The Impact of a Single Intermittent Pneumatic Compression Bout on Performance, Inflammatory Markers, and Myoglobin in Football Athletes

by

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ABSTRACT

Intermittent Pneumatic Compression (IPC) use as a tool for recovery after exercise has recently become widespread among athletes. While there is strong anecdotal support for IPC, little research has been done to show its effectiveness in recovery. Eight collegiate football athletes were recruited and subjected to IPC or control conditions in a randomized crossover manner during off-season training. Countermovement jump (CMJ) and 10m sprint were evaluated before training, at 3 and 24 hours following training. Self-reported soreness, blood markers of inflammation [interleukin-6, interleukin-10, and monocyte chemoattractant protein-1 (MCP-1)] and muscle damage (myoglobin) were measured before training, post-training, post-recovery and at 3 and 24 hours post-training. Significant time effects were observed in MCP-1 and myoglobin ($p < 0.05$) indicating an inflammatory response and muscle damage. No group differences ($p > 0.05$) were observed between recovery interventions for all measures, suggesting that the IPC protocol used was not effective in this population.

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CHAPTER 1 – SCIENTIFIC FRAMEWORK

1.1 Introduction

Physical recovery is a key component of athletic training. Without adequate recovery, the physical demands of an athletic lifestyle can slowly become detrimental and lead to overreaching, overtraining, and decreased performance (Kreher & Schwartz, 2012).

To promote quicker physical recovery – which is to say, recovery of the nervous and muscular systems – athletes have utilised many methods to help restore their baseline levels of performance after exhaustive training and competition (Cheung, Hume, & Maxwell, 2003).The more common modalities thought to improve the rate of recovery include: cryotherapy, active exercise recovery, nonsteroidal anti-inflammatory drugs (NSAIDs), compression garments, massage, stretching and ultrasound (Ascensão, Leite, Rebelo, Magalhães, & Magalhães, 2011; Barnett, 2006; Cheung et al., 2003; Crane et al., 2012; Goto & Morishima, 2014; Hasson et al., 1993; Khamwong, Pirunsan, & Paungmali, 2011; Rantanen, Thorsson, Wollmer, Hurme, & Kalimo, 1999; Roberts, Raastad, et al., 2015; Vaile, Halson, Gill, & Dawson, 2008). In recent years, however, there has been the introduction of a new modality among athletes and sports: intermittent pneumatic compression (IPC; Cochrane, Booker, Mundel, & Barnes, 2013; NormaTec, n.d.-c).

IPC devices are large sleeves designed to cover either the upper or lower limb. These sleeves are divided into individual cells, which are all connected to an electrical compressor that can inflate each cell independently. This allows the cells to be inflated in any order and at different pressures. Many IPC units have adjustable settings, where the sequence of the inflation, as well as the level of pressure exerted can be adjusted. NormaTec claims to have initiated the

application of IPC within the athletic community and introduced their sequential pulse technology (NormaTec, n.d.-c), where the sequence of the compression begins from the most distal of five cells, and cells inflate in sequence towards the most proximal cell of the IPC unit (NormaTec, n.d.-c).

The origin of IPC can be found in the medical field, where it is beneficial to patients with various conditions that negatively impact blood flow in the extremities, such as deep vein thrombosis (Ho & Tan, 2013; Zareba, Wu, Agzarian, Rodriguez, & Kearon, 2014), lymphedema in the lower limb (Muluk, Hirsch, & Taffe, 2013), and venous leg ulcers (Comerota & Arbor, 2011). While unconfirmed in the scientific literature, the company claims that the IPC device can generate pressure up to 100 millimetres of mercury (mmHg) (NormaTec, n.d.-b) compared to 30 mmHg in compression garments (Brophy-Williams, Driller, Shing, Fell, & Halson, 2015), which might explain why IPC has been demonstrated to be highly beneficial at improving blood flow in clinical populations (Abu-Own, Cheatle, Scurr, & Coleridge-Smith, 1993). While the exact physiological mechanisms of muscle recovery that might benefit from IPC are yet to be uncovered, many athletes currently use the devices (Cochrane et al., 2013) and investigation into their effectiveness is warranted. The purpose of this study was to compare IPC as a recovery tool to a control condition following training in football athletes using physical performance tests, inflammatory and muscle damage markers, and self-reported soreness.

1.2 Review of Literature

1.2.1. Recovery. When investigators examine recovery following exercise, it is often described as a return to baseline values that were measured before the exercise intervention (Kellmann, 2002; Tomlin & Wenger, 2001). Acute bouts of high intensity exercise result in

physiological changes that are detrimental to maintaining physical performance without adequate recovery time (Kellmann, 2002). Physiological variables observed can include heart rate, which can return to baseline in as little as 15 to 30 minutes of passive rest (Wu, Hsu, & Chen, 2005), while other parameters can take several hours (e.g. excess post-exercise oxygen consumption [Børsheim & Bahr, 2003]), or days (e.g. delayed-onset muscle soreness [Armstrong, 1984]) to return to baseline. When looking at recovery methods, researchers can examine the time it takes to return to baseline using tools such as subjective scales of soreness or fatigue with the participants (Draper, 2014; Glasgow, Ferris, & Bleakley, 2014; Vaile, Halson, Gill, & Dawson, 2008), blood samples (Ihalainen et al., 2014; Philippou et al., 2009) and muscle biopsies (Gibala et al., 2006; Peterson et al., 2003; Staron et al., 1992).

For the purpose of the current study, full recovery after a single muscle damaging bout of exercise will be defined as a return to measures taken at baseline.

1.2.2 Muscle Damage. Muscle damage can occur as a result of various stresses, often mechanical and chemical in nature (Brancaccio, Lippi, & Maffulli, 2010). In terms of exercise induced muscle damage (EIMD), eccentric muscle action, where the muscle contracts as it is lengthening, have been shown to cause greater amounts of EIMD compared to concentric and isometric muscle actions (Newham, Mills, Quigley, & Edwards, 1983; Pokora, Kempa, Chrapusta, & Langfort, 2014; Poprzęcki, Staszkiewicz, & Hübner-Woźniak, 2004). Concentric and isometric muscle actions involve the shortening or the maintenance of tension with no movement in the muscle (respectively). At the cellular level, the controlled distancing of muscle sarcomere Z-lines during eccentric muscle work leads to disruption of the these Z-lines (Clarkson & Hubal, 2002; Fridén & Lieber, 2001; Fridén, Sjöström, & Ekblom, 1981; Lieber & Fridén, 1988) and thus leads to damage of the muscle fibers. Concentric muscle actions, on the

other hand, involve the approximation of the Z-lines, and they do not experience the same disruption as eccentric muscle actions (Clarkson & Hubal, 2002; Clarkson & Sayers, 1999). Both structural and contractile elements of the muscle cell can be damaged following muscle damaging exercise protocols (Clarkson & Hubal, 2002; Hill, Thompson, Ruell, Thom, & White, 2001), with decrease in force production attributed to contractile protein damage (Brown, Child, Donnelly, Saxton, & Day, 1996; Hill et al., 2001).

While the type of contraction plays a large role in EIMD, several other factors might influence the severity of EIMD following training. Given that lengthening contractions lead to greater damage to muscles, muscles that have shorter fascicles (reduced lengthening capacity), such as pennate muscles, are believed to be more prone to injury in both controlled EIMD settings and in sporting environments (Brockett, Morgan, & Proske, 2004; Timmins, Shield, Williams, Lorenzen, & Opar, 2016). Depending on the length of a muscle, there might be certain ranges in which the muscle is more susceptible to damage. Brockett et al. (2004) found that track athletes that suffered hamstring injuries had peak torque production for that muscle at a shorter muscle length. The authors suggested that the optimal power at shorter lengths meant that when the muscle was loaded in a lengthened position, it would be less likely to withstand the stress and suffer damage.

EIMD has also been found to be greater in untrained compared to trained individuals with the same relative workloads, indicating that training causes muscle fibers to be more resistant to damage (Evans et al., 1986; Koch, Pereira, & Machado, 2014; Newton, Morgan, Sacco, Chapman, & Nosaka, 2008; Vincent & Vincent, 1997). While this has only been demonstrated using eccentric training in humans, rat models have demonstrated that both eccentric (Virgen-

Ortiz, Marin, Trujillo, Huerta, & Muñiz, 2008) and concentric training could elicit this response (Hughes & Gosselin, 2002).

Quantifying muscle damage. In research examining muscle damage from exercise protocols, or the effectiveness of recovery modalities, it is essential that muscle damage is evaluated in some manner. There are several methods to estimate whether muscle damage is occurring, which include taking muscle biopsies, magnetic resonance imaging, muscular strength tests, and blood protein examination (Clarkson & Hubal, 2002). Currently, creatine kinase (CK), myoglobin (Mb), and lactate dehydrogenase are among the more common blood proteins being used to indirectly assess muscle damage (Warren, Lowe, & Armstrong, 1999). A common characteristic of these proteins is that under homeostatic conditions, they are only found in small concentrations in the blood, but following injury to either skeletal or cardiac muscle, there is a substantial increase in the concentration of these markers within the systemic circulation (Clarkson & Hubal, 2002; Koch et al., 2014; Nie et al., 2011). Measuring and quantifying these proteins and examining the change from baseline measurements could give an idea on the degree of muscle damage occurring in the body. The reliability of blood protein measurement as an indicator of muscle damage has been debated (Koch et al., 2014; Warren et al., 1999). Warren et al. (1999) discuss that the time course in performance drop from EIMD does not correlate with any blood protein concentrations. CK also presents high inter-individual variability, and that some individuals might be high-responders, having a greater CK response to the same relative exercise (Koch et al., 2014). On the other hand, Kanda et al. (2013) found a correlation with Mb concentrations and self-reported soreness 72 hours following exercise, and discussed that Mb could be used as a sensitive marker to EIMD. Kanda, Sugama, Sakuma, Kawakami, & Suzuki (2014) argued that the discrepancy between functional detriments and blood protein

concentrations might be that EIMD does not cause sufficient membrane damage to allow the proteins to leave the cell, but that secondary damage from leukocytes at the muscle might allow them to enter the circulation (see sections 1.2.3).

1.2.3 Inflammatory Response to EIMD. EIMD is an insult to skeletal muscle and is subject to an inflammatory response that is reflective of the magnitude of damage to the muscle (Bruunsgaard, Galbo, Johansen, Maclean, & Pedersen, 1997; Paulsen, Mikkelsen, Raastad, & Peake, 2012). The inflammatory process is characterized by the accumulation of leukocytes at the site of injury, which include neutrophils, initially, followed by monocytes, with both of these playing a role in the degradation of damaged tissue and the repair of that tissue (Paulsen et al., 2012; Tidball, 2005). Leukocytes arrive at the injured muscle tissue through an extensive sequence of signalling by proteins, secreted by various cells (Paulsen et al., 2012).

Leukocytes. In order to promote healing and the regeneration process in EIMD, leukocytes need to be able to access the injured muscle cells. When there is skeletal muscle damage, macrophages already found within the damaged tissue (resident macrophages) (Medzhitov, 2008) and satellite cells (Chazaud et al., 2003) are activated from a quiescent state, and secrete interleukin-1β (IL-1β), monocyte chemoattractant protein-1 (MCP-1), and Tumour Necrosis Factor Alpha (TNF-α), among other substances (Dinarello, 1997; L. L. Smith et al., 2000). Satellite cell activation can result from multiple sources, including hepatocyte growth factor (HGF) which is mediated by Nitric Oxide (NO; Anderson, 2000, 2016; De Palma & Clementi, 2012).

Activation of cell adhesion molecules (CAMs) follows the release of TNF-α and IL-1β by the activated satellite cells and macrophages (Jung, Norman, Scharffetter-Kochanek, Beaudet, &

Ley, 1998; Neish, Williams, Palmer, Whitley, & Collins, 1992). CAMs are found on the membrane of endothelial cells of blood vessels and form a large part of the process of extravasation, which is the manner by which leukocytes leave the circulation to reach damaged tissue (Luster, Alon, & von Andrian, 2005). If a leukocyte comes into contact with CAMs when they are active, there is the possibility that a chemical bond will be created with molecules found on the cell membrane, and the leukocyte will get 'caught' to the endothelium (Kolaczkowska & Kubes, 2013; Yago et al., 2004; Zuchtriegel et al., 2015). The leukocyte begins a rolling motion, and the cell velocity begins to slow down as more bonds are created with CAMs (Kolaczkowska & Kubes, 2013). Once stopped, the cell then moves from the circulatory system and into the tissue area, where they begin to exert their effects in the inflammatory and repair process (Jung et al., 1998; Kolaczkowska & Kubes, 2013).

Neutrophils are the first leukocytes to arrive, generally within the first couple of hours following the injury (Kharraz, Guerra, Mann, Serrano, & Muñoz-Cánoves, 2013). Once extravasated and in the tissue, neutrophils begin phagocytosis of damaged tissue (Pizza, Peterson, Baas, & Koh, 2005). Neutrophils are essential in the cascade of inflammation and repair (Tidball, 2005), and the phagocytic actions of the neutrophils leads to the release of reactive oxygen species (ROS) (Pizza, 2008; Pizza et al., 2005; Tidball, 2005). While ROS can cause oxidative damage to healthy muscle cells and lead to secondary damage (not directly from the exercise bout, but from ROS), they are a necessary part of the repair process (Lockhart & Brooks, 2008). It has been shown that ROS play a role in signalling nuclear factor-κB pathway (Fialkow, Wang, & Downey, 2007), which is responsible for the release of several downstream cytokines (A. Chen et al., 2011). The exact influence of neutrophils on the repair of muscle cells, rather than the clearing of debris, is still mostly unknown (Tidball, 2005). Nevertheless, studies

in mice have shown that both immunosenescense (reduction in immune function as a result of aging) and genetic manipulation of phagocytic capacity of neutrophils lead to diminished muscle repair (Grounds, 1987), possibly from hampered activity in the inflammatory process.

In the ensuing stages of inflammation, macrophages begin to arrive at the injury site (Pizza, 2008). An important distinction must be made between two major subsets of the macrophages, as they have very different functions from one another. The M1 subset is known to be phagocytic and might cause secondary damage, similar to neutrophils (Rigamonti, Zordan, Sciorati, Rovere-Querini, & Brunelli, 2014). It has been demonstrated that M1 macrophages signal satellite cell proliferation (Chazaud et al., 2003). The M2 subset is primarily for repair of the tissue and is present at the injury site later than M1 subsets (Massimino et al., 1997; Tidball, 2005).

Markers of inflammation. In response to injury, cells within the sequence of inflammation secrete various substances to coordinate the proper sequence of events mentioned in the previous section. Some promote inflammation, while others are present as part of a feedback cycle and are anti-inflammatory in attempt to reduce excessive inflammation (Dinarello, 1997; Pedersen & Hoffman-Goetz, 2000). When these signalling proteins are secreted by leukocytes or the damaged muscle, as part of an immune response, they can be quantified to get an estimate of the level of inflammation in the body (Bruunsgaard et al., 1997). For example, Suzuki et al. (1999) found that neutrophil cell counts were strongly correlated with inflammatory and muscle damage marker concentrations (IL-6, CK and Mb) following endurance exercise.

The MCP-1 produced and secreted by resident macrophages attracts monocytes from the circulatory system to the injury site, where they convert to macrophages (Chazaud et al., 2003, 2009). These macrophages in turn secrete IL-6, IL-1β, and ROS as a result of tissue degradation, which is thought to promote a pro-inflammatory response by further attracting immune cells to the injury site. Zhang et al. (2013) found that removing the gene coding for IL-6 in mice and chemically inducing muscle damage led to lower myogenin and MyoD expression (indicative of reduced fiber regeneration), macrophage infiltration, lower pro and anti-inflammatory cytokines, and lower MCP-1 when compared to the control that did not receive genetic alterations. It is well documented that IL-6 is also released from contracting skeletal muscles (Febbraio & Pedersen, 2002). Thus, the IL-6 signal is essential to help coordinate the inflammatory sequence and is also utilized to help control the metabolic needs of the muscle. IL-6 may also be described as anti-inflammatory in the context of post-exercise inflammation (Pedersen & Febbraio, 2008; Walsh, Gleeson, Shephard, et al., 2011). IL-6 is a signal for IL-10 and IL-1 receptor antagonist (IL-1ra) protein, both anti-inflammatory proteins which make their way into the circulation later in the inflammatory sequence (Steensberg, Fischer, Keller, Møller, & Pedersen, 2003). Similarly, IL-6 is thought to be an inhibitor of TNF-α which is a strong pro-inflammatory cytokine (Starkie, Ostrowski, Jauffred, Febbraio, & Pedersen, 2003). One of the major differences between the immune response to exercise and other inflammatory conditions (e.g. bacterial and viral infections, sepsis) is the appearance of TNF-α, where in exercise there is rarely an appreciable increase in systemic concentrations of the marker (Walsh, Gleeson, Pyne, et al., 2011).

Further in the inflammatory cycle, M2 macrophages produce IL-10, a cytokine that is anti-inflammatory in nature. IL-10 secretion has been demonstrated to promote myoblast proliferation and subsequent muscle regeneration (Deng, Wehling-Henricks, Villalta, Wang, &

Tidball, 2012). Similarly, IL-10 inhibits the pro-inflammatory M1 macrophages, and is thought to counterbalance the inflammatory environment surrounding injured tissue (Couper, Blount, & Riley, 2008). M2 subtypes are thought to be recruited to injured tissue via interleukin-4 (Loke et al., 2007; Mosser & Edwards, 2008).

Skeletal Muscle Repair. During the degeneration of muscle cells by the leukocytes, activated satellite cells begin proliferating and migrating to damaged muscle fibers (Shortreed, Johnston, & Hawke, 2008; Tedesco, Dellavalle, Diaz-Manera, Messina, & Cossu, 2010). Various chemoattractants contribute to the activation and migration of the satellite cells, including HGF (Tatsumi, Anderson, Nevoret, Halevy, & Allen, 1998), insulin-like growth factor (Musaro, 2005), NO (Anderson, 2000; Wozniak & Anderson, 2007), and transforming growth factor-β (Bischoff, 1997). During the proliferation phase, the satellite cells undergo an asymmetrical mitotic division, where one of the cells continues to differentiate, while the other remains a satellite cell, likely to maintain adequate satellite cell population for future demand (Kuang, Gillespie, & Rudnicki, 2008; Kuang, Kuroda, Le Grand, & Rudnicki, 2007; Schultz, 1996). The differentiating satellite cells convert to myoblasts, which will then fuse with existing undamaged myocytes to reform the muscle tissue (Hurme & Kalimo, 1992; Järvinen, 2005; Järvinen, Järvinen, & Kalimo, 2013; Molnar, Ho, & Schroedl, 1996).

1.2.4 Delayed-Onset Muscle Soreness. Delayed-Onset Muscle Soreness (DOMS) can be described as a dull aching pain, localised in the exercised muscle(s), which tends to manifest approximately 24-72 hours after exercise, especially following eccentric exercise (Armstrong, 1984). DOMS is often more severe in individuals undergoing novel exercise demands (Byrnes & Clarkson, 1986) and following more intense and longer duration bouts of exercise (Talag, 1973; Tiidus & Ianuzzo, 1982).

As it stands, the exact mechanism(s) that cause DOMS is/are not clear, which renders the task of using specific physiological targets to reduce or treat DOMS much more difficult.

Hypotheses of the cause of DOMS. Cheung, Hume, and Maxwell (2003), and Gulick and Kimura (1996) summarised several current and older hypotheses which try to explain the physiological source of DOMS: lactic acid, muscle spasm, connective tissue damage, enzyme efflux, muscle damage, and inflammation are all possible physiological causes that have been suggested in the research literature. Although the literature is rather inconclusive on the physiological source of DOMS, some authors suggest that DOMS is multifactorial and stems from a combination of the theories listed above (Cheung et al., 2003; Gulick & Kimura, 1996; Lewis, Ruby, & Bush-Joseph, 2012); however, the inflammatory response to EIMD is the most plausible physiological explanation that accounts for DOMS.

Some authors have argued that the neutrophils and M1 macrophages responding to EIMD secrete various substances, such as prostaglandins and histamine, that might have an effect of sensitising nociceptors in the area (Armstrong, 1984; Cheung et al., 2003; Ely et al., 2016; L. L. Smith, 1991). Prostaglandin E, released by macrophages (Chensue & Kunkel, 1983) can trigger group III and IV afferent nociceptor neurons, which are thought to cause the dull, aching soreness (Griesbacher & Lembeck, 1987; Moriyama et al., 2005; L. L. Smith, 1991). On the other hand, a few authors claim that the release of histamines, bradykinins and prostaglandins from the damaged tissue as a result of the original injury, or secondary damage by phagocytic reactions, cause an osmotic gradient (Cheung et al., 2003; Gulick & Kimura, 1996). This gradient could promote a fluid shift to the damaged muscle area, causing edema build-up and pressure against the damaged tissue, which would stimulate nociceptors (Cheung et al., 2003; Gulick & Kimura, 1996). Using changes in limb girths to estimate edema accumulation from

exercise is inconclusive, as some studies showed no increase in limb volume (Cornish & Johnson, 2014; Lightfoot, Char, McDermott, & Goya, 1997; Vaile et al., 2008) while some did (French et al., 2008; Kraemer et al., 2001; Talag, 1973). While the literature is not conclusive on the topic, it is likely that DOMS stems from the inflammatory cascade resultant of EIMD.

1.2.5 Performance impact of DOMS and EIMD. Research has shown that several physical performance factors can be impacted by both DOMS and EIMD (Cheung et al., 2003; Clarkson & Hubal, 2002; Clarkson & Sayers, 1999; Gulick & Kimura, 1996; Lewis et al., 2012).

