanics may be more substantial and could provide insight into optimal training paradigms in this group.



As previously stated, foot strike pattern should also be controlled in future projects involving the effects of a downhill DOMS-inducing protocol on running-related variables. This may have had an impact on how our DOMS-inducing protocol affected individuals since there can be large variability in individual gait mechanics. Strictly using heel-strike runners as an example (Paquette et al., 2017), controls for another extraneous variable in a study design.

Our work used a 3.5% menthol-based Biofreeze - a higher concentration may have a greater effect, especially on self-reported pain measures. Potentially combining menthol with other treatment options or with other topical counterirritants/anti-inflammatories may have a greater effect (Hill & Sumida, 2002). As seen in our pain journal (Appendix 8.3), perhaps testing 24-hours after when self-reported pain levels were greatest may have caused a greater difference in biomechanical and pain perception variables before and after the intervention applications.

Muscle activity was not measured in this study and future work could attempt to evaluate muscle activity changes due to DOMS in running or other dynamic activity - as previously noted, other works have found significant increases in tetanic contraction after the application of a menthol-based topical analgesic (Johar et al., 2012).

We cannot conclude, based on our findings, whether or not any changes that occurred to kinematic variables would lead to any further injury from the effects of DOMS or the application of a menthol-based topical analgesic, however, the potential for its use to aid in the relief of pain could be beneficial in other populations (untrained participants, mild chronic pain). Tsatalas et al., (2013) notes that performing exercise

while injured could cause changes to running patterns that might have a negative impact on running performance (Highton, Twist, & Eston, 2009; Marcora & Bosio, 2007; Twist & Eston, 2005) and could lead to more severe injury. The short duration of our study and lack of significant changes after the DOMS protocol suggests well-trained runners might be capable of managing DOMS with only minor alterations to biomechanics that may not have any lasting, negative effect on performance or risk of further injury.

## Section 8: References

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## Section 9: Appendices

### 8.1 Informed consent

#### Informed Consent

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Date: **November 19<sup>th</sup>, 2017**

Project Title: The interactive effect of pain and Biofreeze on the biomechanics of running

Principal Investigator (PI): Michael Holmes, Assistant Professor

Department of Kinesiology

Brock University

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Principal Student Investigator: Ryan Gagnon, MSc. Graduate Student

Department of Kinesiology

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#### INVITATION

You are invited to participate in a study that involves research. Delayed Onset Muscle Soreness (DOMS) can be identified as muscular soreness that occurs typically 24-48 hours after intense unaccustomed exercise. DOMS-related muscular pain can lead to functional deficits and altered movement mechanics that can lead to a greater risk of

further injury or sources of pain. The body does this by simply trying to avoid the initial source of pain by adopting some form of compensation (such as a limp when walking) which may help reduce pain at the initial source but lead to another source of pain or risk injury at another joint or limb. DOMS is a common complaint of many runners from novice to expert and due to the increased forces in running, a compensatory pattern in walking is exaggerated in running and can affect the compensating structures to an even greater extent, further increasing the risk of injury. Biofreeze, a topical analgesic, is used to block the pain signal from the affected structures to the brain when applied to muscles experiencing delayed onset muscle soreness. Blocking the pain signal from DOMS should allow an individual to restore their natural movement mechanics.

The purpose of this study is to assess the interaction between Biofreeze and delayed onset muscle soreness and how it affects movement mechanics and muscle function. Individual muscle and limb contributions to movement will be evaluated using motion capture analysis during submaximal running before and after a DOMS-inducing protocol and after the application of Biofreeze.

## **WHAT'S INVOLVED**

As a participant, you will be asked to perform 4 experiments over 2 separate laboratory visits. You will perform three separated submaximal running sessions, as well as a protocol to induce delayed onset muscle soreness. Our intervention (Biofreeze) will be applied on the second laboratory visit only, the effects of Biofreeze typically only last a few hours and cause a mild cooling effect.

## **Experiment Protocol**

Upon arrival to the lab, the investigators will explain and demonstrate all the tasks to you. We will also familiarize you with the equipment being used and verbally explain and review the consent form.