Muscular strength is perhaps the most commonly measured variable with regards to physical abilities, in particular isometric maximal voluntary contractions (MVC) which are measured using dynamometers (Clarkson & Newham, 1995; Twist & Eston, 2005). Studies looking at changes in muscle strength after a bout of eccentric exercise are almost unanimous in demonstrating a decrease in muscular strength from baseline to post-testing (Byrne & Eston, 2002; Denegar & Perrin, 1992; Fridén & Lieber, 2001; Jakeman, Byrne, & Eston, 2010; Lapointe, Frenette, & Côté, 2002; Zainuddin, Newton, Sacco, & Nosaka, 2005). For example, Zainuddin et al. (2005) saw a decrease in isometric MVC of 40% from baseline one day after an eccentric exercise protocol consisting of 10 sets of 6 repetitions of the elbow flexors.

Another physical performance related factor that is affected after an exercise bout is flexibility, defined as the range of motion (ROM) about a joint or series of joints (Denegar & Perrin, 1992; Khamwong et al., 2011; Saxton et al., 1995; Zainuddin et al., 2005). Decreases in ROM have been found to last as long as 8 days following 5 sets of 60 eccentric repetitions of the wrist extensors (Khamwong et al., 2011).

Neuromuscular function can also be altered following EIMD. Saxton et al. (1995) had 12 participants undergo 50 eccentric contractions of one forearm flexor, while their other arm was used as control. Neuromuscular ability was evaluated with three tests: having the participants hold their arms contracted at 90° of flexion while deviations from the position were monitored via accelerometry; joint position sense at various angles were compared to the control arm; and by comparing differences in force production between each arm while being told to maintain identical tension. It was found that joint position was not significantly altered; however, the participants had significantly greater deviations while holding the arm in place and significant difference in force proprioception.

Strength and flexibility loss as well as altered neuromuscular functioning can all have an impact on sport performance. Athletes executing a motor skill requiring a specific force and movement pattern learned and practiced under conditions with minimal soreness or muscle damage might suffer performance detriments when affected by muscle damage or soreness. For example, when looking at tasks such as walking and running, Paschalis et al. (2007) found that joint kinematics were greatly altered after muscle damage in the knee extensor muscles. In particular, the authors found that the ROM in the knee joint was reduced, and discussed that it was likely a subconscious change in muscle recruitment to reduce the use of the quadriceps to avoid further damage or pain. To further support this, a recent review summarised that running economy is decreased when running with muscle damage (Assumpção, Lima, Oliveira, Greco, & Denadai, 2013). Alteration in muscle recruitment not only impacts running performance, but can increase the likelihood of injury in athletes (Cheung et al., 2003; Gerlach et al., 2005).

It is important that exercise professionals and coaches know that during times when athletes are experiencing EIMD and/or DOMS they can suffer performance detriments, and even

an increased risk of injury. Likewise, when looking at exercise and physical activity adherence for older adults or sedentary individuals, DOMS can sometimes be a reason individuals will avoid physical activity (Chao, Foy, & Farmer, 2000; Trost, France, & Thomas, 2011). For these reasons, various modalities to reduce DOMS, improve recovery, and attenuate performance loss have emerged throughout the years, including IPC.

1.2.6 Intermittent Pneumatic Compression. Originating from the clinical environment of venous disorders, IPC has emerged in recent years as a recovery tool for sport. While using compression as the main mechanism to improve recovery, IPC units are stark contrasts to compression clothing to which they are often compared to. IPC units are bulky and cumbersome, and are not meant to be moved while in operation which reduces versatility compared to the garments. However, IPC can generate high levels of pressure on the limbs beyond the ranges observed in compression garments (Born, Sperlich, & Holmberg, 2013; MacRae, Cotter, & Laing, 2011).

IPC effectiveness has been well documented in clinical populations with vascular conditions of the lower limb. The post-surgical blood clotting in immobilised patients, called deep vein thrombosis (DVT) has received the bulk of the research attention as it can become a severe medical emergency if the clot mobilizes and gets entrapped in a pulmonary artery (pulmonary embolism). Most research supports the use of IPC to help in DVT (Chen, Frangos, Kilaru, & Sumpio, 2001; Dennis, Sandercock, Reid, & Graham, 2013; Ho & Tan, 2013). Two mechanisms are thought to help this condition; increased blood flow to prevent the clots from starting, and greater enzyme activity to increase breakdown of existing clots (Chen et al., 2001; Fanelli et al., 2008). Support for the latter mechanism is still equivocal (Morris et al., 2006). Other conditions in which IPC has been beneficial includes lymphedema (Feldman, Stout, &

Wanchai, 2012; Muluk et al., 2013), and venous leg ulcers (Comerota & Arbor, 2011), both of which are believed to improve by mechanical force exerted, which mobilises pooling blood and lymph fluid from the limbs back to the torso.

Currently, only a few studies have looked at IPC in athletes or exercising populations. Cochrane et al. (2013) recruited ten healthy males for their study. Each participant performed three sets of 100 unilateral eccentric contractions of the quadriceps and was randomized to IPC or a control condition, administered immediately post, and at 24 and 48 hours following. NormaTec devices set at pressures of 70:80:80:80:60 mmHg (from the distal to proximal cells, respectively) for 30 minutes was used for the IPC group, and controls remained seated wearing inactive IPC devices on their legs. The investigators measured isometric torques at the knee in faster (180°/s) and slower (30°/s) conditions for both eccentric and concentric contractions. Average and peak values were recorded for each of the contractions. Additionally, CK concentrations and single-leg vertical jump of the exercised leg were measured. Group comparisons between IPC and control showed no benefit of IPC on the recovery of all outcome measures, however, there were only three of the measured variables (average and peak slow (30°/s) concentric torques, and peak isometric torques) that had significantly declined from pre-exercise values. The exercise intervention was not sufficient to induce noticeable change in most of the outcome variables. Likewise, no time differences were found in any of the jumping variables (average peak power, maximal peak power, average jump height and maximal jump height). This was a limitation of this study, as only three of the fourteen variables had experienced a significant detriment as a result of the intervention. Without the significant reductions there could not have been significant return to baseline. The study was likely

underpowered and could have used a greater number of participants, or employed a more strenuous muscle damaging intervention.

Another study had participants run 20 miles at 70% of $\dot{V}O_2$ max outdoors, and examined the effects of IPC on recovery (Draper, 2014). Using a randomized crossover design, participants used either IPC devices at pressure settings of 90:100:100:100:90 mmHg (from distal to proximal cells, respectively) for 60 minutes, or passive rest as a control following the run. Participants underwent the recovery interventions once per day for five days following the run and the investigators took blood samples for serum concentrations of C-reactive protein (CRP) as well as asked for self-reported DOMS on each day. The IPC condition saw a slight beneficial effect on DOMS ratings in men when genders were compared separately, and contrastingly, there was a significant increase in self-reported DOMS rating in the women group using IPC, while no difference was found in CRP levels. There were only 10 participants, 5 males and 5 females. The separate gender comparisons might explain why there were significant effects observed, as each participant represented a large proportion of the results.

Other studies have also looked at short-term recovery protocols, where participants were exercised immediately after the recovery intervention. Wiener et al. (2001) had eight healthy male participants perform a brisk ten minute treadmill walking protocol followed by two minutes of isometric contractions of the tibialis anterior muscle (against a weight suspended from the foot) to induce fatigue in that muscle. Immediately following the exercises, subjects wore an IPC device on one leg while the other served as a control. The IPC device was set at 80 mmHg of pressure, was used for three minutes, and was then followed by another two minute isometric workload. The authors examined the mean power frequency using an electromyography (EMG) device during the two isometric workloads. They found that when the leg (regardless of

dominance or order) was subject to IPC following fatiguing exercise, that the decay in isometric strength over the course of the second isometric bout was significantly less than the control leg.

Further, a randomized crossover design compared 20 minutes of IPC at 50 mmHg with supine rest as a control condition for 20 minutes. These recovery interventions were placed between two bouts of cycling at 80% of $\dot{V}O_2$ max to fatigue (Zelikovski, Kaye, Fink, Spitzer, & Shapiro, 1993). The participants performed similarly in terms of time to fatigue on the first bout, regardless of order and group assignment; however, time to fatigue was 45% longer on the second bout for the participants using IPC during the recovery time, compared to control (8.7±0.8 minutes for IPC vs. 6.4±0.7 minutes for control). No differences were seen in various biomarkers for energy metabolism (blood lactate, pyruvate, ammonia, and bicarbonate). Similarly, oxygen consumption and carbon dioxide production values did not differ significantly between groups despite being slightly higher in the IPC group.

Lastly, a pilot study by Waller, Caine and Morris (2006) used two different pressure settings on the IPC device as well as a passive rest group. The pressures reported for the high pressure IPC were 70:65:60 mmHg, while the low pressure group was 20:15:10 mmHg, with the values representing the most distal cells to the proximal, in respective order. Participants of the study underwent three 1 hour shuttle runs at least three days apart, each one followed by a one hour recovery intervention which was administered in a randomized crossover manner. Following this, they completed a maximal height vertical jump 1 hour after treatment and reported pain levels to assess DOMS at 1, 24, and 48 hours after the recovery treatment. Both IPC pressures had significantly reduced DOMS ratings compared to control at all the time points, but only the high pressure group had a significantly attenuated loss in vertical jump height from baseline measures.

Currently, there does not appear to be any research that has looked into the physiological mechanisms at play with the use of IPC in the recovery from sport. However, in terms of clinical population studies, there have been a few investigations attempting to uncover what occurs physiologically when wearing the IPC devices.

Physiological mechanism of IPC. Increased hemodynamics, or the movement of blood, is strongly supported by multiple studies that have examined the effects of IPC. Ample evidence shows that venous blood flow is increased in response to the external pressure exerted by IPC devices (Abu-Own et al., 1993; Comerota & Arbor, 2011; Fanelli et al., 2008; Labropoulos, Wierks, & Suffoletto, 2002; Malone et al., 1999; Zaleska et al., 2013). Arterial blood flow has also been shown to increase when using IPC (Morris & Woodcock, 2002). While it may seem counterintuitive with the mechanical pressure exerted against the regular flow of the arteries, the release of pressure in between cycles allows for arterial blood to enter the limb with much less resistance from venous blood (Morris & Woodcock, 2002).

Some studies have examined the dose-response of various pressures on venous blood flow. One study compared two IPC devices by different companies: one exerted 80 mmHg with a 5.5 second inflation time while the other only produced 45 mmHg and had an inflation time of 12 seconds (Labropoulos, Cunningham, Kang, Mansour, & Baker, 2010). They found that the higher pressure produced a greater increase in blood flow as well as quicker acceleration of blood. It is worth noting that the study was comparing the effectiveness of the devices, and not specifically the pressure differences, since the other variable of inflation time could also influence how blood flows.

As the blood flows upward in the limb from the IPC, there is an outward pressure that gets exerted from the inside of the blood vessels (A. H. Chen et al., 2001). The outward shear force stimulates the release of NO, a potent vasodilator, from the endothelial cells of the blood vessels (Chen et al., 2001; Chen et al., 2002; Liu, Chen, Seaber, Johnson, & Urbaniak, 1999; Rifkind et al., 2014). This physiological response is thought to accommodate an increase in blood volume going through the vessels (Palmer, Ferrige, & Moncada, 1987) and is regulated locally, rather than by the central nervous system (Tschakovsky et al., 2004). Tschakovksy et al. (2004) observed that a the vasodilatory response was much quicker than the expected release of NO, which led to the conclusion that the peripheral nervous system likely played a role in vasodilation as well. The study by Tschakovsky et al. (2004) also evaluated change in blood flow in response to different intensities of muscle contractions with the forearm held above the head and found that blood flow and subsequent muscle oxygenation increased as the intensity of contractions increased. These results could be extrapolated to the intensity of compression from IPC, where mechanical stress is applied to the vessels in a similar manner (A. H. Chen et al., 2001). Both the shear stress and vasodilation responses might promote an increased perfusion of blood into the nearby tissues (Bochmann et al., 2005) and subsequently, increased tissue oxygenation (Kolari, Pekanmäki, & Pohjola, 1988).

Revisiting the initial inflammatory response to injury from exercise might offer potential mechanisms by which IPC helps muscle recovery in athletes. It has been shown that leukocytes respond to the injured muscle (Kharraz et al., 2013; L. L. Smith et al., 2000). Once at the site, the leukocytes begin to leave the circulatory system, and might benefit from the dilated vessels, as well as the outward shear stress which might promote extravasation of these immune cells (Alon & Ley, 2008).

Evidence has shown that increased blood flow upregulates the effectiveness of L-selectin molecules, which are CAM, as well as promotes adherence of leukocytes to the endothelium to allow them to begin their rolling process (Yago et al., 2004; Zhu, Yago, Lou, Zarnitsyna, & Rodger, 2009). This might seem paradoxical, as increased flow would be expected to reduce adhesion, but it appears that many cells, including platelets and leukocytes, require blood flow to exceed a certain threshold to extravasate (Finger et al., 1996; Lawrence, Kansas, Kunkel, & Ley, 1997; Zhu et al., 2009). This leads to continued extravasation by leukocytes despite increased blood flow (Cinamon, Shinder, & Alon, 2001), and maybe even lead to an increased amount of leukocytes at the injured muscle than would occur under unaltered flow conditions (Zhu et al., 2009). While no research has yet looked at artificial increases in leukocyte concentrations within an injured tissue and its relation with muscle healing, the opposite has been demonstrated, where lower leukocyte concentrations in injured muscle tissue have led to impaired muscle healing (Summan et al., 2006; Wang et al., 2014). If IPC can promote a greater and quicker influx of leukocytes, this might lead to a quicker inflammatory response and quicker subsequent recovery from muscle damage.

Increases in NO concentrations during the repair sequence by the macrophages, or from the shear forces applied on the endothelial cells of blood vessels could also be another potential mechanism by which IPC could benefit recovery. As discussed in section 1.2.3, NO can stimulate satellite cell proliferation, which is part of the repair sequence of muscle tissue (Filippin et al., 2011). Given that IPC improves hemodynamics and stimulates the release of endothelial NO (A. H. Chen et al., 2001), there may be an influence by NO that could lead to satellite cell proliferation in damaged muscle tissue.

While the present study is not directly evaluating any of the mechanisms considered above, the inflammatory and muscle damage markers response to IPC might hint at various physiological processes that are influenced by IPC and could be used to guide future research that use IPC or other blood flow promoting recovery modalities.

1.3 Statement of Problem

Despite the multitude of positive anectodes supporting IPC from elite athletes in various sports (NormaTec, n.d.-a), as described in section 1.2.6, there is still little empirical evidence to support IPC use as a recovery tool. More research needs to be completed to get a clearer understanding as to whether IPC is effective in enhancing recovery in athletes, particularly in power-based sports.

The primary purpose of this study is to evaluate the effects of IPC on physical parameters of performance recovery after an off-season training day in male football athletes. Secondarily, this study will evaluate the effects of IPC on the changes in: 1) blood biomarkers of inflammation (IL-6, MCP-1, IL-10); 2) a blood biomarker of muscle damage (Mb); and 3) self-reported levels of skeletal muscle pain as assessed by a visual analogue scale following training in male football athletes.

1.4 Hypotheses

The primary hypothesis is that:

1. IPC condition will have greater performance of the Countermovement Jump (CMJ) test 24 hours after training when compared to the control condition.

Secondary hypotheses include:

1. IPC will significantly increase the concentrations of interleukin-6 (IL-6), interleukin-10 (IL-10), monocyte chemoattractant protein-1 (MCP-1), and myoglobin (Mb) 3 hours after training when compared to the control condition.

2. IPC will significantly reduce concentrations of IL-6, IL-10, MCP-1, and Mb 24 hours after training when compared to the control group.

3. IPC will significantly attenuate the decrease in performance on the ten metre sprint 24 hours after training when compared to control condition

4. IPC will significantly reduce self-reported symptoms of pain, as assessed by a visual analogue scale, following the recovery intervention, and 3 and 24 hours following the training sessions when compared to control.

CHAPTER 2 – METHODS

2.1 Participants

The participants recruited for the study were male collegiate football players between the ages of 18 and 35 by word of mouth. The study occurred in the off-season for a Canadian Interuniversity Sport (CIS) football team.

2.1.1 Sample Size Analysis. A sample size analysis was done using vertical jump data from Waller et al. (2006) to determine the adequate amount of participants for the study. The results from the study were derived from nine participants undergoing IPC or control promptly after a 1 hour shuttle run in a randomized crossover manner. The jumps were done before the shuttle run and immediately following the recovery intervention. Participants using high-pressure (60-70 mmHg) IPC lost 1.9 ± 1.4 cm from their jump height while those in the control condition lost 5.9 ± 3.4 cm after the recovery session (Waller et al., 2006). Calculation of the effect size was required before proceeding with the sample size calculation (D'Agostino, Sullivan, & Beiser, 2006). Effect size was first calculated using Equation 1.

$$\text{ES} = \frac{|(\mu_1 - \mu_2)|}{\sigma} \quad (1)$$

where ES = Effect size, μ = mean of groups compared, and σ = common standard deviation of both groups.

Calculations using the results from Waller et al. (2006) results in an effect size of $1.6\overline{6}$. With an α value of 0.05 and a power of 80%, minimum sample was then calculated using Equation 2 (D'Agostino et al., 2006).

$$n = 2(\frac{Z_{1-(\alpha/2)} + Z_{1-\beta}}{ES})^2 \quad (2)$$

where *n* = minimum amount of participants, ES = effect size derived from Equation 1, Z is the standard normal distribution value of 1 - (α/2) and 1 - β. With α = 0.05 and β = 0.2, $Z_{1-(\alpha/2)} = 1.96$, $Z_{1-\beta} = 0.84$, and *n* = 5.644.

For the present study, n = 5.644, or 6 subjects will be required. Using an estimated rate of attrition of 25% (Cornish & Johnson, 2014), a recruitment goal of 8 subjects was used for the study.

2.1.2 Inclusion and Exclusion Criteria. Participants in the study were required to play on a CIS level football team and be eligible to participate in the following season. Participants also had to have one year experience in resistance training to ensure familiarity with the training demands of the team program. Exclusion criteria for participants consisted of: any current or previous musculoskeletal injury that would be have impaired the performance of the athlete during the testing or training, any autoimmune disorders, chronic or required use of anti-inflammatory drugs (e.g. NSAIDS), vascular conditions of the lower limb, use of nutritional supplements that have an anti-inflammatory effects (e.g. omega-3 fatty acids or curcumin), and any known cardiovascular disease.

2.2 Procedures

Ethical approval was sought and granted from the University of Manitoba Education/Nursing Research Ethics Board (ENREB) to conduct the experiment on human

subjects (see Appendix E). Consent to recruit players was then granted from the head coach of the team (see Appendix B).

2.2.1 Study Design. The study employed a randomized crossover design where participants were randomly assigned to either the control or IPC condition following training. The opposite recovery intervention was used the following week. Figure 2.1 demonstrates the timeline of one study arm, and figure 2.2 depicts the participant flow throughout the study.

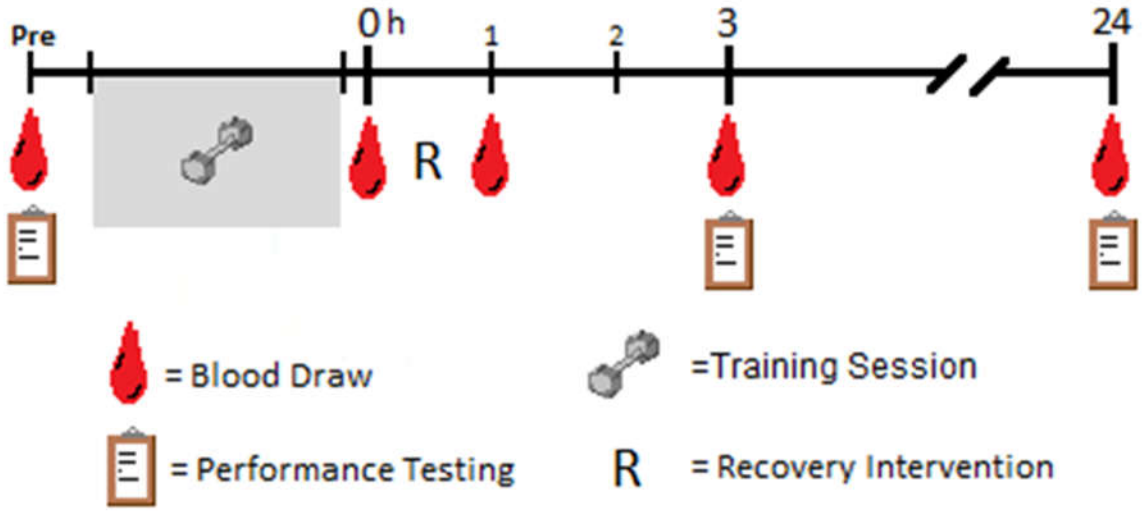


Figure 2.1 Timeline of One Study Arm: Hourly time points are relative to end of the training sessions.

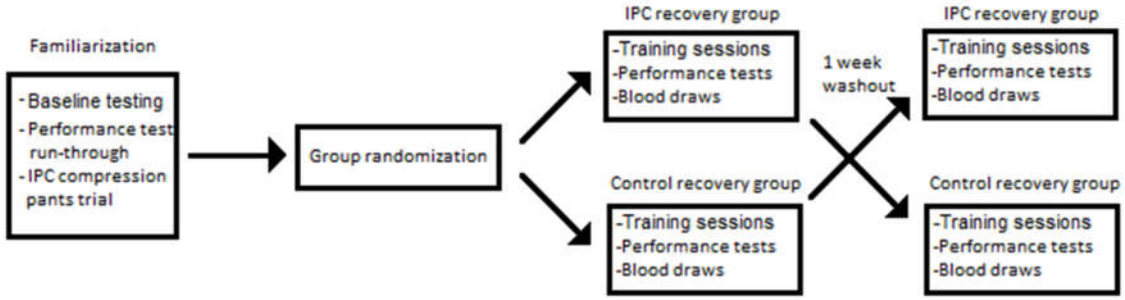


Figure 2.2 Timeline of Participant Intervention: Arrows indicate participant flow through the study

2.2.2 Training Sessions. Participants in the study kept the regular training that was asked of them as athletes. On the training days that the recovery interventions were administered, participants were required to undergo both a speed session as well as a strength session consecutively (back-to-back training day). The off-season training for the football team was planned in four week blocks, allowing for similar training sessions one week apart, both on the same days of the week.

During the off-season, athletes on the team were divided into three training groups. One training group consisted of novice players with no previous CIS experience, and thus was not targeted for recruitment. The remaining two groups had one day per week of back-to-back training sessions (speed and strength training). One group had their back-to-back training scheduled on Wednesdays, while the other was on Thursdays. Weight training metrics were tracked for two days before the intervention day, and included sets, repetitions, and intensity (as a percentage of maximum).

2.2.3 Recovery protocols. After the back-to-back training sessions, the participants underwent the intervention to which they had been randomized to. Resting heart rate (RHR) and resting blood pressure (RBP) were taken before the recovery sessions. Participants in the IPC group received 30 minutes of compression at level 6 pressure settings (70:80:80:80:60 mmHg from the distal to proximal cell, respectively) using IPC devices (NormaTec MVP, NormaTec, Newton Centre, USA). These pressure settings are similar to what other investigators had been using for sport recovery research (Cochrane et al., 2013; Waller et al., 2006) and such pressures have been shown to improve blood flow in healthy populations (Lurie, Awaya, Kistner, & Eklof, 2003; Morris et al., 2006; Morris & Woodcock, 2002). For the duration of the treatment, the subjects were seated in a chair and had their feet elevated to hip level, as per manufacturer

instruction. To standardize the back placement, the same make and model of chairs (Aqua Tilter, Krug, Kitchener, Canada) were used for all the interventions. Feet were kept elevated at the same height as the hips by resting the feet on another chair set at the same height (see figure 2.3). A familiarization session was done prior to the interventions to allow the participants the chance to try the IPC devices and ensure no discomfort was experienced while wearing the devices.

Figure 2.3 Intermittent Pneumatic Compression Recovery: Participant seated with legs elevated by another chair, with compression devices worn.

Contrastingly, participants undergoing the control intervention were seated in the same position as the IPC group, but without wearing the IPC devices.

2.2.4 Performance tests. Athletes were familiarized with the performance tests prior to the interventions to remove learning effect. All testing was performed in the same room and participants were asked to wear the same footwear for all trials. Performance testing occurred at baseline, and at 3 and 24 hours following training. Ten metre (10m) sprints always occurred first and CMJ trials second.

Countermovement jump. The CMJ is one of many jump testing protocols applicable to football players. Higher values on the CMJ test are associated with higher divisional ranking in

collegiate football from the United States (Fry & Kraemer, 1991). It is not clear if these athletes were selected on the higher level teams for their jumping ability, or if their specialised training at more elite levels enabled them to attain higher jump values. Jumping height was measured using a force plate (Quattro Jump, Kistler Instrument Corporation, Amherst, USA). The force plate has piezoelectric sensors that detect when forces are applied to them, and can calculate jump height using the following formula: jump height = $(g * t^2)/8$, where g represents the constant of gravity (9.81 m/s^2) and t is the flight time of the jump (Glatthorn et al., 2011). The force plate is also capable of measuring peak (PkPw) and average power (AvgPw), instantaneous force (FI), and velocity. To execute the jump, participants were instructed to jump as high as possible with their hands on their hips (i.e. to eliminate the influence of the arm swing on the jump [Lees, Vanrenterghem, & De Clercq, 2004]). Each participant was given three attempts at the jump, and the best and average values of all three jumps were used in the analysis. Each attempt was given a five second window to be completed. Sampling rate of the force platform was set at 500 hertz.

Ten metre sprint. Similarly to the CMJ, speed and acceleration are essential components of football (Dupler, Amonette, Coleman, Hoffman, & Wenzel, 2010; Robert, 2012). McBride et al. (2009) found that the split time at 10 yards and total time of a 40 yard sprint were moderately and inversely correlated with one-repetition maximum of the squat in American football athletes (r = -0.5437 and r = -0.6048, respectively). The time taken to complete the 10m sprint was recorded using a photo-electric timing system (SmartSpeed Pro, Fusion Sports, Chicago, USA). Time recording began when the participant crossed the first pair of gates and ended when they crossed the second pair of gates, placed ten metres away. Although the arm could have triggered the gates between 0.03-0.05 seconds quicker than the torso (Haugen, Tønnessen, & Seiler, 2012), the SmartSpeed system has false signal processing to correct and has been independently

validated to eliminate early crossing from arm swing (Earp & Newton, 2012), and timing was initiated and terminated once the torso crossed the respective gate. Participants were given three trials for the sprints at each testing time point. Before the sprint trials, they were told to undergo a self-selected warm-up until they felt ready to perform maximally. To start, participants lined up behind the starting line, taking a split (one foot in front of the other) or three-point stance (one hand on the line, with feet farther behind). Once their choice of starting stance was selected, participants performed all 10m sprint trials with the same stance (Cronin, Green, Levin, Brughelli, & Frost, 2004; Haugen & Buchheit, 2015). The lead foot or hand (for split stance and three-point stance, respectively) was also kept the same for all trials once the participants had selected their desired leading limb.

Adequate room for deceleration was given following the end of the ten metre distance and results were noted to the nearest 0.001 second. Timing gates consisted of one timing unit and a reflector, which were placed on tripods 2.8 metres apart at each timing distance, and set at 0.8 metres in height. A starting line was marked one metre before the first timing gates (see figure 2.4). The sprints occurred on a heavy-duty rubberized flooring surface, which was designed for an all-purpose training room (Sport Impact, Mondo, Laval, Canada).

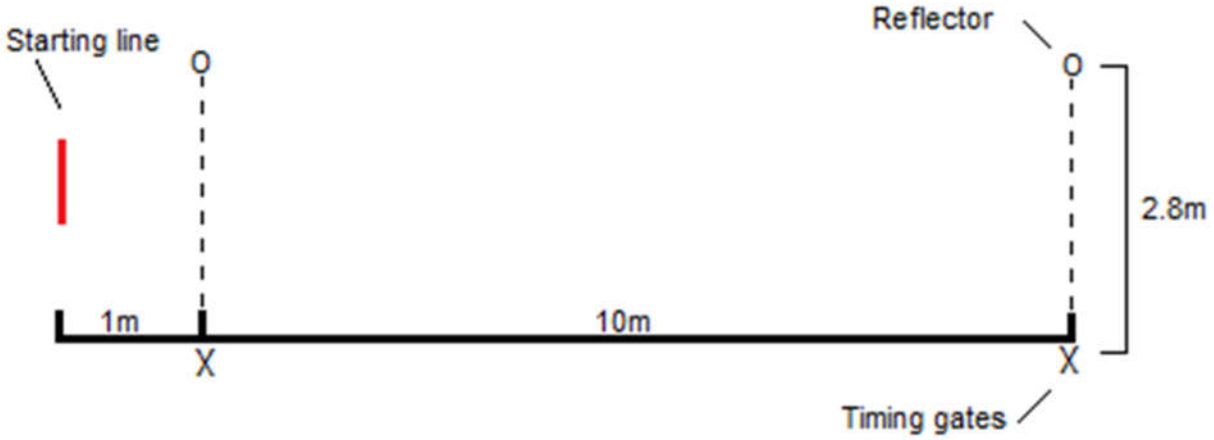


Figure 2.4 Overhead Representation of Ten Metre Sprint Test: Participants began the sprints from an immobile stance at the starting line (one metre behind the first gates). Time taken to cross both timing gates was collected as their ten metre sprint result. Figure not to scale.

2.2.5 Blood sampling and analysis Blood samples were taken before the training sessions, immediately following the exercise training bout and immediately following the recovery intervention (either IPC or control). Likewise, blood samples were taken at 3 hours and 24 hours post-training. Using a venous blood collection needle, a certified phlebotomist drew approximately 20 ml of blood from the antecubital vein into three vacutainers coated with ethylenediaminetetraacetic acid (EDTA) and one serum separating vacutainer tube. Once collected, the samples sat for 30 minutes to allow the serum separator tube to clot, and then were centrifuged at 3000 rotations per minute for 15 minutes at 4°C. Once the centrifugation cycle was complete, the separated plasma from the EDTA tubes and serum from the serum separating tube was aliquoted using a transfer pipette, and placed in a microtube. Each microtube was then stored at -80°C until analysis. Plasma (EDTA) samples were used for the cytokine analyses, while the serum samples were used to evaluate Mb concentrations.

Mb samples were analyzed using enzyme-linked immunosorbent assay (ELISA) kits (Abnova, Walnut, USA). The wells in the ELISA microplate were coated with Mb specific antibodies. When the serum samples were added, the Mb bound to the antibodies that lined the wells. A solution containing enzyme-linked antibodies was then added to the wells, where these antibodies bound to the Mb, creating a "sandwich" of antibodies, with the Mb protein in the middle. After an incubation period of 45 minutes, the wells were then rinsed to remove all unbound protein and residual solution. Tetramethyl-Benzadine was then added to the solution, reacting with the enzyme-linked antibodies. Once incubated for 20 minutes, a stop solution was introduced to the wells, which changed the colour of the solution in the wells to yellow, with the colour intensity in proportion with the concentration of Mb in the well. Absorptiometry was done using a microtiter plate reader (Epoch, Biotek, Winooski, USA) reading at a wavelength of 450 nm and resulted in the absorbance value for each well. Wells on the plate containing standard samples with known concentrations of Mb were used to create a regression equation. This equation was then used to find the concentration of the protein by inputting the value of absorbance obtained for each well.

Luminex bead-based multiplex analysis kits (EMD Millipore, Billerica, USA) were used to evaluate inflammatory marker concentrations in the plasma samples. Similarly to ELISA kits, Luminex multiplex technology utilizes antibodies to bind to the proteins of interest. Contrastingly, however, microscopic colour-coded beads that are coated with the antibodies are added to the wells, rather than the wells of the plate being coated with one specific antibody, which allowed for multiple proteins to be evaluated at once. These protein-specific beads were introduced in solution to the wells where they reacted with the plasma samples during an overnight incubation phase of the preparation. Detection antibodies were then added followed by

a treatment of phycoerythrin-streptavidin which binds with the antibodies and is fluorescent for the analysis. The plates were then placed in a plate reader, which detected the beads using laser optics (Luminex MagPix, Luminex Corp., Toronto, Canada) that both determined colour hue of the bead (specific to the analyte) and the fluorescence (the concentration of the analyte).

2.2.6 DOMS. Muscle soreness experienced by the athletes was evaluated before the blood draws were taken. Subjective muscle soreness levels were reported by the participants on a visual analogue scale (VAS). Participants evaluated their level of soreness by having them mark along a standardized 100 mm line, with 0 and 100 marked at either extremity. They were told that 0 would indicate "no soreness" while 100 was "extreme soreness" (see Appendix C). They were also asked to draw their mark as perpendicular as possible to the VAS line to avoid ambiguity in the measurement. As the soreness sensation can change when attempting to move or ambulate, the VAS was administered when the athlete was sitting before the blood draw to maintain a standardized procedure. Using VAS scales in pain and soreness assessment has been validated in several populations (Breivik, Björnsson, & Skovlund, 2000; Campbell & Lewis, 1990; Jensen & Karoly, 1992). It has also been shown to be better to track DOMS over time than pressure pain threshold (Lau, Muthalib, & Nosaka, 2013) and is more sensitive to change than the Borg or Likert scales as it allows for measurements to fall between the intervals (Grant et al., 1999). For data recording, distance from the "0" to the participant mark was measured twice using different office rulers. To account for the fact that most lines were not perfectly vertical, the recorded measurement was noted at the point immediately inferior to the bold horizontal line of the VAS.

2.2.7 Dietary tracking. Participants in the study were asked to keep a dietary log for the day before the intervention until the 24 hour post-resistance training time point (see Appendix

D). In attempt to keep diet standardized, participants were asked to eat the same foods and supplements after the crossover, when they were subject to the second intervention. It is well known that diet can have an influence on inflammatory variables and recovery (Cockburn, Hayes, French, Stevenson, & St Clair Gibson, 2008; Rowlands et al., 2008; Tipton et al., 2007; Witard et al., 2014). Maintaining a similar diet should lead to a similar impact on the inflammatory variables and performance recovery.

2.3 Statistical Analysis

A two factor (intervention by time) repeated-measures analysis of variance (ANOVA) was used to determine if significant differences between groups were found. Significance was set at a p value of ≤ 0.05. When there were significant results from the repeated-measures ANOVA, a Bonferroni post-hoc analysis was used to determine which means differed from one another, Additionally, Cohen's effect size (d) statistical test was used to determine the magnitude of difference between recovery interventions on all outcome variables (Cohen, 1988). This test was used to examine the breadth of the difference between the means, as opposed to hypothesis testing, which only answers if they are different or not (Palisano, 2011). The stratification of the descriptive values used for effect sizes based on the d are found in table 2.1. An analysis of covariance (ANCOVA) was used on outcome variables with strength training volume as a covariate, as there were training group differences between the participants that had recovery on the Wednesday compared to those that went on Thursday.

Descriptive statistics, data presentation, and Cohen's d test were done using Microsoft Excel 2010, software version 14.0.7166.5 (Microsoft Corporation, Redmond, USA). All

remaining statistical analysis (e.g. ANOVA, ANCOVA) was completed using Statistica, software version 13.2.92.0 (Statsoft Inc., Tulsa, USA).

Table 2.1 Descriptive Label for Corresponding *d* Statistic

Cohen's Effect Size *d* value	Descriptive result
0 - <0.2	Trivial
0.2 - <0.5	Small
0.5 - <0.8	Moderate
≥0.8	Large

Note: The result of the difference between means divided by the common standard deviation is the *d* value for a given comparison. Values of this calculation can be negative; however, absolute values are used when referencing the table.

2.3.1 Missing Data. A post-hoc decision to compute missing data was taken, due to the nature of the repeated-measures ANOVA requiring no missing data to be completed. Using k-Nearest Neighbour (k-NN) test with a $k = 3$ and using the Euclidian distance function, missing data was computed to allow all participant results to be analyzed and ensure that the sample remained above the threshold for adequate statistical power.

RESULTS

3.1 Participant Characteristics

A total of eight (n = 8) participants were recruited and completed the study. Two (n = 2) additional participants had gone through portions of the baseline testing, but were unable to participate in the remainder of the study for scheduling reasons. Descriptive characteristics of the participants that completed the interventions are listed in Table 3.1 below.

Table 3.1 Participant Descriptive Characteristics

Characteristic	Mean ± SD	Range
Age (years)	21.1 ± 2.1	19-24
Playing Experience (seasons)	1.8 ± 1.2	1-4
Height (cm)	183.2 ± 6.3	173.6-189.7
Weight (kg)	96.2 ± 15.8	74.2-125.6
BMI (kg/m^2)	28.6 ± 3.6	24.6-33.3
RHR (bpm)	65.5 ± 10.2	52-80
Systolic Brachial BP (mmHg)	128.5 ± 10.9	108-140
Systolic Ankle BP (mmHg)	155.3 ± 18.2	132-184
Ankle Brachial Index	1.21 ± 0.11	1.06-1.35

Note: BMI = Body mass index, RHR = Resting heart rate, BP = Blood Pressure, Ankle Brachial Index = Ankle BP/Brachial BP. Playing Experience denotes the seasons played at the CIS level.

A broad range of positions played were represented in the sample: two defensive backs, two linebackers, one receiver, one offensive lineman, one running back, and one wide receiver.

3.2 Missing Data

Three (n = 3) participants were unable to attend some of the testing times, which led to missing data (performance, blood samples, and DOMS) at certain time points. One control pre-training (n = 1), one IPC 3 hour post-training (n = 1), and two 24 hour post-training (n = 2) measurements were missing. Both missing measurements 24 hours post-training were from the same participant, and thus represented a missing data point from both recovery interventions.

The k-NN algorithm was used to calculate all missing data to maintain the sixteen total cases (two per participant) that could be used for the repeated-measures ANOVA, rather than reducing it to 12 cases (which would have resulted in an n = 6 per recovery intervention).

Listed in table 3.2 are the mean and standard deviations of the measured variable before and after alteration using k-NN, as well as the change in the mean and standard deviation resulting from the calculation.

Table 3.2 Change in Outcome Variables from k-Nearest Neighbour Algorithm

Variable	Mean ± SD before k-NN	Mean ± SD after k-NN	Change in mean (%)	Change in SD (%)
CMJ (cm) pre-training	50.81 ± 11.23	51.59 ± 11.29	0.78 (1.5)	0.059 (0.5)
CMJ (cm) 3 hours post-training	50.39 ± 8.43	50.42 ± 8.14	0.03 (0.1)	-0.285 (-3.4)
CMJ (cm) 24 hours post-training	51.30 ± 6.33	51.26 ± 5.89	-0.04 (-0.1)	-0.436 (-6.9)
10m sprint (s) pre-training	1.775 ± 0.094	1.772 ± 0.091	-0.003 (-0.2)	-0.003 (-2.7)
10m sprint (s) 3 hours post-training	1.774 ± 0.091	1.774 ± 0.088	<0.001 (<0.1)	-0.003 (-3.4)
10m sprint (s) 24 hours post-training	1.763 ± 0.088	1.766 ± 0.083	0.003 (0.2)	-0.005 (-6.0)
IL-6 (pg/ml) pre-training	6.11 ± 7.98	5.90 ± 7.76	-0.215 (-3.5)	-0.223 (-2.8)
IL-6 (pg/ml) 3 hours post-training	7.88 ± 10.64	7.65 ± 10.32	-0.223 (-2.8)	-0.323 (-3.0)
IL-6 (pg/ml) 24 hours post-training	6.68 ± 8.73	6.16 ± 8.25	-0.523 (-7.8)	-0.476 (-5.5)
IL-10 (pg/ml) pre-training	22.64 ± 24.60	21.83 ± 23.99	-0.814 (-3.6)	-0.613 (-2.5)
IL-10 (pg/ml) 3 hours post-training	24.35 ± 31.06	23.47 ± 30.21	-0.876 (-3.6)	-0.849 (-2.7)
IL-10 (pg/ml) 24 hours post-training	23.69 ± 27.61	21.93 ± 26.15	-1.758 (-7.4)	-1.456 (-5.3)

Variable	Mean ± SD before k-NN	Mean ± SD after k-NN	Change in mean (%)	Change in SD (%)
MCP-1 (pg/ml) pre-training	230.19 ± 47.24	230.54 ± 45.54	0.351 (0.2)	-1.696 (-3.4)
MCP-1 (pg/ml) 3 hours post-training	290.48 ± 72.10	292.47 ± 70.01	1.988 (0.7)	-2.085 (-2.8)
MCP-1 (pg/ml) 24 hours post-training	252.08 ± 48.88	253.79 ± 44.18	1.708 (0.7)	-4.703 (-6.2)
Mb (ng/ml) pre-training	12.58 ± 9.39	12.34 ± 9.12	-0.24 (-1.9)	-0.269 (-2.9)
Mb (ng/ml) 3 hours post-training	80.22 ± 41.59	82.42 ± 41.12	2.20 (2.7)	-0.468 (-1.1)
Mb (ng/ml) 24 hours post-training	26.61 ± 33.50	26.11 ± 31.53	-0.50 (-1.9)	-1.972 (-5.9)
DOMS (mm) pre-training	25.2 ± 18.3	24.9 ± 17.7	-0.3 (-1.2)	-0.579 (-3.2)
DOMS (mm) 3 hours post-training	27.3 ± 14.8	27.1 ± 14.3	-0.2 (-0.8)	-0.478 (-3.2)
DOMS (mm) 24 hours post-training	28.9 ± 14.7	29.1 ± 14.0	0.2 (0.8)	-0.697 (-4.7)

Note: k-NN = k-Nearest Neighbour; SD = standard deviation, CMJ = Countermovement Jump, 10m = ten metres, IL = Interleukin, MCP-1 = monocyte chemoattractant protein-1, Mb = Myoglobin, DOMS = delayed-onset muscle soreness.

3.3 Dietary Analysis

A total of three (n = 3) participants returned both food logs while two (n = 2) participants returned one week of food logs. Food logs were analyzed for approximate macronutrient content between intervention arms. Table 3.3 shows the differences in macronutrient intake between recovery interventions. A student t-test was done to compare total kilocalories consumed and kilocalories derived from carbohydrate, fat, and protein by recovery intervention.

Table 3.3 Nutritional Intake by Intervention

Day	kcal	IPC (n = 4) Mean ± SD	Control (n = 4) Mean ± SD	*p* - value
Two days before intervention	Total	2994 ± 1132	2785 ± 1221	0.73
	Protein	728 ± 548	630 ± 528	0.59
	Fat	916 ± 487	987 ± 731	0.77
	Carbohydrate	1322 ± 376	1171 ± 160	0.57
One day before intervention	Total	3388 ± 1900	3498 ± 2256	0.89
	Protein	628 ± 442	847 ± 759	0.34
	Fat	1264 ± 937	1455 ± 1294	0.57
	Carbohydrate	1505 ± 657	1198 ± 298	0.50
Recovery intervention day	Total	1710 ± 1265	2808 ± 2289	0.42
	Protein	402 ± 438	598 ± 693	0.68
	Fat	495 ± 310	1039 ± 848	0.30
	Carbohydrate	793 ± 621	1169 ± 745	0.35
Daily Average	Total	2698 ± 1526	3030 ± 1828	0.51
	Protein	586 ± 456	692 ± 615	0.52
	Fat	892 ± 662	1160 ± 920	0.17
	Carbohydrate	1206 ± 601	1179 ± 427	0.89

Note: Data presented as Mean ± SD. kcal = kilocalorie.

3.4 Pre-Recovery Measures

RHR and RBP (systolic and diastolic values) were taken before the recovery interventions as potential covariates to the outcome variables. Student *t*-tests were used to compare these measures by recovery modalities and determined there were no differences for all measures ($p = 0.57$, $p = 0.49$, and $p = 0.62$ for RHR, systolic blood pressure, and diastolic blood pressure, respectively).