### **Day 1:**

#### **Experiment 1 (Running Analysis - BASELINE):**

Each participant will first be familiarized with laboratory facility (Thistle room 141 - TH141). The motion capture, EMG, Running analysis protocol, DOMS protocol, and

Biofreeze application will all be explained verbally to the participant. Anthropometric (body) measurements will be taken that are used to enter into our motion capture system so that the system knows the relative structure of each participant. Once these objectives have been satisfied, the participant will then be prepared with reflective markers for the motion capture system. The participant will be allowed a period to be familiarized with the treadmill once fully outfitted for motion capture. Once ready, the participant will complete a 5-10 minute submaximal running analysis where the muscle and limb contributions will be measured. Submaximal running will be completed at 2.5, 3.0, and 3.5m/s. The treadmill has a large red emergency stop button that will be shown to each participant prior to any treadmill running and have its use explained and demonstrated to ensure safety of the participant.

### Experiment 2 (Delayed Onset Muscle Soreness):

Once completed, the participant will have some time to rest and remove motion capture reflective markers while preparing to complete the DOMS inducing protocol. The DOMS protocol will consist of participants a 30 minute downhill treadmill running protocol. The participant will first warm up for 5 minute at an easy walking pace on a 0% grade. Once the participant is familiar with the treadmill and is prepared, the treadmill will be set to a -10% grade (decline) and will work up to 85% of their predicted heart rate maximum (PHRM). Once 85% PHRM is achieved, a 30 minute timer will begin and the participant will aim to maintain that level of intensity with monitoring by a spotter that will remain by the participants side. The participant will be clipped into the safety key on the treadmill at all times when on the treadmill.

### **Day 2 (48 hours after Day 1):**

### Experiment 3 (Running Analysis POST-DOMS):

Delayed Onset Muscle Soreness will be measured via Pressure Pain Threshold and Subjective Pain Analysis. Pressure Pain Threshold is measured with the application of manual pressure with an algometer (tool used to measure the amount of force being applied - see Appendix) to the muscles to determine if there is sufficient DOMS to complete the final experiment. This testing will produce mild discomfort. If you decide the protocol is producing greater discomfort than you wish to experience testing will be stopped immediately and you can withdraw from the study with no consequence. EMG and motion capture will be outfitted to participant again in identical locations. Running analysis will be repeated as on Day 1.



#### Experiment 4 (Running Analysis POST-BIOFREEZE):

Participants will have Biofreeze applied to their quadriceps muscles of both legs. After a rest break, running analysis will be repeated as on Day 1. Motion capture outfitting in experiment 3 will be preserved for final running analysis.

#### **Instrumentation**

Once familiarized with all of the tasks, you will be instrumented for our biomechanical measures.

#### 3D Kinematics

Three-dimensional movements of the upper extremity will be tracked using a 10-camera Vicon System (Vicon, Oxford, UK). Individual markers will be placed over various areas of your lower extremities and trunk, including your feet, lower legs, thighs, pelvis, and torso.

#### **Eligibility**

Males and females are eligible to participate (age range, 17-40 years). We are seeking individuals who have a minimum recreational running experience (minimum 20km/week) with no recent or current injuries that affect the ability to run. Any neurological disorders or chronic injuries reported warrant exclusion from participation in this study.

#### **Timeline**

Including instrumentation and experimental setup, it is expected that you will be in the neuromechanics and ergonomics (TH141) laboratory for approximately 1.5 hours for session 1 and 1.5 hours for session 2. There will be two to three sessions in total, separated by at least 48 hours.

#### **POTENTIAL BENEFITS AND RISKS**

There are no known or anticipated direct benefits to you for your involvement in this project. The scientific community will benefit from this research because these findings may lead to changes in the field of neuromechanics and the treatment or

management of pain when one is experiencing delayed onset muscle soreness. These finds can be applied to, but are not limited to running and any physical activity where delayed onset muscle soreness may affect performance including sports, work environments, and activities of daily living.

There may be risk associated with this study. For instance, reflective markers will be affixed to the body either via double-sided tape or a strap that will be snug but will not be so tight as to cause discomfort. This study requires participants to have a menthol based topical analgesic (Biofreeze) applied to their quadriceps muscles on the second experiment day. The sensation is described as a cooling sensation to reduce the affects of pain in the musculature. Biofreeze is the number one clinically recommended topical analgesic for pain reduction. The ingredients in Biofreeze will be provided at the bottom of this document to ensure there are no known allergies or sensitivities to the product before use. Any broken skin, windburn, sunburn, rash, dry or otherwise irritated skin should be reported to the investigator prior to application.