3.5 Participant Training

The days on which the recovery interventions were administered consisted of two training sessions: speed training followed by strength training. Half (n = 4) of the study

participants were in a group that did their back-to-back training sessions on the Wednesdays and the other half (n = 4) were from the group that completed those training sessions on Thursdays. The speed training sessions lasted approximately one hour and were focused on sprinting, cutting, acceleration, and deceleration drills. The approximate volume of these speed sessions was targeted to be between 250 and 300 metres of total sprinting. As the team was in the last two weeks of off-season training, sprints were maximal, but shorter (~30-40 m) with quicker deceleration phases, contrasting the longer but less intense sprints earlier in the off-season. Three to four cutting drills were also added to these sessions, and athletes were asked to perform five to ten repetitions of the drills. Speed training was implemented identically between groups. The speed sessions were then followed by the strength sessions.

Strength training sessions were more individualized than sprint training. Athletes training programs were first divided by playing position to meet positional requirements, and subsequently, each athlete was given individual exercises to address individual needs. Total strength training metrics were accounted for and compared for group and time differences. Using a student *t*-test, subgroup analyses found that the Thursday intervention group was significantly older and had more playing experience, but no difference on other characteristic measured at baseline (see table 3.4).

Table 3.4 Participant Characteristic by Intervention Day Subgroup

Characteristic	Wednesday Group (n = 4) Mean ± SD	Thursday group (n = 4) Mean ± SD	*p* – value
Age (years)	19.25± 0.5	23 ± 0.8	<0.01 *
Playing Experience (seasons)	1 ± 0	2.5 ± 1.3	<0.01 *
Height (cm)	183.2 ± 7.8	183.3 ± 4.2	0.96
Weight (kg)	93.2 ± 21.1	99.2 ± 5.8	0.45
BMI (kg/m^2)	27.5 ± 3.8	29.6 ± 3.0	0.23
RHR (bpm)	62 ± 9.8	69 ± 9.3	0.16
Systolic Brachial BP (mmHg)	128 ± 12.4	129 ± 9.3	0.85
Systolic Ankle BP (mmHg)	161 ± 18.3	150 ± 16.2	0.24
Ankle Brachial Index	1.25 ± 0.07	1.16 ± 0.12	0.10

Note: BMI = Body mass index, RHR = Resting heart rate, BP = Blood Pressure, Ankle Brachial Index = Ankle BP/Brachial BP. * indicates significant group differences ($p < 0.05$).

Total strength training differences between recovery interventions are presented in table 3.5, while subgroup comparisons of training by week are presented in table 3.6. Volume is represented by the quantity of repetitions completed in the training session and intensity is the average percentage of one repetition maximum (1RM) at which the repetitions were performed. A repeated-measures ANOVA was used to compare the recovery interventions and the volume and intensity of the strength sessions of the intervention days and the two strength sessions preceding (see table 3.5). No significant differences were found. Contrastingly, when the training metrics were analyzed by week (time effect) and training day (Wednesday or Thursday for recovery intervention) using a two-way repeated-measures ANOVA, significant differences were observed between training volumes on all days preceding the recovery session. An ANCOVA was then completed on outcome variables, with training volume as a covariate, however, no interaction effects were observed.

Table 3.5 Strength Training Metrics by Recovery Modality

Day	Variable	IPC (n = 8) Mean ± SD	Control (n = 8) Mean ± SD	*p* - value
Two days before intervention	Volume	82.4 ± 40.7	81.3 ± 36.0	0.69
	Intensity	69.2 ± 33.1	69.4 ± 30.0	0.75
One day before intervention	Volume	78.9 ± 9.9	77.9 ± 6.0	0.53
	Intensity	69.2 ± 7.4	69.2 ± 4.4	0.50
Recovery intervention day	Volume	99.5 ± 41.3	103.3 ± 46.3	0.12
	Intensity	70.9 ± 29.4	70.6 ± 31.6	0.13
Total	Volume	260.9 ± 18.0	262.5± 21.0	0.87
Average	Intensity	69.8 ± 5.0	69.8 ± 5.7	1.00

Note: Volume is represented by the quantity of repetitions performed in a given session while intensity is the average fractional value of the one-repetition maximum prescribed for each set, expressed as % of 1RM. No significant results were observed.

Table 3.6 Strength Training Metrics by Intervention Day and Week

Day	Metric	Wednesday Week 1 (n = 4) Mean ± SD	Thursday Week 1 (n = 4) Mean ± SD	Wednesday Week 2 (n = 4) Mean ± SD	Thursday Week 2 (n = 4) Mean ± SD
Two days before intervention	Volume	121.8 ± 4.5	45.3 ± 10.5 *†	112 ± 4.2	48.3 ± 10.5 *†
	Intensity	68.2 ± 2.8	71.1 ±17.4	69.0 ± 3.0	70.6 ± 16.4
One day before intervention	Volume	86.5 ± 7.0	70.3 ± 0.5 *†	84.0 ± 2.0	73 ± 0.5 *†
	Intensity	67.7 ± 5.8	70.7 ± 0.01	68.2 ± 2.1	70.5 ± 0.01
Recovery intervention day	Volume	63.5 ± 15	137 ± 0.5 *†	60.5 ± 15	145.3 ± 0.5 *†
	Intensity	71.0 ± 16.5	70.3 ± 6.2	70.9 ± 17.4	70.5 ± 5.8
Total	Volume	271.8 ± 26.6	266.8 ± 41.8	256.5 ± 22.5	251.8 ± 39.1
Average	Intensity	69.0 ± 6.2	70.7 ± 11.1	69.4 ± 5.9	70.5 ± 9.5

Note: Volume is represented by the quantity of repetitions performed in a given session while intensity is the average fractional value of the one-repetition maximum prescribed for each set, expressed as % of 1RM. * indicates $p < 0.05$ compared to Wednesday of same week. † indicates $p < 0.05$ compared to Wednesday of opposite week. No significant differences were observed with intensity at all time points and with total (sum of three days) volume.

3.6 Performance Results

3.6.1 Countermovement Jump. All resulting variables from the force platform were analyzed using a two-way repeated-measures ANOVA. The average of all three jumps at each time point was used in the calculations. While all participants had three trials of the CMJ at each

of the performance tests, there were four instances of an improper recording by either the force platform or the computer software. Three (n = 3) had only one of the three trials missed, allowing the average to be calculated. On the other hand, one (n = 1) trial had only one successful recording, meaning the best and average results for that time point were identical.

Jump Height. Maximal jump height for each jump was computed by the Quattro Jump software. Mean and standard deviation of the average jump height of all three trials are shown in figure 3.1. No statistically significant results were found between groups ($p = 0.62$), for time ($p = 0.88$) or group by time ($p = 0.18$). Effect size between groups were moderate at baseline (d = 0.560) and 3 hours post-training (d = 0.277), while trivial at 24 hours post-training (d = -0.103). Results of the absolute and relative changes from baseline are presented in table 3.7.

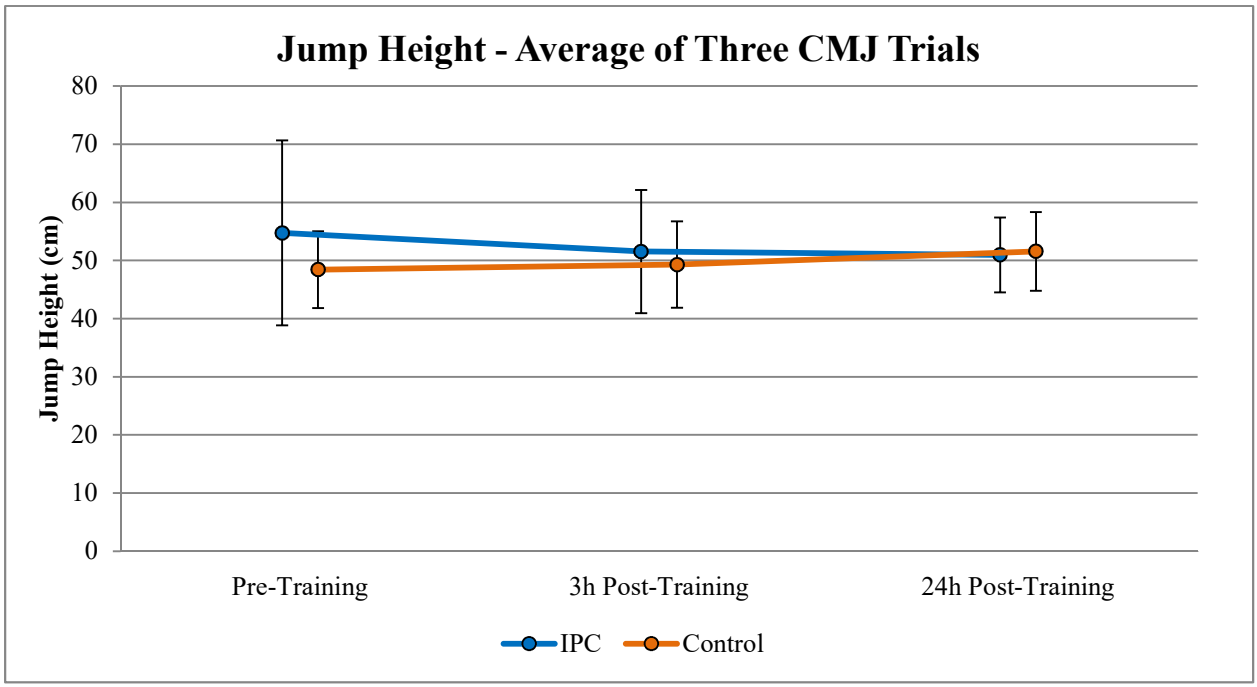


Figure 3.1 Jump Height Results: Jump height of the CMJ test before, 3 hours and 24 hours post-training, in centimetres. IPC recovery condition is represented by the blue line and control condition is represented by orange line. Data presented as mean ± SD. No significant differences were observed.

Table 3.7 Countermovement Jump Height Change from Baseline

Group	Pre-training	3 hours post-training			24 hours post-training		
	Jump height (cm)	Jump height (cm)	Absolute change (cm)	Relative change (%)	Jump height (cm)	Absolute change (cm)	Relative change (%)
IPC	54.75 ± 14.3	51.55 ± 9.3	-3.2	-5.9	50.96 ± 6.5	-3.8	-6.9
Control	48.44 ± 6.7	49.29 ± 7.3	0.8	1.8	51.56 ± 5.6	3.1	6.5

Note: Changes in jump height are expressed as a change from the pre-training measurement.

Velocity. The means and standard deviations of the peak jump velocity are presented in figure 3.2. No statistically significant results were found between groups ($p = 0.94$), for time ($p = 0.77$) or group by time ($p = 0.39$). Effect sizes were trivial at both baseline and at 3 hours post-training ($d = -0.001$ and $d = -0.084$, respectively), while there was a small effect between recovery groups at 24 hours post-training ($d = 0.289$).

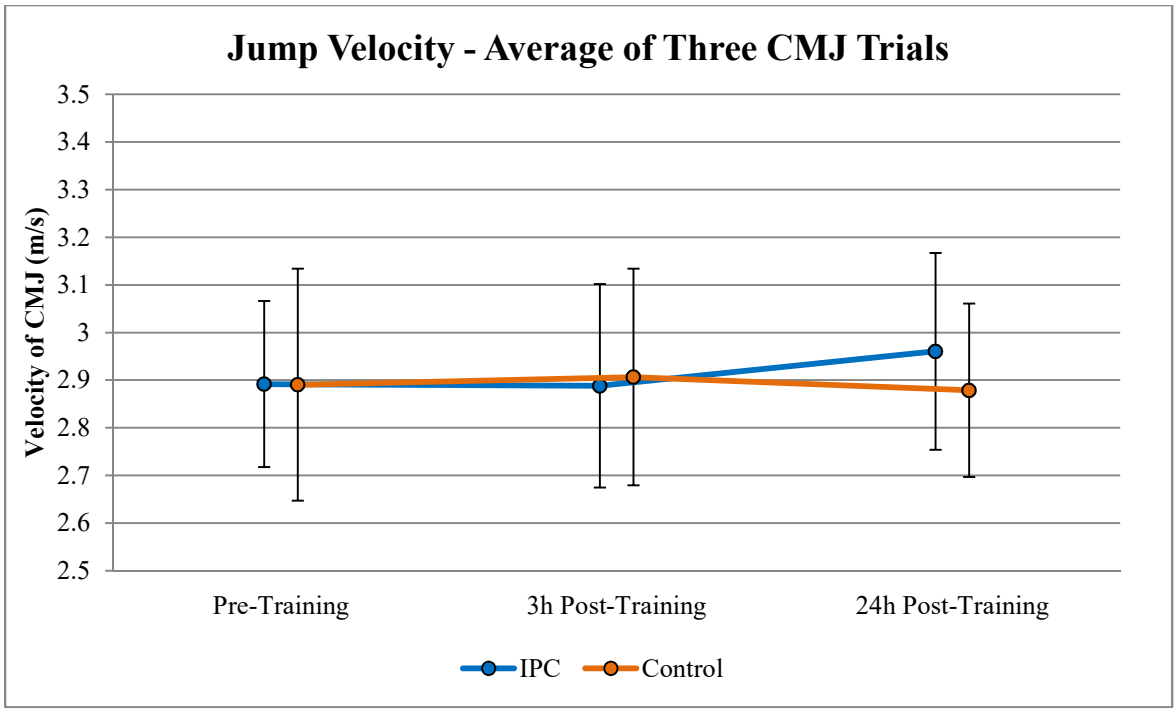


Figure 3.2 Jump Velocity Results: Average velocity of three jump trials at each time point. IPC recovery condition is represented by the blue line and control condition is represented by orange line. No significant differences were observed**.**

Other jump parameters. Other jump parameters were also not statistically significant, and IPC use was found to have a small or trivial effect at all time points. Results for FI, PkPw, AvgPw are presented in table 3.8.

Table 3.8 Statistical Analysis Results for Force Plate Outcome Variables

Variable		Repeated-measures ANOVA			Cohen's Effect Size *d*			
		Recovery	Time	Recovery by Time		Pre	3h post	24h post
Force Instantaneous	F	0.03	1.77	2.5	*d*	0.16	<0.01	0.21
	p	0.85	0.20	0.11	Desc.	Trivial	Trivial	Small
Peak power	F	<0.01	1.39	1.86	*d*	-0.08	-0.16	0.23
	p	0.96	0.27	0.18	Desc.	Trivial	Trivial	Small
Average Power	F	0.10	1.13	1.61	*d*	<0.01	-0.16	0.44
	p	0.76	0.34	0.22	Desc.	Trivial	Trivial	Small

Note: F = Fisher statistic of analysis of variance statistical test; *d* = Cohen's effect size; Desc. = Qualitative descriptor of Cohen's effect size.

3.6.2 Ten Metre Sprint. Results of the 10m sprint were analyzed using a two-way repeated-measures ANOVA. Means and standard deviations for the best of three attempts are shown in figure 3.3.

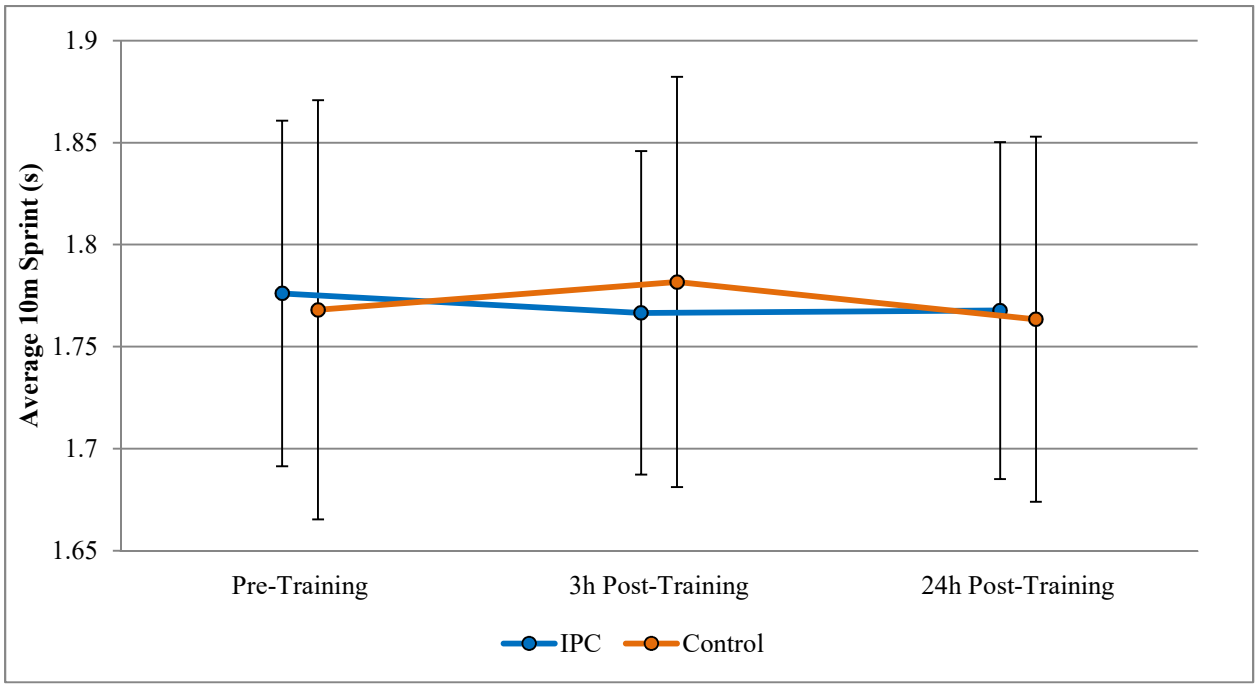


Figure 3.3 Ten Metre Sprint Testing Results: Average time (s) for three sprints completed. IPC recovery condition is represented by the blue line, control condition by the orange line. Data presented as mean ± SD. No significant differences were observed**.**

No statistically significant results were found between groups (p = 0.88), for time (p = 0.84) or group by time (p = 0.63). A trivial effect size was observed at 3 hours post-training for average sprint times (d = -0.172) and at 24 hours post-training (d = 0.051). Changes from baseline are presented in table 3.9.

Table 3.9 Ten Metre Sprint Change from Baseline

Group	Pre-training	3 hours post-training			24 hours post-training		
	Sprint time (s)	Sprint time (s)	Absolute change (s)	Relative change (%)	Sprint time (s)	Absolute change (s)	Relative change (%)
IPC	1.776 ± 0.08	1.767 ± 0.08	-0.009	-0.51	1.768 ± 0.08	-0.008	-0.45
Control	1.768 ± 0.10	1.782 ± 0.10	0.014	0.79	1.764± 0.09	-0.005	-0.28

Note: Changes in 10m sprint times are expressed as a change from the pre-training measurement. Decreased (quicker) sprint times are considered beneficial.

3.7 Biochemistry Results

Plasma and serum samples were thawed and analyzed for their respective systemic biomarkers. Compiled and intervention group means and SD of the biochemistry analyses are presented in table 3.10.

3.7.1 Inflammatory Markers. Inflammatory marker concentrations in plasma samples were analyzed using a multiplex kit. Repeated-measures ANOVAs and Cohen's effect size statistical tests were performed on each marker separately.

Interleukin-6. No statistically significant results were seen between groups ($p = 0.86$), for time ($p = 0.19$) or group by time ($p = 0.23$) with IL-6 concentrations. A small effect was noted between means ($d = -0.215$), while trivial effect sizes were observed at pre-training, post-training, post-recovery and 24 hours post-training ($d = -0.159$, $d = -0.077$ $d = -0.159$ $d = -0.133$, respectively). Results for IL-6 concentrations are presented in figure 3.4.

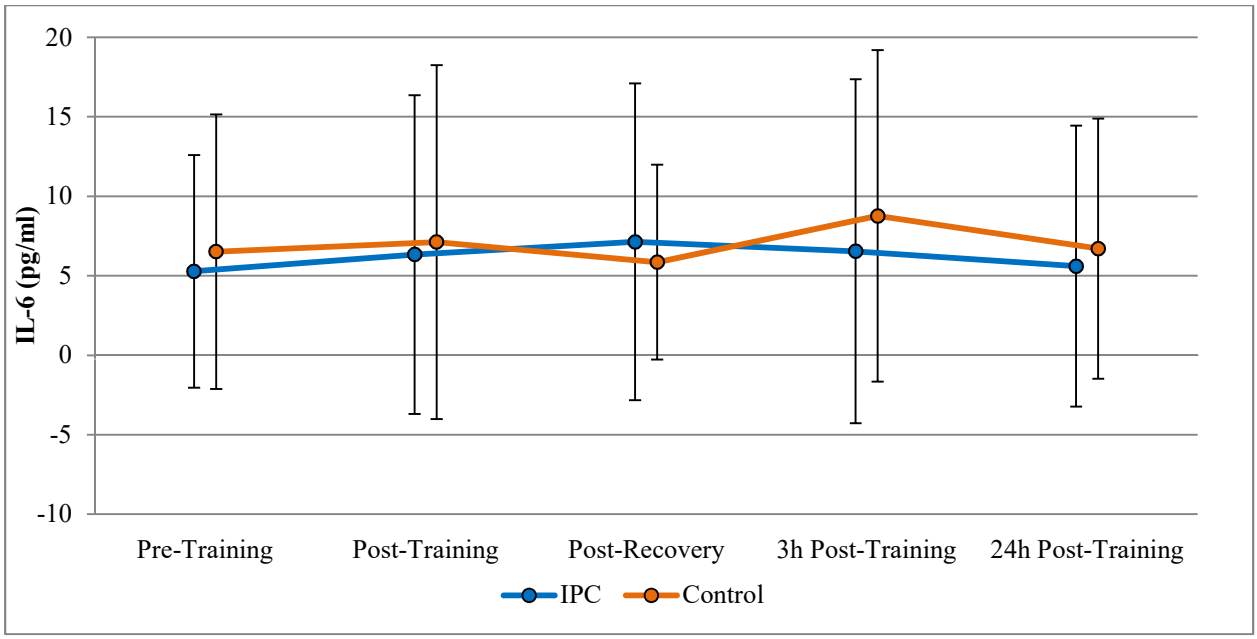


Figure 3.4 Interleukin-6 Plasma Concentrations: Mean ± SD of plasma IL-6 concentrations at all time points, by recovery group. IPC recovery condition is represented by the blue line, control condition by the orange line. Concentration values are in pg/ml. No significant differences were observed**.**

Interleukin-10. Similarly to IL-6, no statistically significant results were observed between recovery groups ($p = 0.92$), for time ($p = 0.82$) or group by time ($p = 0.42$) with IL-10 concentrations (see figure 3.5). Trivial effect sizes were observed at all time points ($d = -0.048$, $d = -0.066$, $d = 0.136$, $d = -0.148$, $d = -0.067$, respectively by time point).