All tasks being simulated for this study are considered to have some risk and involve submaximal running for short durations, and a protocol to induce delayed onset muscle soreness on the first day of the experiment. The submaximal running will be well within each individual's easy running pace. This task will not be uncomfortable and will pose no risk. The protocol to induce delayed onset muscle soreness is an intense task as it requires each individual to work at a level of unaccustomed exercise. Due to the delayed onset muscle soreness protocol, the intention is for each participant to experience delayed onset muscle soreness and therefore discomfort and pain will be expected in the musculature of the lower limbs within 24-48 hours post-protocol. This delayed onset muscle soreness typically lasts for 5 days but sometimes may linger for up to 10 days presenting as a discomfort in the affected musculature. In the very unlikely event of injury (for example, you may experience discomfort to the lower limbs or trunk musculature), we do not have funds in our grant to cover treatment expenses. We encourage any individuals with persistent irritation or discomfort to please visit the Campus Wellness Centre or your healthcare provider.

You are free to withdraw or discontinue the study at any time without consequence. Your withdrawal from the study will not affect your standing at Brock University or otherwise. You will also still be reimbursed for your time if you complete the first day of testing and decide to not return to complete the study. Any participant who has any interaction with an experimenter involved with the study (such as a student in a class under the involved experimenters - ie. A professor or Teaching Assistant) can participate and / or withdraw from the study at any given time and should understand that this will have no effect on their academic pursuits or otherwise at Brock University.

## **CONFIDENTIALITY**

Your identity will be kept confidential and only made available to the researchers. You will be identified only by a subject identification code during the data collection phase of this study. All data, including written records and electronic data, will be placed in a locked cabinet or stored on a secured computer in the locked office of the principal investigator. Data will be originally recorded on a computer that is password protected and only available to the researcher's in a locked and secure room. The data will remain at this institution. The data will not be linked with any other data set and the data will not be sent outside of the institution where it is collected. Any images and videos we release publicly will remain confidential by blurring out any identifying factors of any of the participants involved. This includes the blurring of participants faces. Should you request that your images or video not be released they will be withheld from public release with no consequence to the participant. Data will be kept until publication of the results; this can sometimes take 1-2 years. After this time, all subject identification codes will be removed from the data and kept indefinitely.

Access to this data will be restricted to Dr. Holmes and the graduate student involved in this work.

## **VOLUNTARY PARTICIPATION**

Participation in this study is voluntary. If you wish, you may decline to answer any questions or participate in any component of the study. Further, you may decide to withdraw from this study at any time and may do so without any penalty or loss of benefits to which you are entitled. If you are a Brock student, withdrawing from the study will in no way affect your academic standing. If you wish to withdraw during a study, simply tell the investigator that you no longer wish to participate. If you wish to withdraw between sessions, simply contact the principal investigator.

Participation, non-participation or withdrawal from the study will not affect one's standing at Brock University. If you are a student of the principal investigator (they are your Professor or Teaching Assistant), testing and recruitment will be handled by a third-party individual to avoid real or perceived coercion that you may feel.

## **Compensation for Participation**

You will receive a \$10 Tim Horton's gift card for participating in this study (this means that you have completed both laboratory sessions). You will be reimbursed for your time for each session (so you do not have to complete both sessions for compensation). You can receive a \$5 gift card for each session (day) completed.

## **PUBLICATION OF RESULTS**

Results of this study may be published in professional journals and presented at conferences. Any images and videos we release publicly will remain confidential by blurring out any identifying factors of any of the participants involved. This includes the blurring of participants faces. Feedback about the details of this study and your participation will be available to you by contacting Dr. Holmes at the address at the top of the form after your participation has been completed or after you withdraw from the study if you wish to. Results should be made available approximately 6 months after your completion of the study. The results will be group data about the main findings of the study. If you wish to know more about individual data, we can arrange to meet.

## **CONTACT INFORMATION AND ETHICS CLEARANCE**

If you have any questions about this study or require further information, please contact Dr. Holmes using the contact information provided above. This study has been reviewed and received ethics clearance through the Research Ethics Board at Brock University (File # 16-263). If you have any comments or concerns about your rights as a research participant, please contact the Research Ethics Office at (905) 688-5550 Ext. 3035, reb@brocku.ca.

Thank you for your assistance in this project. Please keep a copy of this form for your records.