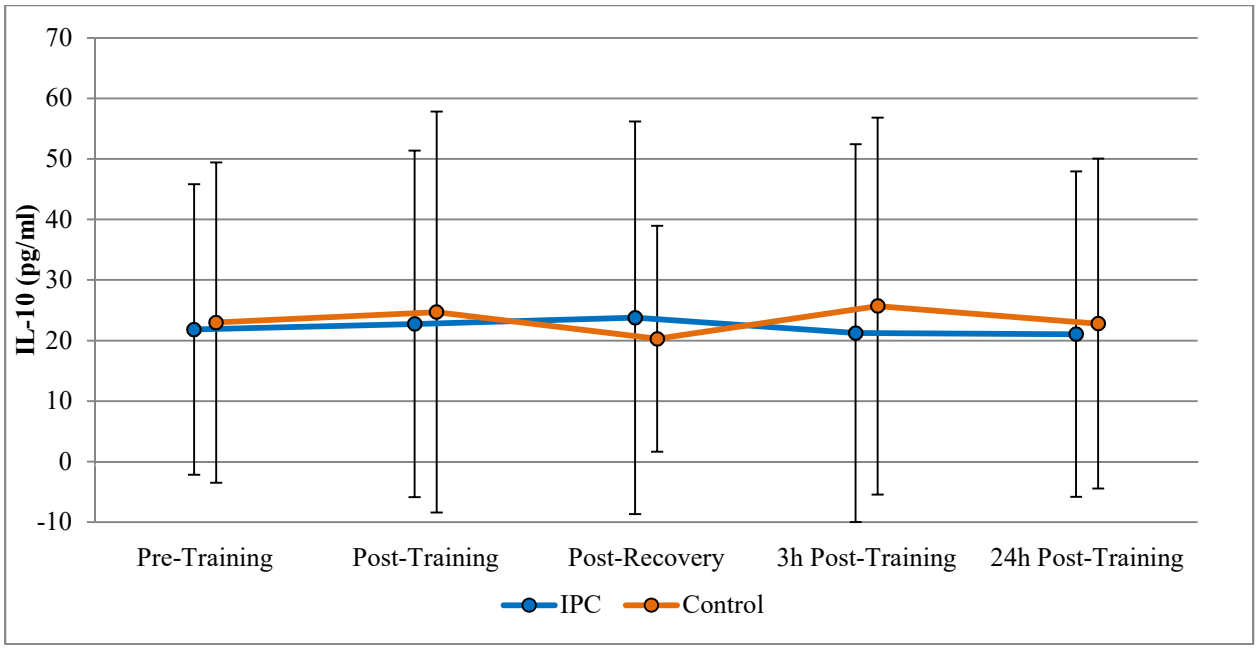


Figure 3.5 Interleukin-10 Plasma Concentrations: Mean ± SD of plasma IL-10 concentrations at all time points, by recovery group. IPC recovery condition is represented by the blue line, control condition by the orange line. Concentration values are in pg/ml. No significant differences were observed**.**

Interleukin 6/ Interleukin 10 ratio. Concentrations of IL-6 were divided by the IL-10 concentrations of the respective time points. No statistically significant results were found between groups ($p = 0.10$), for time ($p = 0.21$) or group by time ($p = 0.52$). Trivial effects were noted before training and at 3 hours post-training ($d = -0.058$ and $d = -0.010$, respectively), while a small effect was observed post-training, post-recovery and 24 hours post-training ($d = -0.299$, $d = 0.235$, and $d = -0.499$, respectively). Data are presented in figure 3.6.

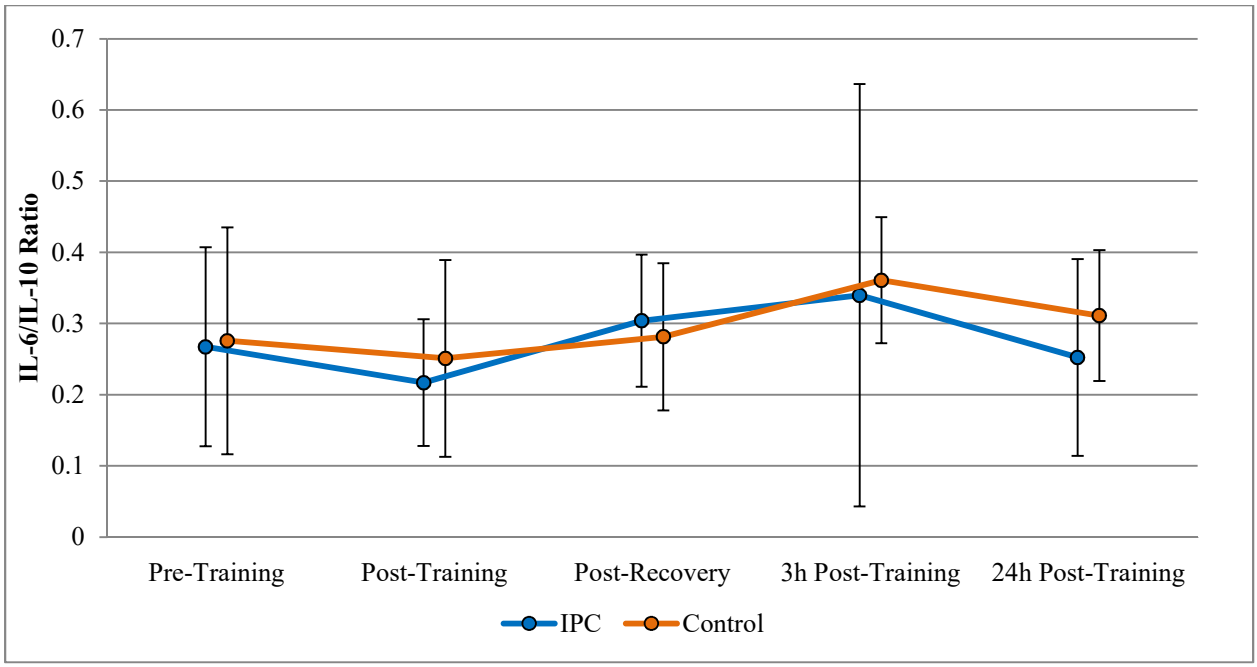


Figure 3.6 Interleukin-6/Interleukin-10 Ratio: Mean ± SD of the IL-6/IL-10 ratio at all time points, by recovery group. IPC recovery condition is represented by the blue line, control condition by the orange line. No significant differences were observed**.**

Monocyte chemoattractant protein-1. While no group or interaction effects were present ($p = 0.81$ and $p = 0.96$, respectively), a main effect for time was observed for MCP-1 concentrations ($p < 0.01$). A Bonferroni post-hoc analysis was applied to the results, and indicated that post-training, post-recovery and 3 hours post-training MCP-1 concentrations were greater than those before training. Furthermore, post-recovery concentrations were greater than those seen at 24 hours post-training. Data subdivided by recovery intervention are presented in figure 3.7 while averaged results demonstrating the effect for time are presented in figure 3.8. Effect sizes between recovery groups were trivial post-training and post-recovery ($d = 0.110$ and $d = 0.012$, respectively), while small effect sizes were noted at baseline, and at 3 and 24 hours post-training ($d = 0.462$, $d = -0.323$, and $d = 0.464$, respectively).

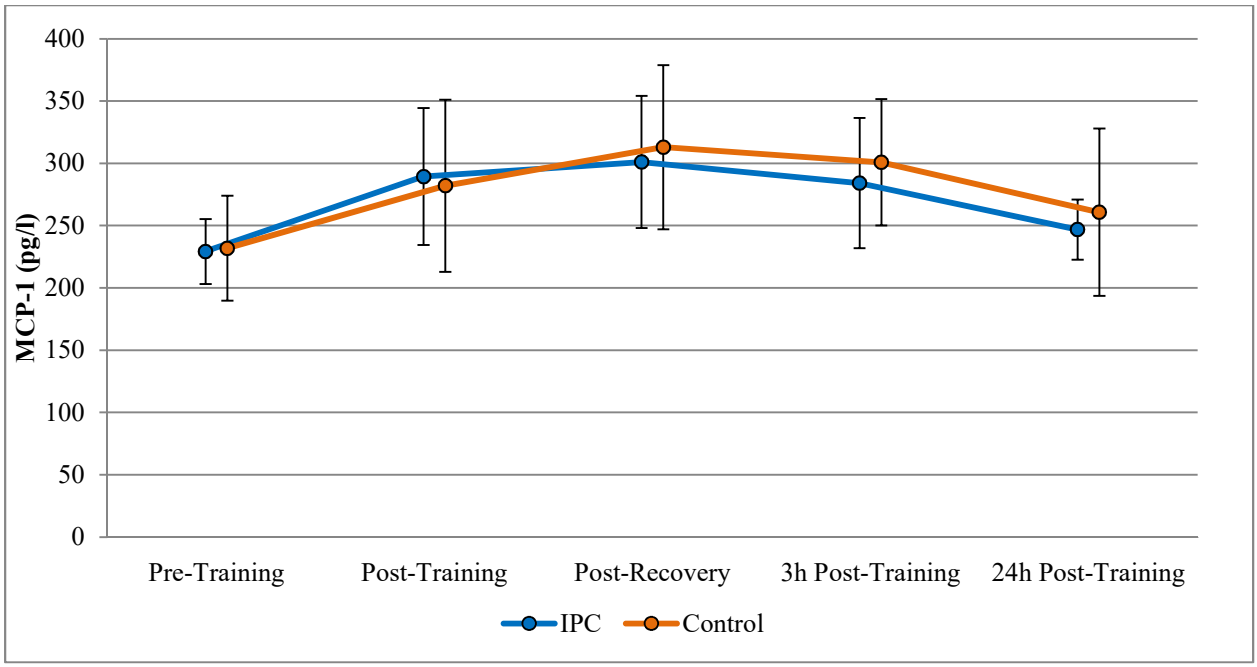


Figure 3.7 Recovery Group Differences in Monocyte Chemoattractant Protein-1 Plasma Concentrations: Mean ± SD of plasma MCP-1 concentrations at all time points, by recovery group. IPC recovery condition is represented by the blue line, control condition by the orange line. Concentration values are in pg/ml. No significant group differences were observed**.**

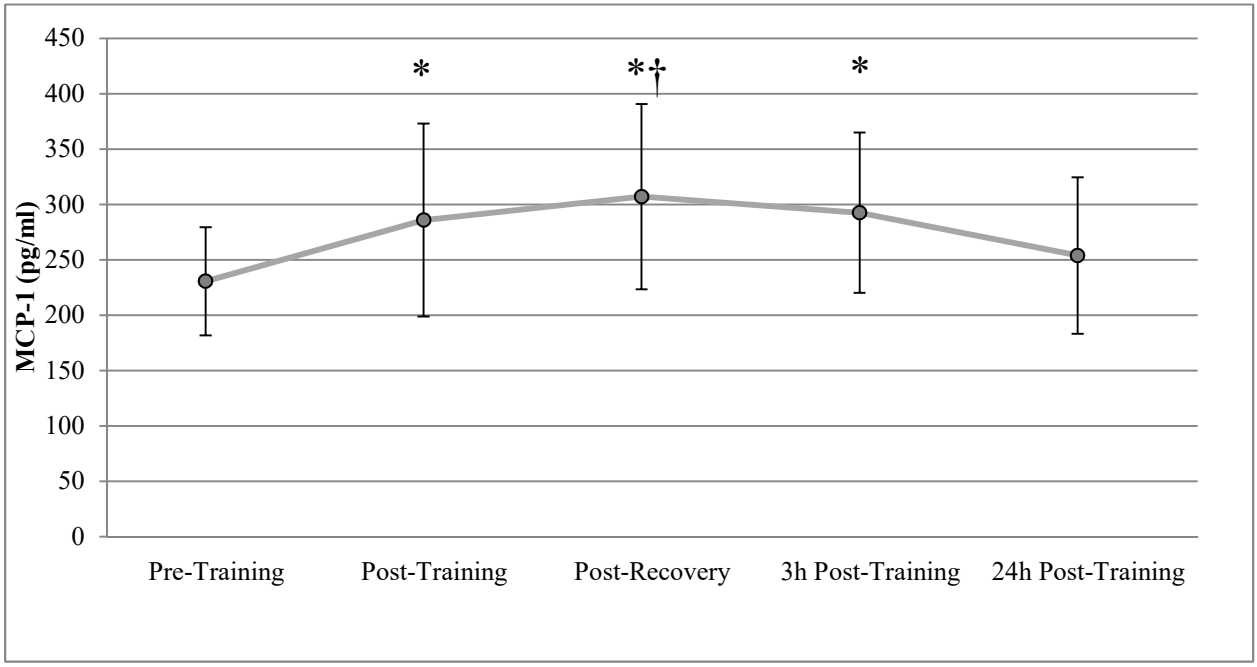


Figure 3.8 Aggregate Monocyte Chemoattractant Protein-1 Plasma Concentrations: Mean ± SD of plasma MCP-1 concentrations at all time points. Concentration values are in pg/ml. * indicates $p < 0.05$ compared to Pre. † indicates $p < 0.05$ compared to 24h Post-Training.

3.7.2 Muscle Damage Marker. Myoglobin concentrations were evaluated using an ELISA analysis kit. A repeated-measures ANOVA revealed a significant effect for time ($p < 0.01$). A Bonferroni post-hoc analysis was applied, and post-training, post-recovery and 3 hours post-training were found to be significantly greater than pre-training and 24 hours post-training Mb concentrations (see figure 3.10). No group ($p = 0.16$) or interaction ($p = 0.37$) effects were observed (see figure 3.9). A small effect size between groups ($d = 0.346$) was observed at baseline, while a moderate effect was seen post-training, post-recovery, and 3 hours post-training ($d = -0.627$, $d = -0.559$, and $d = -0.545$, respectively). A large group effect size was seen at 24 hours post-training ($d = -1.002$).

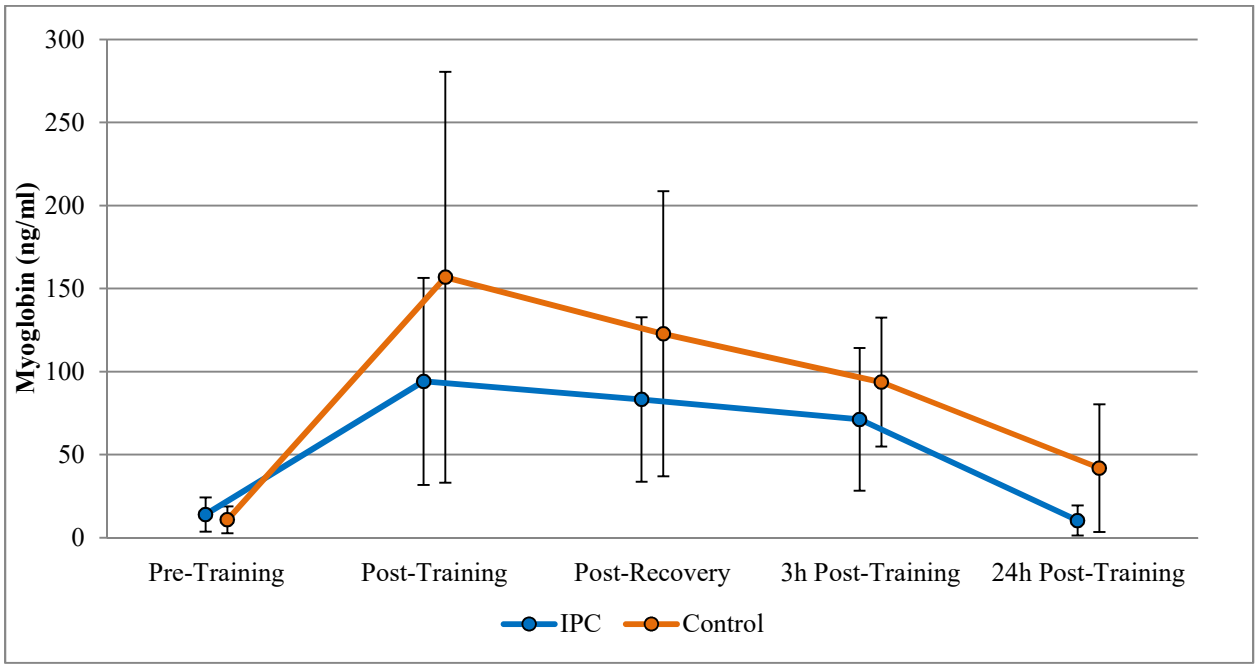


Figure 3.9 Recovery Group Differences in Myoglobin Serum Concentrations: Mean ± SD of serum Mb concentrations at all time points, by recovery group. IPC recovery condition is represented by the blue line, control condition by the orange line. Concentration values are in ng/ml. No significant group differences were observed.

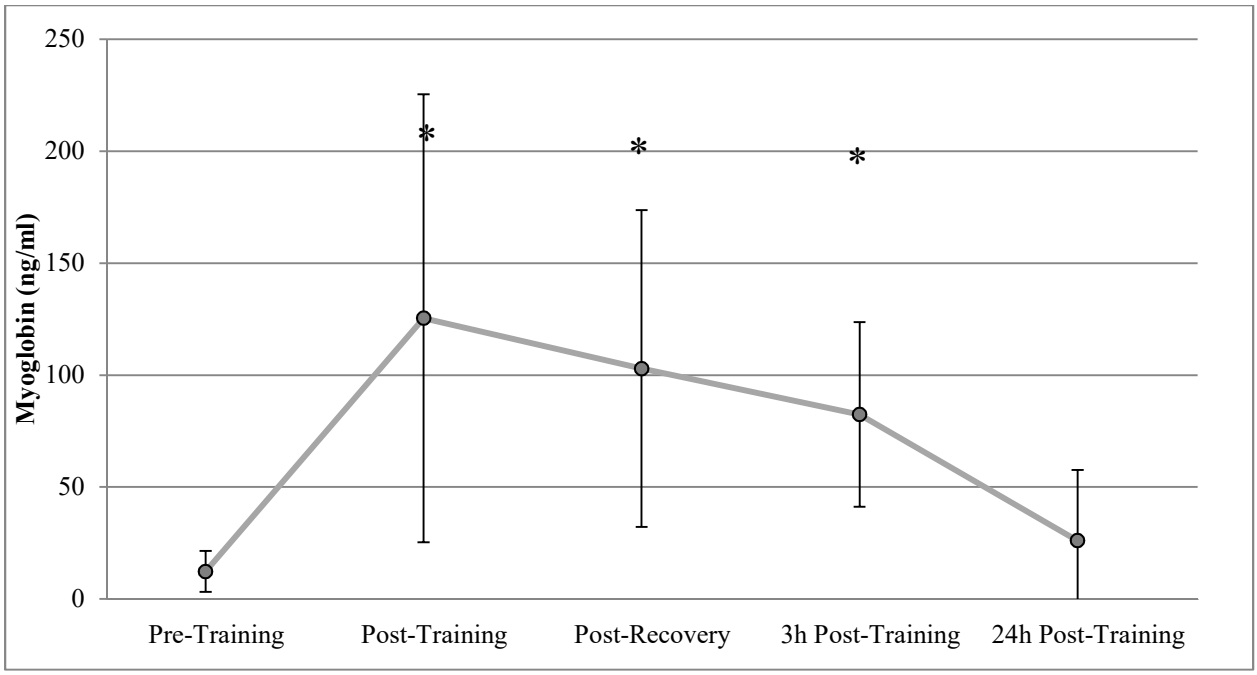


Figure 3.10 Aggregate Myoglobin Serum Concentrations: Mean ± SD of serum Mb concentrations at all time points. Concentration values are in ng/ml. * indicates $p < 0.05$ compared to Pre and 24h Post-Training.

Table 3.10 Numerical Data for Systemic Protein Concentrations

Variable	Pre-training (Mean ± SD)	Post-training (Mean ± SD)	Post-recovery (Mean ± SD)	3 hours post-training (Mean ± SD)	24 hours post-training (Mean ± SD)
Compiled IL-6 (pg/ml)	5.90 ± 7.8	6.73 ± 10.2	6.50 ± 8.0	7.65 ± 10.3	6.16 ± 8.3
IPC IL-6 (pg/ml)	5.28 ± 7.3	6.33 ± 10.0	7.14 ± 10.0	6.54 ± 10.8	5.61 ± 8.8
Control IL-6 (pg/ml)	6.51 ± 8.6	7.12 ± 11.1	5.86 ± 6.1	8.76 ± 10.4	6.71 ± 8.2
Compiled IL-10 (pg/ml)	21.83 ± 24.0	23.73 ± 29.9	22.04 ± 25.6	23.47 ± 30.2	21.93 ± 26.2
IPC IL-10 (pg/ml)	21.83 ± 23.0	22.74 ± 28.6	23.78 ± 32.4	21.23 ± 31.2	21.05 ± 26.9
Control IL-10 (pg/ml)	22.98 ± 26.4	24.71 ± 33.12	20.30 ± 18.7	25.71 ± 31.1	22.81 ± 27.2
Compiled MCP-1 (pg/ml)	230.54 ± 45.5	285.73 ± 76.9	306.97 ± 72.5	292.47 ± 70.1	253.79 ± 44.2
IPC MCP-1 (pg/ml)	229.23 ± 37.6	289.39 ± 79.4	301.00 ± 76.6	284.14 ± 75.6	246.81 ± 34.9
Control MCP-1 (pg/ml)	231.84 ± 60.9	282.06 ± 99.7	312.94 ± 95.1	300.80 ± 73.1	260.77 ± 96.9
Compiled Mb (ng/ml)	12.34 ± 9.1	125.44 ± 100.0	102.93 ± 70.6	82.41 ±41.2	26.11 ±31.5
IPC Mb (ng/ml)	13.91 ± 10.4	94.09 ± 62.4	83.19 ± 49.5	71.19 ± 43.0	10.31 ± 9.1
Control Mb (ng/ml)	10.76 ± 8.1	156.78 ± 123.7	122.66 ± 85.8	93.64 ± 38.8	41.91 ± 38.4

Note: SD = standard deviation, IL = Interleukin, MCP-1 = monocyte chemoattractant protein-1, Mb = Myoglobin.

3.8 Delayed-Onset Muscle Soreness

Self-reported soreness was evaluated using a VAS scale, and the distance to the mark was measured in mm. This data was analyzed using a two-way repeated-measures ANOVA. No statistically significant effects were observed between recovery groups ($p = 0.86$), for time ($p = 0.52$) or group by time ($p = 0.07$; see figure 3.11).

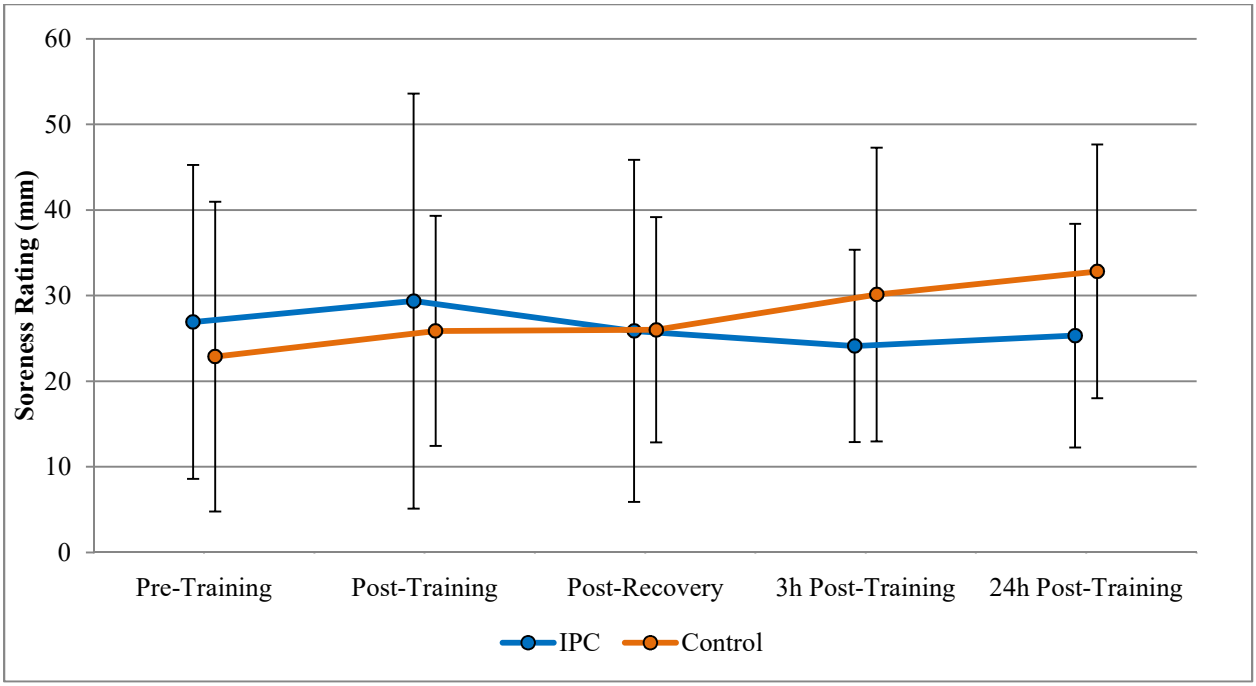


Figure 3.11 Self-Reported Delayed-Onset Muscle Soreness: Mean ± SD of DOMS at all time points, by recovery group. IPC recovery condition is represented by the blue line, control condition by the orange line. Results represent the distance in mm from the "0" mark on the visual analogue scale. No significant differences were observed**.**

Cohen's effect size statistic indicate trivial effects from IPC use post-training and post-recovery (d = 0.184 and d = -0.008, respectively), a small effect at baseline and 3 hours post-training (d = 0.228 and d = -0.418, respectively) and a moderate effect at 24 hours post-training (d = -0.534) on DOMS.

3.9 Responder and Non-Responder Analysis

Subgroups were created to observe if there were participants that had benefited from the IPC treatment compared to some that did not. Using CMJ height data comparing pre-training values to 24 hours post-training, the participants were placed into the responder group if they had no change or had increased their jump height when compared to baseline (≥ 0%) or in the non-responder if they had experienced a decrease in jump height (< 0%). A total of four (n = 4) participants were placed in each group, with equal distribution of participants that completed

their training and recovery on the Wednesday and Thursday (n = 2 from either per subgroup). No differences in participant characteristics were found.

Repeated-measures ANOVAs with responder/non-responder coding as categorical factors were run on all outcome variables using data from the IPC condition, reducing the number of cases to eight. There were no significant group effects or interaction effects; however, main effects for time were still significant for Mb and MCP-1. Results are presented in table 3.11.

Table 3.11 Repeated-Measures Analysis of Variance Results for Responder Analysis

Variable		Responder/Non-Responder	Time	Responder/Non-Responder by Time
CMJ Height	F	0.01	0.79	0.45
	p	0.94	0.49	0.66
CMJ Velocity	F	0.57	0.27	0.70
	p	0.49	0.77	0.53
CMJ Fi	F	1.76	1.52	0.10
	p	0.26	0.27	0.90
CMJ Peak Power	F	0.85	2.03	2.38
	p	0.41	0.19	0.15
CMJ Avg Power	F	2.19	1.34	0.86
	p	0.21	0.32	0.46
10m Sprint	F	1.39	0.35	0.01
	p	0.30	0.72	0.99
IL-6	F	0.67	1.49	0.92
	p	0.44	0.24	0.47
IL-10	F	0.64	0.58	1.29
	p	0.45	0.68	0.30
IL6/IL10 Ratio	F	1.28	0.58	0.49
	p	0.30	0.68	0.74
MCP-1	F	5.57	4.12	0.66
	p	0.06	0.01 *	0.62
Mb	F	2.22	12.81	1.32
	p	0.19	<0.01 *	0.29
DOMS	F	0.52	1.41	0.41
	p	0.51	0.27	0.80

Note: F = Fisher statistic of analysis of variance statistical test, CMJ = Countermovement Jump, 10m = ten metres, IL = Interleukin, MCP-1 = monocyte chemoattractant protein-1, Mb = Myoglobin, DOMS = delayed-onset muscle soreness. * indicates $p < 0.05$

DISCUSSION

The primary purpose of the study was to evaluate the effectiveness of an IPC protocol over a control condition for recovery following a strenuous training day in college football athletes. A total of eight (n = 8) male football athletes were recruited for the study. They underwent either 30 minutes of IPC or control condition following an intensive training day, and in a randomized crossover manner, were subject to the opposite recovery intervention the following week. Outcome variables included performance on two performance tests (CMJ and 10m sprint), blood markers of inflammation (IL-6, IL-10, and MCP-1) and muscle damage (Mb), as well as self-reported soreness (DOMS). Performance tests were done before training and at 3 hours and 24 hours post-training. Blood draws and self-reported soreness was assessed at the same time points as the performance measures, as well as immediately after training and immediately after the recovery session.

The main hypothesis was that the session of IPC would attenuate the loss in CMJ following training. It was thought that increase in blood flow from IPC use could promote a quicker inflammatory process which may then lead to quicker muscle damage recovery (A. H. Chen et al., 2001). Correspondingly, it was also hypothesized that there would be an increase in inflammatory marker (IL-6, IL-10, MCP-1) and muscle damage marker (Mb) concentrations 3 hours following training, and lower concentrations of the markers 24 hours following training with IPC use. Likewise, it was thought that IPC use would result in reduced soreness and attenuate the loss of performance on the 10m sprint.

The results of the present study indicate that there was no effect of the IPC protocol used in the study on the performance measures, inflammatory and muscle damage markers, or soreness during off-season training in the football athletes recruited.

4.1 Performance Test Results

Contrastingly to the primary hypothesis, no changes were observed in CMJ performance following 30 minutes of IPC use compared to passive recovery. Likewise, none were observed for the 10m sprint. While these results might suggest IPC does not enhance the recovery of performance on the given tests, there was also no significant drop in performance on either test, regardless of recovery intervention, when comparing baseline to 3 or 24 hours post-training.

Comparisons with normative data for vertical jump is complicated by the use of different measurement devices (Buckthorpe, Morris, & Folland, 2012). The Vertec (JumpUSA, Sunnyvale, USA) device is used for most testing combines and assessments in football athletes, including the National Football League (NFL; Fry & Kraemer, 1991; McGee & Burkett, 2003; Sawyer, Ostarello, Suess, & Dempsey, 2002; Sierer, Battaglini, Mihalik, Shields, & Tomasini, 2008), due to ease of use and affordability. That being said, vertical jump standards for football are set based on the use of the Vertec, and tend to be greater than those achieved on a force platform (Buckthorpe et al., 2012). Nevertheless, Harman and Garhammer (2008) indicated that college football athletes had a mean jump height of 53 cm (p. 278), which is near the 51.6 cm average achieved for pre-training measures in this study.

The results of the CMJ contrast the findings by Johnston et al. (2016), who found a significant decrease in vertical jump height, peak power, and rate of force development 24 hours following a speed and strength session in elite rugby players, similar to the training protocol employed by the present study. The lack of change in the participant physical performance might be indicative that they were either already experiencing performance detriments at baseline from previous training, or that their training sessions the day of the interventions were inadequate to

cause a statistically significant decrease in test performance. Johnston et al. (2016), unlike the present study, had given the subjects two days off of training leading into the training day which might have elicited ideal baseline test performance.

The lack of change in DOMS could also offer an explanation to the lack of change in jump height. Pain is often seen as an evolutionary protective mechanism to limit movement and prevent further tissue damage (Bonavita & De Simone, 2011; E. S. J. Smith & Lewin, 2009). If the participants were not experiencing greater soreness from their training, and thus no activation of the type III and IV afferent pain receptors (Murase et al., 2010), participants might have been uninhibited in their physical performance testing.

Despite no statistically significant difference, Cohen's effect size statistic noted a small effect size between groups at 3 hours post with IPC use, and the relative change in jump height at 24 hours post-training surpassed the intersession variability found by Moir, Shastri, and Connaboy (2008), where physically active men of various sporting backgrounds returned every week for four weeks under identical conditions, and performed three CMJs. They had established an intersession coefficient of variation (CV) between 4 and 5.6% (Moir et al., 2008). Hilmersson, Edvardsson, and Tornberg (2015) compared different vertical jump testing mats and observed an inter-session CV between 5.7 and 6.1% from CMJ height, depending on the measurement device.

On the other hand, circadian changes in jump height could have also played a role in some of the variability seen in the data (Guette, Gondin, Duclay, & Martin, 2005; Unver & Atan, 2015). Unver and Atan (2015) observed significantly lower CMJ height values at 09:00 when compared to measurements taken at 14:00 and 19:00 (34.58 cm compared to 36.27 cm and 36.88

cm, respectively). Similarly, Guette et al. (2004) observed significantly lower CMJ results at 06:00 when compared to measurements taken at 10:00, 14:00, 18:00, and 22:00, with an average difference of 1.3 cm between 06:00 and the other time points. Morning jumps appear to be lower than afternoon and evening time points, however, there are many factors that can influence circadian rhythm (Reilly & Waterhouse, 2009). Reilly and Waterhouse (2009) discussed that there are environmental, sleep/wake changes, and internal (e.g. metabolic and hormonal) factors that can play a role in circadian changes to sport performance. For the present study, pre-training testing occurred at 14:00, 3 hours post-training was at approximately 20:00 (± 15 minutes) and 24 hours post-training at approximately 17:00 (± 15 minutes) the following day. Since the jumps all occurred in the afternoon and evenings, it is unclear whether such a pattern was present within the span of testing times. The challenge with the use of CMJ height and power output from a force platform is that, within the context of elite sports, a clinically significant change in jump height is within or marginally larger than the expected test-retest and circadian variability seen with CMJ. Fry and Kraemer (1991) contrasted performance of football athletes within each of the three divisions in the National Collegiate Athletic Association (NCAA). They observed that Division II players jumped an average of 4.4% lower than Division I, while Division III players jumped approximately 7.0% lower than Division I athletes (Fry & Kraemer, 1991). The authors also performed a subgroup analysis and divided each participant by playing position, and whether they were a “starter” or “non-starter”. Non-starters jumped an average of 7.9% lower than starting players. On a professional level, test results from the National Football League (NFL) indicate that those drafted jumped an average of 3.2% higher than those that were not drafted (Sierer et al., 2008). The relative change in CMJ height for this study from pre-training to 24 hours post-training was -6.9% for IPC and 6.5% for control (see table 3.7).

When examining sprinting times, Fry and Kraemer (1991) found that for the 40 yard dash test, NCAA football athletes in Division I completed the test 0.81% and 1.61% quicker than Division II and III athletes, respectively. Likewise, starters completed the 40 yard dash 2.78% quicker than non-starting athletes. NFL drafted football players completed the test 2.04% faster than non-drafted athletes (Sierer et al., 2008). While the distance covered in the 10m sprint test is shorter than the 40 yard dash (36.6 metres), results from 10 and 20 yard splits in the 40 yard dash tests are strongly correlated to one another (McGee & Burkett, 2003). That being said, the relative changes observed in 10m sprint time for the present study did not exceed 0.45% (see table 3.9) at all time points, which suggests that there were no clinically meaningful changes in sprint times.

4.2 Inflammation and Muscle Damage Markers

In section 1.2.6, a couple of physiological mechanisms that could be altered by IPC to improve recovery were discussed. These included an increase in NO secretion by endothelial cells and greater leukocyte extravasation into damaged tissues. Through surrogate measures, the present study sought to evaluate whether any of these systems were altered. Circulating inflammatory cytokines could have given an indication of changes to the inflammatory phase of healing following tissue injury. There were significant increases in MCP-1 and Mb following training until 3 hours post-training, however, no group differences were noted with these markers. IL-6 and IL-10 concentrations had no significant recovery group or time differences in any of the markers measured. When examining the IL-6/IL-10 ratio to get an idea of pro-/anti-inflammatory balance, no statistically significant effect was observed either.

There was a main effect for time in Mb concentrations following training, suggesting that muscle damage was likely sustained during the training sessions. Typical serum concentrations of Mb range from 5 – 100 ng/ml (Davis, Barrett, Torre, & Wacasey, 1996; Stone, Waterman, Harimoto, & Murray, 1977), however, younger individuals tend to have lower values (Davis et al., 1996). Bombardieri et al. (1982) observed a moderate positive correlation with body mass and resting myoglobin concentrations, with concentrations ranging from 10 ng/ml to 40 ng/ml. No correlation was observed between body mass and pre-training measures (data not shown) with the participants in this study. Despite most measurement points not surpassing the threshold of 100 ng/ml post-training in the current study, Barrett et al. (1996) discussed that Mb has a wide range of basal concentrations, and that an increase of 50% from those baseline values could also be declared clinically significant when considering myocardial infarction diagnosis, even without surpassing the 100 ng/ml threshold. In the context of EIMD, a 4-fold increase from resting values can be expected following exercise (Brancaccio et al., 2010). An approximately 10-fold increase from baseline to post-training in Mb was observed in the current study suggesting skeletal muscle damage occurred.

Changes in Mb observed follow slight deviations from the typical response seen in Mb following physical exertion. Typically, concentrations increase post-exercise, peaking between three and six hours following exercise and decline at approximately 24 hours post-exercise (Bailey et al., 2007; Brancaccio et al., 2010; Peake, Wilson, Mackinnon, & Coombes, 2005; Townsend, Kahanov, & Eberman, 2014). This response is not exclusive to controlled muscle damaging protocol, as it has also been observed following standard weight training sessions (French et al., 2008; Ihalainen et al., 2014), endurance training (Peake, Wilson, et al., 2005), and competitive football games (Hoffman et al., 2002; Kraemer et al., 2009). The present study

found the greatest Mb concentrations immediately post-exercise and decreased progressively until 24 hours post-exercise. While it is interesting that the surrogate measure for muscle damage increased post-training without a drop in physical performance, other research groups have found similar results in muscle damaging studies, where blood concentrations of muscle damage markers such as CK or Mb increase significantly, without a manifested change in performance (French et al., 2008; Gilson et al., 2010).

Small and trivial effect sizes were observed for all markers except Mb, where a moderate effect was seen post-training, post-recovery, and 3 hours post-training, as well as a large effect 24 hours post-training. Mb concentrations were lower for the IPC condition at all time points except for baseline, however, since this effect was also noted before the recovery session, the data does not suggest that there was any influence of the IPC devices on reducing Mb concentrations.

Concentrations of IL-6 observed tended to be near values that were expected for healthy individuals over the age of 18, while IL-10 concentrations were slightly higher than what would be expected (Kleiner, Marcuzzi, Zanin, Monasta, & Zauli, 2013). Other studies examining the inflammatory response to EIMD through various protocols have found similar results where systemic IL-6 and IL-10 concentrations undergo no significant changes following the muscle damaging stimulus (Cornish & Johnson, 2014; Kanda et al., 2013; Robson-Ansley, Cockburn, Walshe, Stevenson, & Nimmo, 2010). Contrastingly, others studies found significant increases in post-exercise IL-6 (Peake, Suzuki, et al., 2005; L. L. Smith et al., 2000) or both IL-6 and IL-10 (Izquierdo et al., 2009). Robson-Ansley et al. (2010) found that systemic concentrations of IL-6 did not increase following eccentric muscle damage. The authors discussed that muscle damage might be an insufficient stimulus to cause increases in this cytokine, especially in comparison to

endurance exercise where oxidative stress and glycogen depletion also occur and can cause stress to the intramuscular environment (Robson-Ansley et al., 2010). IL-6 has been demonstrated to increase in response to intramuscular glycogen depletion (Febbraio & Pedersen, 2002). This could indicate that while the training the participants completed was muscle damaging, it might not have led to a sufficient depletion of intramuscular glycogen to increase systemic IL-6. Peake, Della Gatta, Suzuki, and Nieman (2015) discussed that the concentration of intramuscular cytokine likely needs to hit a certain threshold before it can diffuse into systemic circulation.

MCP-1 concentrations increased significantly over time, where post-exercise, post-recovery, and 3 hours post-training levels were greater than pre-training measures. MCP-1 post-recovery concentrations were also significantly greater than those at 24 hour post-training. This response observed follows a typical pattern for post-exercise MCP-1 values (Tidball, 2005), however, not all research has observed changes post-exercise (Brancaccio et al., 2010). Typical resting concentrations in a healthy population ranges from 20.1 pg/ml to 78.9 pg/ml (Kleiner et al., 2013), which was much lower than all values measured as part of the current study. It is interesting to note that messenger Ribonucleic Acid (mRNA – indicative of gene expression) of MCP-1 has been demonstrated to be upregulated following 150 minutes of IPC treatment in mice (Roseguini et al., 2010), however, no group differences in plasma MCP-1 concentrations were noted in the present study. While vast protocol differences exist between the studies (species, treatment time, method of MCP-1 measurement), it could be worthwhile to investigate mRNA response of various genes in future research.

The repeated bout effect could offer an explanation as to why no differences were observed in IL-6 and IL-10 concentrations. The athletes participating in the study were at the end of a four week training block, where they had already completed two weeks of similar workouts

before the first intervention week. Prescribed exercises were identical from week to week, and the only changes were in the quantity of repetitions and the intensity of sets (as a percentage of 1RM) for the main exercises. The changes were minimal and counterbalanced due to the randomized crossover design of the study (see table 3.4) which allowed for better weekly comparison, however, such training schedule could have resulted in the repeated bout effect, where similar training stimuli no longer elicit as great of a performance detriment and inflammatory response due to a yet to be identified mechanism (Cleary, Kimura, Sitler, & Kendrick, 2002; McHugh, 2003; Peake, Nosaka, & Suzuki, 2005). Systemic concentrations of IL-6 following eccentric exercise has been observed to be reduced or similar to basal concentrations if an identical bout of eccentric exercise has been performed beforehand (T. C. Chen & Hsieh, 2001; L. L. Smith et al., 2007). T. C. Chen and Hsieh (2001) evaluated the inflammatory response to seven consecutive days of eccentric training of the elbow flexor. There was a systemic elevation of IL-6 following the bout on the first day; however, IL-6 concentrations returned to baseline for the remaining six days (T. C. Chen & Hsieh, 2001). In the case of IL-10, there appears to be an increase in concentrations subsequent to the second bout of eccentric training (Hirose et al., 2004; L. L. Smith et al., 2007). Contrastingly, IL-10 has not been as extensively studied as IL-6 and there have been no studies to date examining IL-10 concentrations for more than two bouts, making it unclear as to whether this increased IL-10 concentration is sustained over multiple bouts. If this were to be the case, then perhaps the repeated bout effect played a role in IL-10 concentrations for the current study.

While the repeated bout effect is mostly studied in eccentric exercises (McHugh, 2003; Paulsen et al., 2012; L. L. Smith et al., 2007; L. L. Smith, Keating, et al., 1994), this effect has also been seen as part of standard weight training with both concentric and eccentric phases

(Coratella, Chemello, & Schena, 2015; Doma et al., 2015) and plyometric training (Jamurtas et al., 2000). The repeated bout effect can last between one (T. C. Chen, Chen, Lin, Yu, & Nosaka, 2016) and six months following the initial stimulus (Nosaka, Sakamoto, Newton, & Sacco, 2001), well beyond the two-week span of this study.

Beyond the repeated bout effect, there is the possibility that participants were undergoing pre-training measures in an already inflamed state. Both IL-10 and MCP-1 concentrations were above the expected ranges for healthy individuals (Kleiner et al., 2013), even at pre-training measures, which might have masked an acute immune response from the training stimulus.