## **CONSENT FORM**

I agree to participate in this study described above. I have made this decision based on the information I have read in the Information-Consent Letter. I have had the opportunity to receive any additional details I wanted about the study and understand that I may ask questions in the future. I understand that I may withdraw this consent at any time.

Name: \_\_\_\_\_

Signature: \_\_\_\_\_ Date: \_\_\_\_\_

## COMPARATIVE PAIN SCALE CHART (Pain Assessment Tool)

										
0 Pain Free	1 Very Mild	2 Discomforting	3 Tolerable	4 Distressing	5 Very Distressing	6 Intense	7 Very Intense	8 Utterly Horrible	9 Excruciating Unbearable	10 Unimaginable Unspeakable
<b>No Pain</b>	<b>Minor Pain</b>			<b>Moderate Pain</b>			<b>Severe Pain</b>			
Feeling perfectly normal	Nagging, annoying, but doesn't interfere with most daily living activities. Patient able to adapt to pain psychologically and with medication or devices such as cushions.			Interferes significantly with daily living activities. Requires lifestyle changes but patient remains independent. Patient unable to adapt pain.			Disabling; unable to perform daily living activities. Unable to engage in normal activities. Patient is disabled and unable to function independently.			

FOR PROFESSIONAL RE-SALE ONLY

ROLL-ON

### Drug Facts

**Active Ingredients:** Menthol USP 5%  
**Purpose:** Cooling Pain Relief

**Uses:** Temporary relief from minor aches and pains of sore muscles and joints associated with arthritis, backache, strains and sprains

### Warnings:

#### For external use only

**Flammable:** Keep away from excessive heat or open flame

**Ask a doctor before use if you have:** Sensitive skin

**When using this product:** Avoid contact with the eyes or mucous membranes; Do not apply to wounds or damaged skin; Do not use with other ointments, creams, sprays or liniments; Do not apply to irritated skin or if excessive irritation develops; Do not bandage; Wash hands after use with cool water; Do not use with heating pad or device; Store in a cool dry place

**Stop use and ask a doctor if:** Condition worsens, or if symptoms persist for more than 7 days, or clear up and recur

**If pregnant or breast-feeding:** Ask a health professional before use

**Keep out of reach of children:** If accidentally ingested, get medical help or contact a Poison Control Center immediately

### Directions:

**Adults and Children 2 years of age and older:** Rub a thin film over affected areas not more than four times daily; massage not necessary

**Children under 2 years of age:** Consult physician

**Inactive Ingredients:** Aloe Barbadosis Leaf Extract, Arctium Lappa Root (Burdock) Extract, Arnica Montana Flower Extract, Blue 1, Boswellia Carterii Resin Extract, Calendula Officinalis Extract, Camellia Sinensis Leaf Extract, Carbomer, Glycerin, Ilex Paraguariensis Leaf Extract, Isopropyl Alcohol, Isopropyl Myristate, Melissa Officinalis (Lemon Balm) Leaf Extract, Silica, Tocopheryl Acetate, Triethanolamine, Water, Yellow 5