Alterations to the inflammatory response should not be discounted as a possibility when using IPC. Only three cytokines of the multitude that can be implicated in the post-exercise inflammatory response have been measured (Kleiner et al., 2013) Likewise, only the systemic response was evaluated, rather than the muscle specific response, which can be drastically different to the systemic milieu (Malm et al., 2004; Peake et al., 2015).

4.3 Self-Reported Soreness

There was no significant difference in DOMS at all time points and no effect for recovery measures. While there was a small effect size for lower DOMS in the IPC group compared to control at 3 and 24 hours post-training, this effect might result from a placebo effect arising from expected results. Within a clinical context, a meaningful change in pain is usually established between a 30 to 40 point reduction on a 100 point scale, or a decrease of $\geq$ 33% from the initial pain rating (Bird & Dickson, 2001; Cepeda, Africano, Polo, Alcala, & Carr, 2003; Farrar, Berlin, & Strom, 2003; Farrar, Portenoy, Berlin, Kinman, & Strom, 2000; Lee, Hobden, Stiell, & Wells, 2003). A few report a lower range, between 10 and 15 mm along a 100 mm VAS (Kelly, 2001;

Todd, Funk, Funk, & Bonacci, 1996), however, these studies examined acute pain in patients admitted to hospital emergency departments. Currently, the literature is unclear as to whether patients with higher self-reported pain will require a greater drop along a VAS to be considered clinically significant (Bird & Dickson, 2001; Kelly, 2001; Todd et al., 1996). Fewer studies have evaluated DOMS specifically, however, a pilot study evaluating massage post-race in ultramarathon athletes indicated that a change between 3.75 and 3.90 along a 10 point VAS scale was considered significant (Visconti, Capra, Carta, Forni, & Janin, 2015). The greatest difference observed in the present study is a 10.0 mm increase from pre-training to 24 hours post-training in the control group. As the 10.0 mm minimum clinically meaningful difference from Kelly (2001) was established for acute pain in emergency rooms, the applicability of the results to the present study is limited and thus, the changes in DOMS did not achieve clinical significance.

Unfortunately, due to the recovery modalities employed in the study, participants could not be blinded to allocation, and thus might have had pre-existing expectations of the IPC devices to reduce soreness, which could have influenced their subjective rating of pain (Driller & Halson, 2013; MacRae et al., 2011). While not formally tracked, casual conversation between subjects revealed that two (n = 2) participants expressed that they did not believe the devices provided any benefit to soreness, and may have been subject to the *nocebo effect* (Colloca, Sigaudo, & Benedetti, 2008), where the belief of a treatment will cause a detriment to outcome variables and can influence subjective results. Other factors may also influence pain sensitivity, such as genetic makeup (Fernandez Robles, Degnan, & Candiotti, 2012), ethnicity (Edwards, Doleys, Fillingim, & Lowery, 2001), and mood (Tang et al., 2008). Athletes might also be less susceptible to pain than regularly active individuals (Pen & Fisher, 1994; Tesarz, Schuster, Hartmann, Gerhardt, & Eich, 2012), which might explain the lack of significant change over

time observed. As previously discussed, the repeated bout effect can also lead to lower reported soreness following a subsequent bout of exercise (L. L. Smith, Fulmer, et al., 1994), which could have also manifested itself in the DOMS results of the present study.

To account for the placebo effect, a third intervention consisting of a sham treatment, such as a sham ultrasound (Howatson & Van Someren, 2003) could have been used to elucidate whether that small effect size observed on DOMS was in fact from IPC or if there was a placebo effect present.

4.4 Study Limitations

The current study presents several limitations including:

- The study occurred in the off-season, where recovery might not be as critical as before a competitive match
- Insufficient returned food logs for accurate analysis of covariance
- Inconsistent volume for exercise intervention
- Limited body composition analysis
- Missing data had to be computed to retain sample size
- Assumption that IPC increased blood flow with current population
- Indirect measures of muscle damage

To address some of the limitations, the familiarization trial could have included a more in-depth body composition analysis, either using bioelectric impedance or multi-site skinfold measurements. This could have elucidated whether body composition had any influence on the variables measured in the study, rather than simply using BMI which presents many issues with

collegiate athlete populations when trying to predict body fat percentage (Ode, Pivarnik, Reeves, & Knous, 2007). While the average BMI of 28.6 would indicate the participants were overweight, the participants likely had greater muscle mass than most members of the general public with a similar BMI score due to the nature of their training and sporting demands.

A greater level of control could have been exerted by modifying the study design to have a washout period with no training ahead of the intervention day, and implementing an entirely identical workout on the following week, similar to Johnston et al. (2016). That being said, such conditions are rarely encountered in the life of high performance athletes, and would reduce the external validity of the study. Likewise, the coaching staff would have been unlikely to grant consent to withdraw athletes for an extended period of time away from their training to accommodate the study. Implementing a design that includes a washout would require the study to occur during a break where athletes are not required to train, or the use of recreationally active populations rather than athletes.

4.5 Generalizability

The results of the study will have varied applicability to the sporting community beyond the sport of American or Canadian football, depending on the outcome variable of interest. For the performance measures (CMJ and 10m sprint), results from these tests will likely be constrained to power based sports, such as long jump, diving, or 100m dash, where explosive actions are a dominant portion of the sport (Pincivero & Bompa, 1997). These performance tests were selected due to their broad application in other sports. The training prior to the recovery interventions was specific to football, and thus the inflammatory and muscle damage response was reflective of the demands of off-season football. That being said, the biochemical response

that occurred as a result of the training might suggest a similar response (e.g. no change in IL-6 and IL-10, but increases in MCP-1 and Mb) might occur in athletes of different sports, but at different magnitudes based on the degree of EIMD experienced. Lastly, while perception of pain and soreness is a subjective experience, there might be some differences between athletes of various sports and the likelihood of reporting a similar painful stimulus based on their participation. While no research has been done to compare athletes of different sports and DOMS following a relatively identical training stimulus, it would not be unreasonable to hypothesize that contact sport athletes might rate a given soreness level lower than non-contact athletes, which would reduce the applicability of the DOMS results beyond contact sports.

IPC is but one of the many modalities that have been used in the recovery process for athletes (Cheung et al., 2003; Lewis et al., 2012). While soreness has been linked to performance detriments, scientific evidence appears to point to the inflammatory process following muscle damage – with soreness as a by-product - as an essential step to repair and strengthening of the tissue. Seeking to reduce inflammation might not be in the best interest of those seeking benefits of training, such as athletes in a build phase and individuals training to increase fitness. For example, NSAIDs used following resistance training showed acute reductions in protein synthesis and impaired satellite cell activation in participants using them (Bondesen, Mills, Kegley, & Pavlath, 2004; Bondesen, Mills, & Pavlath, 2006; Markworth, Vella, Figueiredo, & Cameron-Smith, 2014; Mikkelsen & Langberg, 2009; Schoenfeld, 2012; Trappe et al., 2002). This could lead to impaired regeneration of damaged muscle tissue (Almekinders & Gilbert, 1986; Mishra, Friden, Schmitz, & Lieber, 1995) or ligaments (Elder, Dahners, & Weinhold, 2001). Similarly, Roberts, Raastad, et al. (2015) found that chronic cold water immersion (CWI) can reduce protein synthesis and strength gains. They had 21 men undergo strength training

twice weekly for 12 weeks, followed by an active recovery or CWI treatment post-training, and found that increases in muscle mass, strength, and type II muscle fiber area were attenuated in the CWI group after the 12 weeks when compared to the active recovery group (Roberts, Raastad, et al., 2015). Likewise, they looked at the acute molecular response of satellite cell activation and phosphorylation of downstream products of mammalian target of rapamycin (mTOR), key markers of muscle hypertrophy signalling. Participants in the CWI group presented a blunted response of these markers, which suggest that the hypertrophic response was negatively altered from the CWI (Roberts, Raastad, et al., 2015). Similarly, studies with animal models found reduced inflammation following cryotherapy use after inducing injury (Takagi et al., 2011; Vieira Ramos et al., 2016). That being said, outcome results are mixed with Takagi et al. (2011) observing hampered muscle repair, while Vieira Ramos et al. (2016) observed similar healing when compared to control conditions.

Cryotherapy can also reduce the intramuscular temperature, slowing metabolic reactions (including aerobic metabolism) which results in decreased production of ROS (Carvalho et al., 2010; White & Wells, 2013). While touted as a beneficial effect for post-exercise recovery due to reduced secondary damage (White & Wells, 2013), ROS is essential in signalling for subsequent inflammatory and regeneration processes (Lockhart & Brooks, 2008). While these reactions were not completely ablated as a result of cryotherapy in both Carvalho et al. (2010) and White and Wells (2013), reduction of ROS below a given threshold might hamper downstream steps of the inflammatory cascade.

Studies examining local (topical) cryotherapy of tissue found that the cooling led to significantly reduced leukocyte rolling on the endothelium and infiltration into skeletal muscle (Menth-Chiari, Curl, Smith, & Smith, 1999; Westermann, Vollmar, Thorlacius, & Menger,

2000). This could be explained by the negative influence of vasoconstriction on the events of leukocyte extravasation, which include reduced blood flow to muscle tissue (Roberts, Muthalib, et al., 2015; Thorsson, Lilja, Ahlgren, Hemdal, & Westlin, 1985), and lower vascular permeability (Eston & Peters, 1999; Wilcock, Cronin, & Hing, 2006).

On the other hand, recovery modalities that promote vasodilation following training could do the opposite, by promoting increased permeability of the blood vessels and greater blood flow (Wilcock et al., 2006). Such modalities include active recovery (Pearson et al., 2011; Roberts, Raastad, et al., 2015), heat application (Knight & Londeree, 1980; Wilcock et al., 2006), IPC (A. H. Chen et al., 2001), and massage (Munk, Symons, Shang, Cheng, & Yu, 2012; Portillo-Soto, Eberman, Demchak, & Peebles, 2014).

Research models examining thermal stress of a fever response have shown upregulation in CAMs near lymphatic tissue (Appenheimer, Chen, Girard, Wang, & Evans, 2005; Q. Chen et al., 2006; Shah et al., 2002) and vasodilation, resulting in increased blood flow (Blatteis, 2003). These physiological changes result in increased leukocytes in those tissues (Appenheimer et al., 2005; Blatteis, 2003; Tulapurkar et al., 2012). While long term studies in humans have yet to be done, heat application in EIMD models in mice between one and four weeks in duration have shown improved muscle regeneration when compared to control conditions (Kojima et al., 2007; Selsby et al., 2007). Kojima et al. (2007) also found greater satellite cell proliferation and greater protein synthesis in the heat treatment group of their study.

All things considered, the use of post-exercise recovery modalities to improve skeletal muscle recovery should emphasize an increase in blood flow to maximize the immune system potential and to improve satellite cell proliferation within the tissue. Nevertheless, from a

practical standpoint, reducing inflammation might still be advised as a course of action, despite indications that recovery might be impaired. Athletes facing upcoming competition, where long term adaptations are not a concern, could consider anti-inflammatory modalities that may reduce soreness to remove discomfort while training and competing.

It appears that a homeostatic balance must be found for proper adaptation to EIMD, where inadequate inflammation could hamper repair processes, and excessive inflammation could lead to pathological conditions such as overtraining (Tiidus, 1998, 2010). The use of therapies by athletes and their prescription or recommendation by coaching staff should recognize that falling outside of the homeostatic bandwidth of inflammation might be detrimental to recovery.

CONCLUSION

The results of the present study indicate that the IPC protocol employed did not alter CMJ and 10m sprint performance following a heavy off-season training day for collegiate football athletes when compared to a passive rest control condition. Likewise, the single bout of IPC had no effect on inflammatory and muscle damage markers, nor was there an influence on DOMS. A back-to-back training day in the last two weeks of a training block consisting of speed and strength workouts can lead to significant increases in blood concentrations of MCP-1 and Mb, but not IL-6 or IL-10, with no apparent detriment to physical performance or any increase in DOMS.

5.1 Recommendations for Future Research

Owing to the fact that research on IPC as an athletic recovery tool is still in its infancy, many suggestions can be made for future research based on the findings of the present study.

Examining other immune markers, such as those with a greater link to muscle metabolism (e.g. IL-15, Decorin) could be of value. Likewise, the use of multiplex technology to evaluate a plethora of inflammatory markers could uncover other potential proteins that are either directly or indirectly influenced by IPC. Evaluation of messenger ribonucleic acid (mRNA) could also be of value to uncover changes in gene expression and the potential for transcription and translation of new proteins. Furthermore, the protein content of specific immunomodulators could be analyzed in skeletal muscle tissue, rather than in the circulation.

Beyond the immune system, investigation in metabolic changes from IPC within the damaged muscle tissue, such as changes in satellite cell proliferation or muscle protein synthesis

could help unravel the mechanism whereby IPC putatively is effective. Knowing this information could elucidate whether other aspects of muscle recovery are influenced by IPC, and to discover what upstream mechanisms the compression or increased blood flow might impact.

Lastly, evaluating changes in muscle and tissue oxygenation and blood flow using various inflation protocols (e.g. pressure setting, inflation time, cycle time) to establish predictive equations and to find optimal pressure settings for improved blood flow could greatly improve research protocols for future work with IPC. It could also simplify the programming of protocols into IPC devices and ease comparisons between makes and models from different companies manufacturing the devices, both in the athletic and medical fields.

BIBLIOGRAPHY

Abu-Own, A., Cheatle, T., Scurr, J. H., & Coleridge-Smith, P. D. (1993). Effects of intermittent pneumatic compression of the foot on the microcirculatory function in arterial disease. *European Journal of Vascular Sugery*, *7*(5), 488–492.

Almekinders, L. C., & Gilbert, J. A. (1986). Healing of experimental muscle strains and the effects of nonsteroidal antiinflammatory medication. *American Journal of Sports Medicine*, *14*(4), 303–308. http://doi.org/10.1177/036354658601400411

Alon, R., & Ley, K. (2008). Cells on the run: shear-regulated integrin activation in leukocyte rolling and arrest on endothelial cells. *Current Opinion in Cell Biology*, *20*(5), 525–532. http://doi.org/10.1016/j.ceb.2008.04.003

Anderson, J. E. (2000). A role for nitric oxide in muscle repair: Nitric oxide–mediated activation of muscle satellite cells. *Molecular Biology of the Cell*, *11*(5), 1859–1874.

Anderson, J. E. (2016). Hepatocyte growth factor and satellite cell activation. In J. White & G. Smythe (Eds.), *Growth Factors and Cytokines in Skeletal Muscle Development, Growth, Regeneration, and Disease* (pp. 1–25). Cham, Switzerland: Springer International Publishing.

Appenheimer, M. M., Chen, Q., Girard, R. A., Wang, W.-C., & Evans, S. S. (2005). Impact of fever-range thermal stress on lymphocyte-endothelial adhesion and lymphocyte trafficking. *Immunological Investigations*, *34*(3), 295–323. http://doi.org/10.1081/IMM-200064501

Armstrong, R. B. (1984). Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. *Medicine & Science in Sports & Exercise*, *16*(6), 529–538.

Ascensão, A., Leite, M., Rebelo, A. N., Magalhães, S., & Magalhães, J. (2011). Effects of cold water immersion on the recovery of physical performance and muscle damage following a one-off soccer match. *Journal of Sports Sciences*, *29*(3), 217–225. http://doi.org/10.1080/02640414.2010.526132

Assumpção, C. D. O., Lima, L. C. R., Oliveira, F. B. D., Greco, C. C., & Denadai, B. S. (2013). Exercise-induced muscle damage and running economy in humans. *The Scientific World Journal*, *2013*, 1–11. http://doi.org/10.1155/2013/189149

Bailey, D. M., Erith, S. J., Griffin, P. J., Dowson, A., Brewer, D. S., Gant, N., & Williams, C. (2007). Influence of cold-water immersion on indices of muscle damage following prolonged intermittent shuttle running. *Journal of Sports Sciences*, *25*(11), 1163–1170. http://doi.org/10.1080/02640410600982659

Barnett, A. (2006). Using recovery modalities between training sessions in elite athletes: Does it help? *Sports Medicine*, *36*(9), 781–796. http://doi.org/10.2165/00007256-200636090-00005

Bird, S. B., & Dickson, E. W. (2001). Clinically significant changes in pain along the visual

analog scale. *Annals of Emergency Medicine*, *38*(6), 639–643. http://doi.org/10.1067/mem.2001.118012

Bischoff, R. (1997). Chemotaxis of skeletal muscle satellite cells. *Developmental Dynamics*, *208*(4), 505–515. http://doi.org/10.1002/(SICI)1097-0177(199704)208:4<505::AID-AJA6>3.0.CO;2-M

Blatteis, C. M. (2003). Fever: Pathological or physiological, injurious or beneficial? *Journal of Thermal Biology*, *28*(1), 1–13. http://doi.org/10.1016/S0306-4565(02)00034-7

Bochmann, R. P., Seibel, W., Haase, E., Hietschold, V., Rödel, H., & Deussen, A. (2005). External compression increases forearm perfusion. *Journal of Applied Physiology*, *99*(6), 2337–2344. http://doi.org/10.1152/japplphysiol.00965.2004

Bombardieri, S., Clerico, A., Riente, L., Grazia, M., Chicca, D. E. L., & Al, B. E. T. (1982). Circadian variations of serum myoglobin levels in normal subjects and patients with polymyositis. *Arthritis and Rheumatism*, *25*(12), 1419–1424.

Bonavita, V., & De Simone, R. (2011). Pain as an evolutionary necessity. *Neurological Sciences*, *32*(suppl. 1), 61–66. http://doi.org/10.1007/s10072-011-0539-y

Bondesen, B. A., Mills, S. T., Kegley, K. M., & Pavlath, G. K. (2004). The COX-2 pathway is essential during early stages of skeletal muscle regeneration. *American Journal of Physiology - Cell Physiology*, *287*(2), 475–483. http://doi.org/10.1152/ajpcell.00088.2004

Bondesen, B. A., Mills, S. T., & Pavlath, G. K. (2006). The COX-2 pathway regulates growth of atrophied muscle via multiple mechanisms. *American Journal of Physiology - Cell Physiology*, *290*(6), C1651–C1659. http://doi.org/10.1152/ajpcell.00518.2005

Born, D.-P., Sperlich, B., & Holmberg, H. (2013). Bringing light into the dark : Effects of compression clothing on performance and recovery. *International Journal of Sports Physiology and Performance*, *8*(1), 4–18.

Børsheim, E., & Bahr, R. (2003). Effect of exercise intensity, duration and mode on post-exercise oxygen consumption. *Sports Medicine (Auckland, N.Z.)*, *33*(14), 1037–1060. http://doi.org/10.2165/00007256-200333140-00002

Brancaccio, P., Lippi, G., & Maffulli, N. (2010). Biochemical markers of muscular damage. *Clinical Chemistry and Laboratory Medicine*, *48*(6), 757–767. http://doi.org/10.1515/CCLM.2010.179

Breivik, E. K., Björnsson, G. A., & Skovlund, E. (2000). A comparison of pain rating scales by sampling from clinical trial data. *The Clinical Journal of Pain*, *16*(1), 22–28. http://doi.org/10.1097/00002508-200003000-00005

Brockett, C. L., Morgan, D. L., & Proske, U. (2004). Predicting hamstring injury in elite athletes. *Medicine and Science in Sports and Exercise*, *36*(3), 379–387. http://doi.org/10.1249/01.MSS.0000117165.75832.05

Brophy-Williams, N., Driller, M. W., Shing, C. M., Fell, J. W., & Halson, S. L. (2015). Confounding compression: the effects of posture, sizing and garment type on measured interface pressure in sports compression clothing. *Journal of Sports Sciences*, *33*(13), 1–8. http://doi.org/10.1080/02640414.2014.990489

Brown, S. J., Child, R. B., Donnelly, A. E., Saxton, J. M., & Day, S. H. (1996). Changes in human skeletal muscle contractile function following stimulated eccentric exercise. *European Journal of Applied Physiology and Occupational Physiology*, *72*(5), 515–521. http://doi.org/10.1007/BF00242284

Bruunsgaard, H., Galbo, H., Johansen, T. L., Maclean, D. A., & Pedersen, B. K. (1997). Exercise-induced increase in serum interleukin-6 in humans is related to muscle damage. *Journal of Physiology*, *499*(3), 833–841.

Buckthorpe, M., Morris, J., & Folland, J. P. (2012). Validity of vertical jump measurement devices. *J Sports Sci*, *30*(1), 63–69. http://doi.org/10.1080/02640414.2011.624539

Byrne, C., & Eston, R. (2002). Maximal-intensity isometric and dynamic exercise performance after eccentric muscle actions. *Journal of Sports Sciences*, *20*(12), 951–959. http://doi.org/10.1080/026404102321011706

Byrnes, W. C., & Clarkson, P. M. (1986). Delayed onset muscle soreness and training. *Clinics in Sports Medicine*, *5*(3), 605–614.

Campbell, W. I., & Lewis, S. (1990). Visual analogue measurement of pain. *Ulster Medical Journal*, *59*(2), 149–154.