**Questions or Comments:** 1-800-246-3733

SOOTHING MENTHOL



for arthritis, back pain, sore muscles and joints

**BIOFREEZE**  
**PROFESSIONAL**  
VANISHING SCENT • PARABEN-FREE



PERFORMANCE HEALTH

3 fl oz / 89 mL

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NDC 59316-117-10

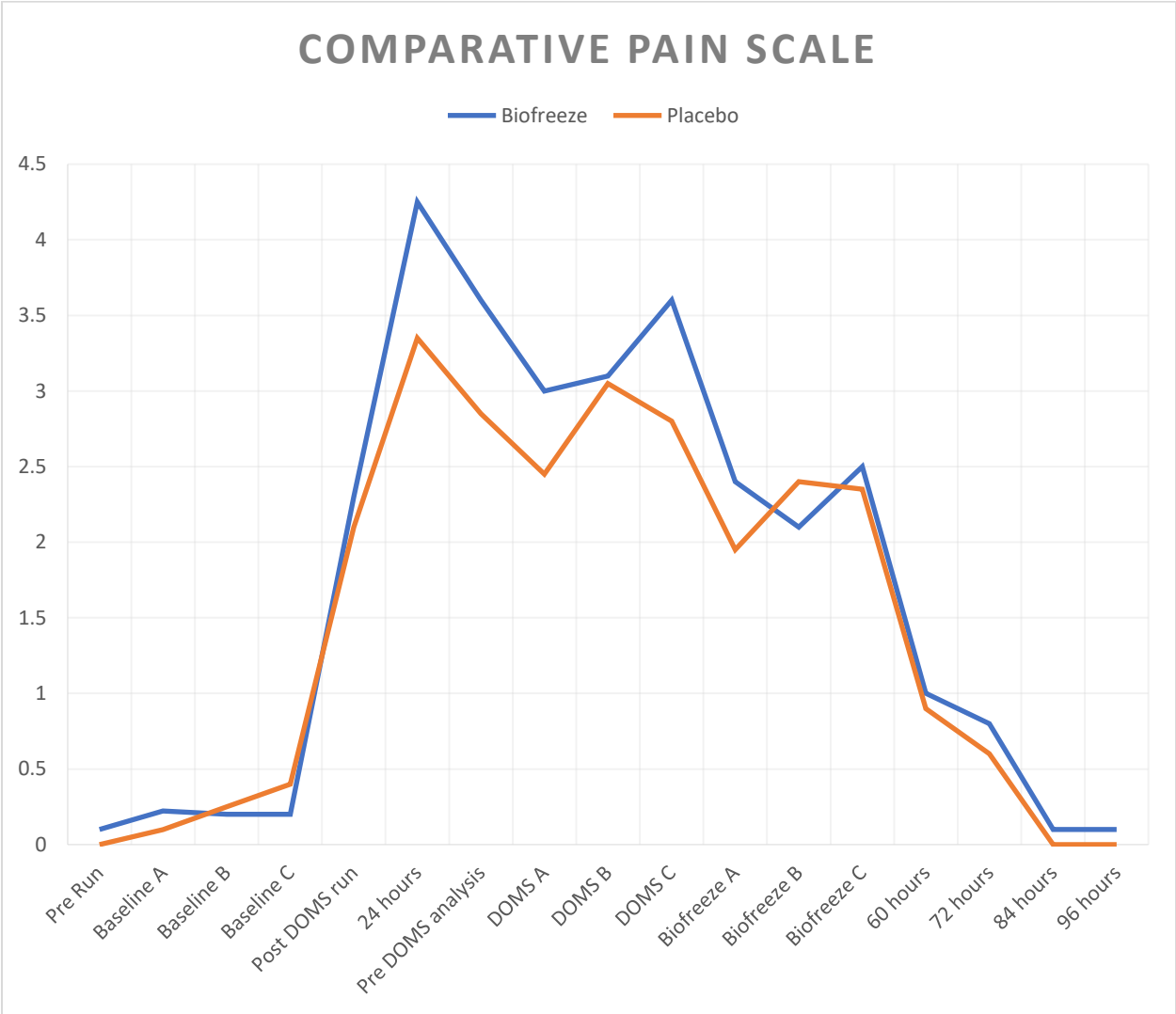
**Does not contain NSAIDs, Ibuprofen, Aspirin, or Salicylate**



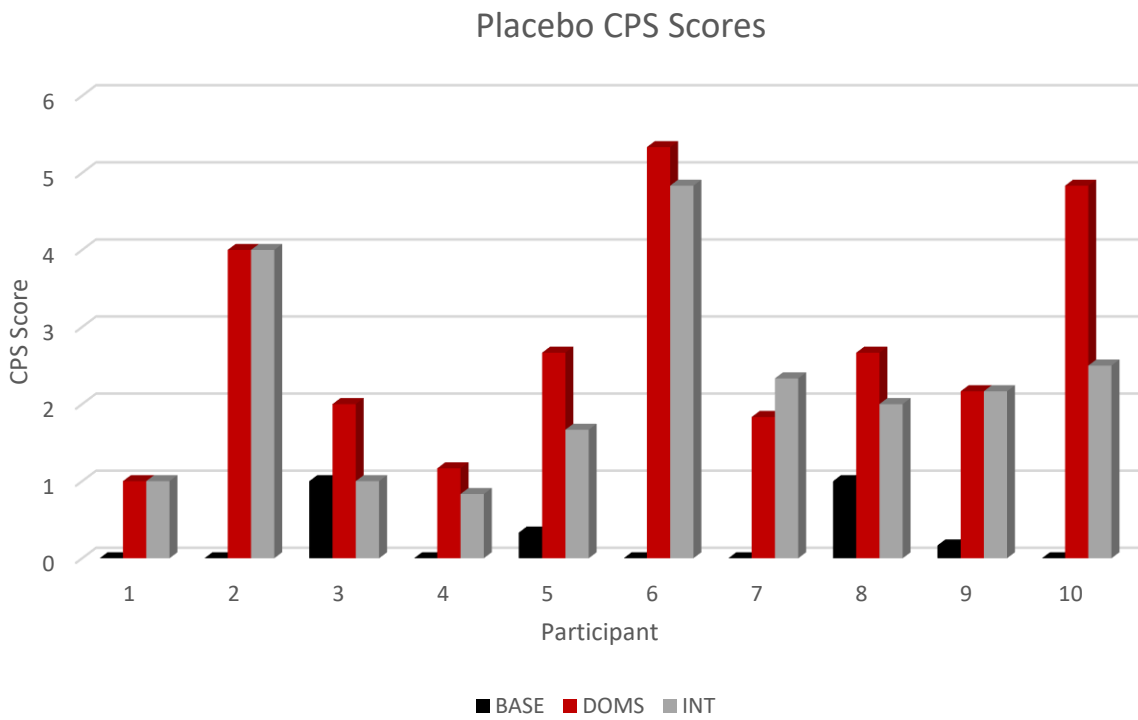
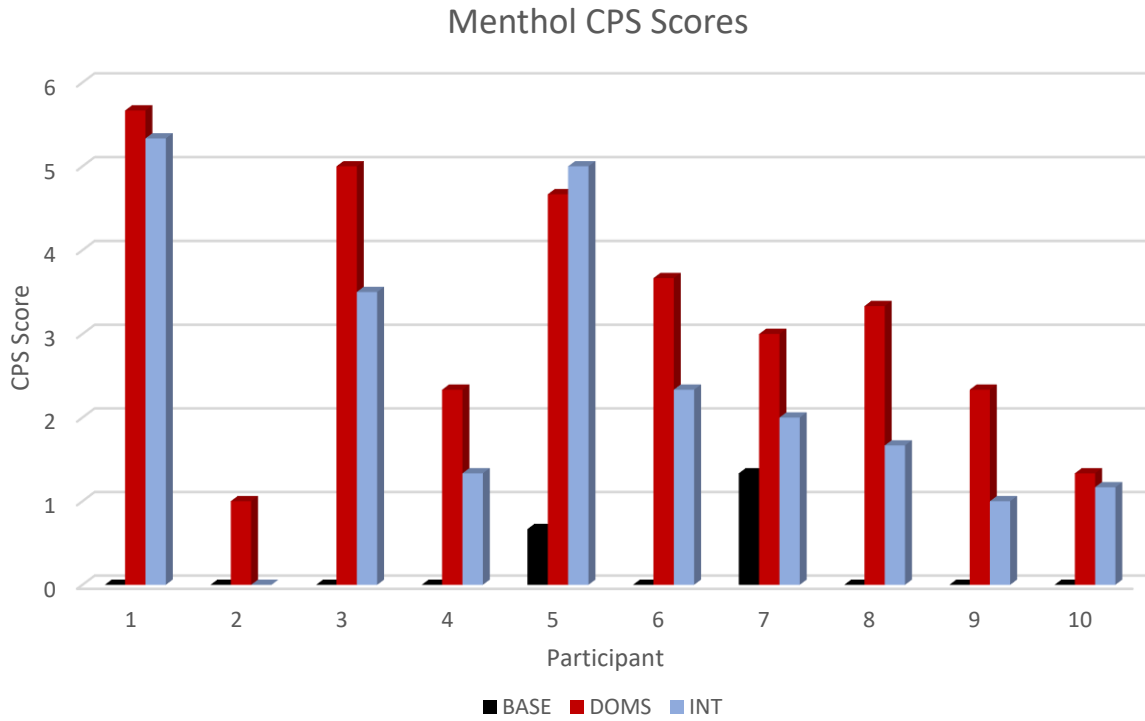
## 8.2 Speed translations

<b>m / s</b>	<b>2.5</b>	<b>3.0</b>	<b>3.5</b>
m / min	150	180	210
mph	5.6	6.71	7.83
km/h	9	10.8	12.6
min/mile	10:43	9:00	7:39
min/km	6:40	5:33	4:45

### 8.3 Pain journal

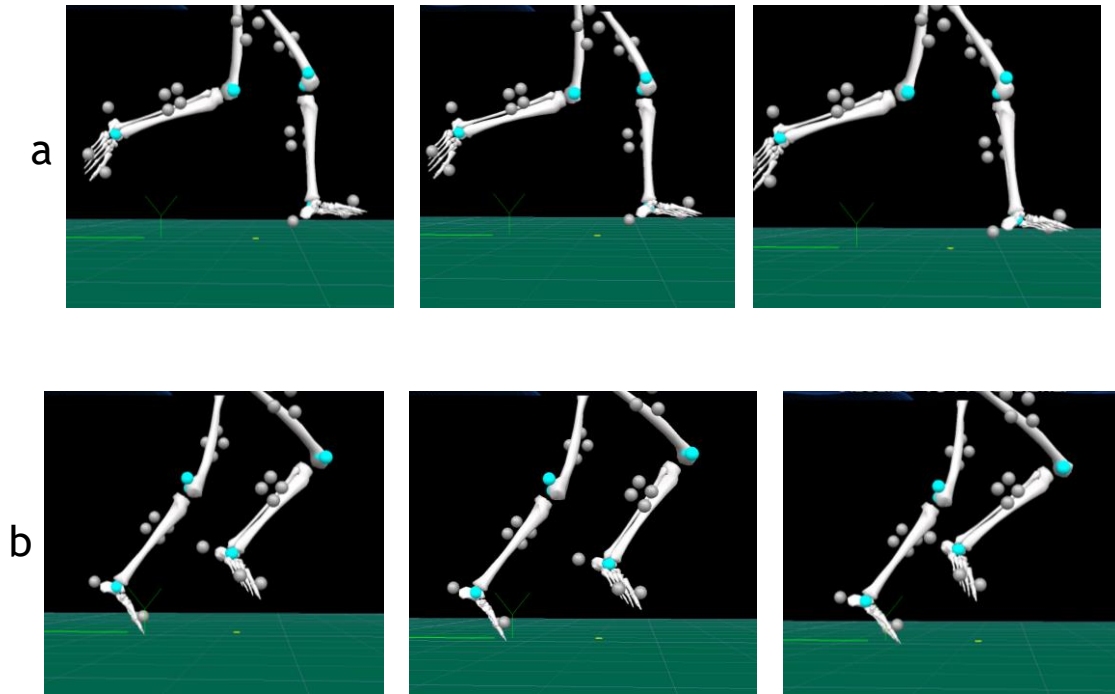


## 8.4 Individual CPS / Pressure threshold responses



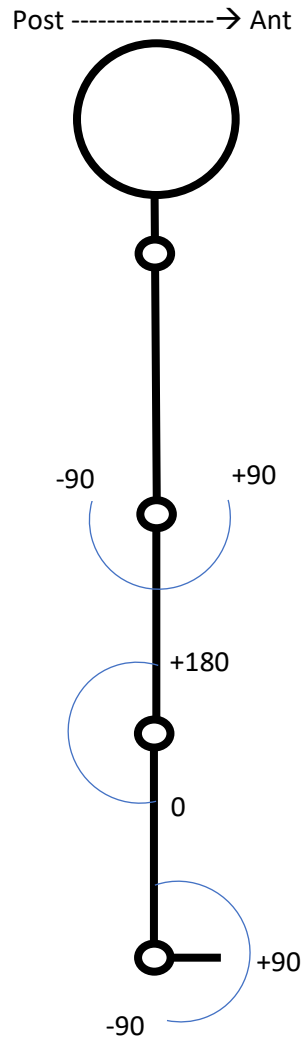


## 8.5 Gait event selection



Visual representation of the gait event selection accuracy. (a) Represents three consecutive foot strikes from a randomly selected participant. (b) Represents three consecutive toe offs from the same participant.

## 8.6 Joint angle directions



Hip: where  $+90^\circ$  is flexion and  $-90^\circ$  is extension

Knee: where  $0^\circ$  is extension and  $+180^\circ$  is flexion

Ankle: where  $+90^\circ$  is neutral ( $>90^\circ$  dorsiflexion) and  $-90^\circ$  is plantarflexion