Carvalho, N., Puntel, G., Correa, P., Gubert, P., Amaral, G., Morais, J., … Soares, F. (2010). Protective effects of therapeutic cold and heat against the oxidative damage induced by a muscle strain injury in rats. *Journal of Sports Sciences*, *28*(9), 923–935. http://doi.org/10.1080/02640414.2010.481722

Cepeda, M. S., Africano, J. M., Polo, R., Alcala, R., & Carr, D. B. (2003). What decline in pain intensity is meaningful to patients with acute pain? *Pain*, *105*(2003), 151–157. http://doi.org/10.1016/S0304-3959(03)00176-3

Chao, D., Foy, C. G., & Farmer, D. (2000). Exercise adherence among older adults: challenges and strategies. *Controlled Clinical Trials*, *21*(5), 212S–217S. http://doi.org/10.1016/S0197-2456(00)00081-7

Chazaud, B., Brigitte, M., Yacoub-Youssef, H., Arnold, L., Gherardi, R., Sonnet, C., … Chretien, F. (2009). Dual and beneficial roles of macrophages during skeletal muscle regeneration. *Exercise and Sport Sciences Reviews*, *37*(1), 18–22. http://doi.org/10.1097/JES.0b013e318190ebdb

Chazaud, B., Sonnet, C., Lafuste, P., Bassez, G., Rimaniol, A. C., Poron, F., … Gherardi, R. K. (2003). Satellite cells attract monocytes and use macrophages as a support to escape apoptosis and enhance muscle growth. *Journal of Cell Biology*, *163*(5), 1133–1143.

http://doi.org/10.1083/jcb.200212046

Chen, A., Arany, P. R., Huang, Y.-Y., Tomkinson, E. M., Sharma, S. K., Kharkwal, G. B., … Hamblin, M. R. (2011). Low-level laser therapy activates NF-κB via generation of reactive oxygen species in mouse embryonic fibroblasts. *PloS One*, *6*(7), 1–8. http://doi.org/10.1371/journal.pone.0022453

Chen, A. H., Frangos, S. G., Kilaru, S., & Sumpio, B. E. (2001). Intermittent pneumatic compression devices - physiological mechanisms of action. *European Journal of Vascular and Endovascular Surgery*, *21*(5), 383–92. http://doi.org/10.1053/ejvs.2001.1348

Chen, L.-E., Liu, K., Qi, W.-N., Joneschild, E., Tan, X., Seaber, A. V, … Urbaniak, J. R. (2002). Role of nitric oxide in vasodilation in upstream muscle during intermittent pneumatic compression. *Journal of Applied Physiology*, *92*(2), 559–566. http://doi.org/10.1152/japplphysiol.00365.2001

Chen, Q., Fisher, D. T., Clancy, K. a, Gauguet, J.-M. M., Wang, W.-C., Unger, E., … Evans, S. S. (2006). Fever-range thermal stress promotes lymphocyte trafficking across high endothelial venules via an interleukin 6 trans-signaling mechanism. *Nature Immunology*, *7*(12), 1299–1308. http://doi.org/10.1038/ni1406

Chen, T. C., Chen, H. L., Lin, M. J., Yu, H. I., & Nosaka, K. (2016). Contralateral repeated bout effect of eccentric exercise of the elbow flexors. *Medicine and Science in Sports and Exercise*, *48*(10), 2030–2039. http://doi.org/10.1249/MSS.0000000000000991

Chen, T. C., & Hsieh, S. S. (2001). Effects of a 7-day eccentric training period on muscle damage and inflammation. *Medicine & Science in Sports & Exercise*, *33*(10), 1732–1738. http://doi.org/10.1097/00005768-200110000-00018

Chensue, S. W., & Kunkel, S. L. (1983). Arachidonic acid metabolism and macrophage activation. *Clinics in Laboratory Medicine*, *3*(4), 677–694.

Cheung, K., Hume, P. A., & Maxwell, L. (2003). Delayed onset muscle soreness: Treatment strategies and performance factors. *Sports Medicine*, *33*(2), 145–164.

Cinamon, G., Shinder, V., & Alon, R. (2001). Shear forces promote lymphocyte migration across vascular endothelium bearing apical chemokines. *Nature Immunology*, *2*(6), 515–522. http://doi.org/10.1038/88710

Clarkson, P. M., & Hubal, M. J. (2002). Exercise-induced muscle damage in humans. *American Journal of Physical Medicine & Rehabilitation*, *81*(11), S52–S69. http://doi.org/10.1097/01.PHM.0000029772.45258.43

Clarkson, P. M., & Newham, D. J. (1995). Associations between muscle soreness, damage, and fatigue. In S. C. Gandevia, R. M. Enoka, A. J. McComas, D. G. Stuart, & C. K. Thomas (Eds.), *Fatigue: Neural and Muscular Mechanisms, advances in experimental medicine and biology* (pp. 457–469). New York, NY: Plenum Press.

Clarkson, P. M., & Sayers, S. P. (1999). Etiology of exercise-induced muscle damage. *Journal of Applied Physiology*, *24*(3), 234–248.

Cleary, M. A., Kimura, I. F., Sitler, M. R., & Kendrick, Z. V. (2002). Temporal pattern of the repeated bout effect of eccentric exercise on delayed-onset muscle soreness. *Journal of Athletic Training*, *37*(1), 32–36.

Cochrane, D. J., Booker, H. R., Mundel, T., & Barnes, M. J. (2013). Does intermittent pneumatic leg compression enhance muscle recovery after strenuous eccentric exercise? *International Journal of Sports Medicine*, *34*(11), 969–974. http://doi.org/10.1055/s-0033-1337944

Cockburn, E., Hayes, P. R., French, D. N., Stevenson, E., & St Clair Gibson, A. (2008). Acute milk-based protein-CHO supplementation attenuates exercise-induced muscle damage. *Applied Physiology, Nutrition, and Metabolism*, *33*(4), 775–783. http://doi.org/10.1139/H08-057

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*. (J. Cohen, Ed.)*Statistical Power Analysis for the Behavioral Sciences* (2nd ed., Vol. 2nd). Hillsdale, New Jersey, USA: Lawrence Erlbaum Associates. http://doi.org/10.1234/12345678

Colloca, L., Sigaudo, M., & Benedetti, F. (2008). The role of learning in nocebo and placebo effects. *Pain*, *136*(1), 211–218. http://doi.org/10.1016/j.pain.2008.02.006

Comerota, A. J., & Arbor, A. (2011). Intermittent pneumatic compression: Physiologic and clinical basis to improve management of venous leg ulcers. *Journal of Vascular Surgery*, *53*(4), 1121–1129. http://doi.org/10.1016/j.jvs.2010.08.059

Coratella, G., Chemello, A., & Schena, F. (2015). Muscle damage and repeated bout effect induced by enhanced-eccentric squat exercise. *The Journal of Sports Medicine and Physical Fitness*, *56*(12), 1540–1546.

Cornish, S. M., & Johnson, S. T. (2014). Systemic cytokine response to three bouts of eccentric exercise. *Results in Immunology*, *4*(4), 23–29. http://doi.org/10.1016/j.rinim.2014.04.002

Couper, K. N., Blount, D. G., & Riley, E. M. (2008). IL-10: The master regulator of immunity to infection. *Journal of Immunology*, *180*(9), 5771–5777. http://doi.org/10.4049/jimmunol.180.9.5771

Crane, J. D., Ogborn, D. I., Cupido, C., Melov, S., Hubbard, A., Bourgeois, J. M., & Tarnopolsky, M. A. (2012). Massage therapy attenuates inflammatory signaling after exercise-induced muscle damage. *Science Translational Medicine*, *4*(119), 1–8. http://doi.org/10.1126/scitranslmed.3002882

Cronin, J. B., Green, J. P., Levin, G. T., Brughelli, M. E., & Frost, D. M. (2004). Effect of starting stance on initial sprint performance. *Journal of Strength and Conditioning Research*, *18*(3), 561–566.

D'Agostino, R. B. S., Sullivan, L. M., & Beiser, A. S. (2006). Statistical inference concerning

(μ1-μ2). In *Introductory Applied Biostatistics* (pp. 231–292). Toronto, ON: Thomson Brooks/Cole.

Davis, C. P., Barrett, K., Torre, P., & Wacasey, K. (1996). Serial myoglobin levels for patients with possible myocardial infarction. *Acad Emerg Med*, *3*(6), 590–597. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8727630

De Palma, C., & Clementi, E. (2012). Nitric oxide in myogenesis and therapeutic muscle repair. *Molecular Neurobiology*, *46*(3), 682–692. http://doi.org/10.1007/s12035-012-8311-8

Denegar, C. R., & Perrin, D. H. (1992). Effect of transcutaneous electrical nerve stimulation, cold, and a combination treatment on pain, decreased range of motion, and strength loss associated with delayed onset muscle soreness. *Journal of Athletic Training*, *27*(3), 200–206.

Deng, B., Wehling-Henricks, M., Villalta, S. A., Wang, Y., & Tidball, J. G. (2012). IL-10 triggers changes in macrophage phenotype that promote muscle growth and regeneration. *Journal of Immunology*, *189*(7), 3669–3680. http://doi.org/10.4049/jimmunol.1103180

Dennis, M., Sandercock, P., Reid, J., & Graham, C. (2013). Effectiveness of intermittent pneumatic compression in reduction of risk of deep vein thrombosis in patients who have had a stroke (CLOTS 3): a multicentre randomised controlled trial. *Lancet*, *382*(9891), 516–524. http://doi.org/10.1016/S0140-6736(13)61050-8

Dinarello, C. A. (1997). Role of pro- and anti-inflammatory cytokines during inflammation: Experimental and clinical findings. *Journal of Biological Regulators and Homeostatic Agents*, *11*(3), 91–103.

Doma, K., Schumann, M., Sinclair, W. H., Leicht, A. S., Deakin, G. B., & Häkkinen, K. (2015). The repeated bout effect of typical lower body strength training sessions on sub-maximal running performance and hormonal response. *European Journal of Applied Physiology*, *115*(8), 1789–99. http://doi.org/10.1007/s00421-015-3159-z

Draper, S. N. (2014). *Effects of intermittent pneumatic compression on Delayed Onset Muscle Soreness (DOMS) in long distance runners*. Cleveland State University, Cleveland, Ohio, USA.

Driller, M. W., & Halson, S. L. (2013). The effects of wearing lower body compression garments during a cycling performance test. *International Journal of Sports Physiology and Performance*, *8*(3), 300–306.

Dupler, T. L., Amonette, W. E., Coleman, A. E., Hoffman, J. R., & Wenzel, T. (2010). Anthropometric and performance differences among high-school football players. *Journal of Strength and Conditioning Research*, *24*(8), 1975–1982. http://doi.org/10.1519/JSC.0b013e3181e4f9ec

Earp, J. E., & Newton, R. U. (2012). Advances in electronic timing systems: Considerations for selecting an appropriate timing system. *Journal of Strength and Conditioning Research*,

26(5), 1245–1248. http://doi.org/10.1519/JSC.0b013e3182474436

Edwards, R. R., Doleys, D. M., Fillingim, R. B., & Lowery, D. (2001). Ethnic differences in pain tolerance: clinical implications in a chronic pain population. *Psychosomatic Medicine*, *63*(2), 316–323. http://doi.org/10.1097/00006842-200103000-00018

Elder, C. L., Dahners, L. E., & Weinhold, P. S. (2001). A cyclooxygenase-2 inhibitor impairs ligament healing in the rat. *The American Journal of Sports Medicine*, *29*(6), 801–805.

Ely, M. R., Romero, S. A., Sieck, D. C., Mangum, J. E., Luttrell, M. J., & Halliwill, J. R. (2016). A single dose of histamine-receptor antagonists prior to downhill running alters markers of muscle damage and delayed onset muscle soreness. *Journal of Applied Physiology*, *1240*(541), 1–35. http://doi.org/10.1152/japplphysiol.00518.2016

Eston, R., & Peters, D. (1999). Effects of cold water immersion on the symptoms of exercise-induced muscle damage. *Journal of Sports Sciences*, *17*(3), 231–238. http://doi.org/10.1080/026404199366136

Evans, W. J., Meredith, C. N., Cannon, J. G., Dinarello, C. A., Frontera, W. R., Hughes, V. A., … Knuttgen, H. G. (1986). Metabolic changes following eccentric exercise in trained and untrained men. *Journal of Applied Physiology*, *61*(5), 1864–1868.

Fanelli, G., Zasa, M., Baciarello, M., Mazzani, R., Di Cianni, S., Rossi, M., & Casati, A. (2008). Systemic hemodynamic effects of sequential pneumatic compression of the lower limbs: a prospective study in healthy volunteers. *Journal of Clinical Anesthesia*, *20*(5), 338–342. http://doi.org/10.1016/j.jclinane.2008.02.005

Farrar, J. T., Berlin, J. A., & Strom, B. L. (2003). Clinically important changes in acute pain outcome measures: A validation study. *Journal of Pain and Symptom Management*, *25*(5), 406–411. http://doi.org/10.1016/S0885-3924(03)00162-3

Farrar, J. T., Portenoy, R. K., Berlin, J. A., Kinman, J. L., & Strom, B. L. (2000). Defining the clinically important difference in pain outcome measures. *Pain*, *88*, 287–294.

Febbraio, M. A., & Pedersen, B. K. (2002). Muscle-derived interleukin-6: mechanisms for activation and possible biological roles. *The FASEB Journal*, *16*(11), 1335–1347. http://doi.org/10.1096/fj.01-0876rev

Feldman, J., Stout, N., & Wanchai, A. (2012). Intermittent pneumatic compression therapy: a systematic review. *Lymphology*, *45*(1), 13–25.

Fernandez Robles, C. R., Degnan, M., & Candiotti, K. A. (2012). Pain and genetics. *Current Opinion in Anaesthesiology*, *25*(4), 444–449. http://doi.org/10.1097/ACO.0b013e3283556228

Fialkow, L., Wang, Y., & Downey, G. P. (2007). Reactive oxygen and nitrogen species as signaling molecules regulating neutrophil function. *Free Radical Biology and Medicine*, *42*(2), 153–164. http://doi.org/10.1016/j.freeradbiomed.2006.09.030

Filippin, L. I., Cuevas, M. J., Lima, E., Marroni, N. P., Gonzalez-Gallego, J., & Xavier, R. M. (2011). Nitric oxide regulates the repair of injured skeletal muscle. *Nitric Oxide - Biology and Chemistry*, *24*(1), 43–49. http://doi.org/10.1016/j.niox.2010.11.003

Finger, E. B., Puri, K. D., Alon, R., Lawrence, M. B., von Andrian, U. H., & Springer, T. A. (1996). Adhesion through L-selectin requires a threshold hydrodynamic shear. *Nature*, *379*(6562), 266–269. http://doi.org/10.1038/379266a0

French, D. N., Thompson, K. G., Garland, S. W., Barnes, C. A., Portas, M. D., Hood, P. E., & Wilkes, G. (2008). The effects of contrast bathing and compression therapy on muscular performance. *Medicine and Science in Sports and Exercise*, *40*(7), 1297–306. http://doi.org/10.1249/MSS.0b013e31816b10d5

Fridén, J., & Lieber, R. L. (2001). Eccentric exercise-induced injuries to contractile and cytoskeletal muscle fibre components. *Acta Physiologica Scandinavica*, *171*(3), 321–326. http://doi.org/10.1046/j.1365-201X.2001.00834.x

Fridén, J., Sjöström, M., & Ekblom, B. (1981). A morphological study of delayed muscle soreness. *Experientia*, *37*(5), 506–507. http://doi.org/10.1007/BF01986165

Fry, A., & Kraemer, W. (1991). Physical performance characteristics of American collegiate football players. *Journal of Strength & Conditioning Research*, *5*(3), 126–138.

Gerlach, K. E., White, S. C., Burton, H. W., Dorn, J. M., Leddy, J. J., & Horvath, P. J. (2005). Kinetic changes with fatigue and relationship to injury in female runners. *Medicine and Science in Sports and Exercise*, *37*(5), 657–663. http://doi.org/10.1249/01.MSS.0000158994.29358.71

Gibala, M. J., Little, J. P., Van Essen, M., Wilkin, G. P., Burgomaster, K. A., Safdar, A., … Tarnopolsky, M. A. (2006). Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *The Journal of Physiology*, *575*(3), 901–911.

Gilson, S. F., Saunders, M. J., Moran, C. W., Moore, R. W., Womack, C. J., & Todd, M. K. (2010). Effects of chocolate milk consumption on markers of muscle recovery following soccer training: a randomized cross-over study. *Journal of the International Society of Sports Nutrition*, *7*(19), 1–10. http://doi.org/10.1186/1550-2783-7-19

Glasgow, P. D., Ferris, R., & Bleakley, C. M. (2014). Cold water immersion in the management of delayed-onset muscle soreness: Is dose important? A randomised controlled trial. *Physical Therapy in Sport*, *15*(4), 228–233. http://doi.org/10.1016/j.ptsp.2014.01.002

Glatthorn, J. F., Gouge, S., Nussbaumer, S., Stauffacher, S., Impellizzeri, F. M., & Maffiuletti, N. A. (2011). Validity and reliability of optojump photoelectric cells for estimating vertical jump height. *Journal of Strength and Conditioning Research*, *25*(2), 556–560. http://doi.org/10.1519/JSC.0b013e3181ccb18d

Goto, K., & Morishima, T. (2014). Compression garment promotes muscular strength recovery

after resistance exercise. *Medicine & Science in Sports & Exercise*, *46*(12), 2265–2270. http://doi.org/10.1249/MSS.0000000000000359

Grant, S., Aitchison, T., Henderson, E., Christie, J., Zare, S., McMurray, J., & Dargie, H. (1999). A comparison of the reproducibility and the sensitivity to change of visual analogue scales, Borg scales, and likert scales in normal subjects during submaximal exercise. *Chest*, *116*(5), 1208–1217. http://doi.org/10.1378/chest.116.5.1208

Griesbacher, T., & Lembeck, F. (1987). Effect of bradykinin antagonists on bradykinin-induced plasma extravasation, venoconstriction, prostaglandin E2 release, nociceptor stimulation and contraction of the iris sphincter muscle in the rabbit. *British Journal of Pharmacology*, *92*(2), 333–340.

Grounds, M. D. (1987). Phagocytosis of necrotic muscle in muscle isografts is influenced by the strain, age, and sex of host mice. *The Journal of Pathology*, *153*(1), 71–82. http://doi.org/10.1002/path.1711530110

Guette, M., Gondin, J., Duclay, J., & Martin, A. (2005). Time-of-day effect on vertical jumps performances. In *Proceedings of the 10th Annual Congress of the European College of Sport Science*. Belgrade, Serbia: European College of Sport Science.

Gulick, D. T., & Kimura, I. F. (1996). Delayed onset muscle soreness: What is it and how do we treat it? *Journal of Sport Rehabilitation*, *5*(3), 234–243.

Harman, E., & Garhammer, J. (2008). Administration, scoring, and interpretation of selected tests. In T. R. Baechle & R. W. Earle (Eds.), *NSCA - Essentials of Strength Training and Conditioning* (3rd ed., pp. 249–294). Windsor, ON: Human Kinetics.

Hasson, S. M., Daniels, J. C., Divine, J. G., Niebuhr, B. R., Richmond, S., Stein, P. G., & Williams, J. H. (1993). Effects of ibuprofen use on muscle soreness, damage, and performance: a preliminary investigation. *Medicine & Science in Sports & Exercise*, *7*(1), 9–17.

Haugen, T. A., & Buchheit, M. (2015). Sprint running performance monitoring: methodological and practical considerations. *Sports Medicine*, *46*(5), 641–656. http://doi.org/10.1007/s40279-015-0446-0

Haugen, T. A., Tønnessen, E., & Seiler, S. K. (2012). The difference is in the start: impact of timing and start procedure on sprint running performance. *Journal of Strength and Conditioning Research / National Strength & Conditioning Association*, *26*(2), 473–479. http://doi.org/10.1519/JSC.0b013e318226030b

Hill, C. A., Thompson, M. W., Ruell, P. A., Thom, J. M., & White, M. J. (2001). Sarcoplasmic reticulum function and muscle contractile character following fatiguing exercise in humans. *Journal of Physiology*, *531*(3), 871–878. http://doi.org/10.1111/j.1469-7793.2001.0871h.x

Hilmersson, M., Edvardsson, I., & Tornberg, Å. B. (2015). Power of counter movement jumps with external load - coherence of three assessment methods. *BMC Research Notes*, *8*(1), 1–

7. http://doi.org/10.1186/s13104-015-1122-z

Hirose, L., Nosaka, K., Newton, M., Laveder, A., Kano, M., Peake, J., & Suzuki, K. (2004). Changes in inflammatory mediators following eccentric exercise of the elbow flexors. *Exercise Immunology Review*, *10*, 75–90.

Ho, K. M., & Tan, J. A. (2013). Stratified meta-analysis of intermittent pneumatic compression of the lower limbs to prevent venous thromboembolism in hospitalized patients. *Circulation*, *128*(9), 1003–1020. http://doi.org/10.1161/CIRCULATIONAHA.113.002690

Hoffman, J. R., Maresh, C. M., Newton, R. U., Rubin, M. R., French, D. N., Volek, J. S., … Kraemer, W. J. (2002). Performance, biochemical, and endocrine changes during a competitive football game. *Medicine and Science in Sports and Exercise*, *34*(11), 1845–1853. http://doi.org/10.1097/00005768-200211000-00023

Howatson, G., & Van Someren, K. A. (2003). Ice massage: Effects on exercise-induced muscle damage. *Journal of Sports Medicine and Physical Fitness*, *43*(4), 500–505.

Hughes, W., & Gosselin, L. E. (2002). Impact of endurance concentric contraction training on acute force deficit following in vitro lengthening contractions. *European Journal of Applied Physiology*, *87*(3), 283–289. http://doi.org/10.1007/s00421-002-0635-z

Hurme, T., & Kalimo, H. (1992). Activation of myogenic precursor cells after muscle injury. *Medicine and Science in Sports and Exercise*, *24*(2), 197–205.

Ihalainen, J., Walker, S., Paulsen, G., Häkkinen, K., Kraemer, W. J., Hämäläinen, M., … Mero, A. A. (2014). Acute leukocyte, cytokine and adipocytokine responses to maximal and hypertrophic resistance exercise bouts. *European Journal of Applied Physiology*, *114*(12), 2607–2616. http://doi.org/10.1007/s00421-014-2979-6

Izquierdo, M., Ibañez, J., Calbet, J. a L., Navarro-Amezqueta, I., González-Izal, M., Idoate, F., … Gorostiaga, E. M. (2009). Cytokine and hormone responses to resistance training. *European Journal of Applied Physiology*, *107*(4), 397–409. http://doi.org/10.1007/s00421-009-1139-x

Jakeman, J. R., Byrne, C., & Eston, R. G. (2010). Lower limb compression garment improves recovery from exercise-induced muscle damage in young, active females. *European Journal of Applied Physiology*, *109*(6), 1137–1144. http://doi.org/10.1007/s00421-010-1464-0

Jamurtas, A. Z., Fatouros, I. G., Buckenmeyer, P., Kokkinidis, E., Taxildaris, K., Kambas, A., & Kyriazis, G. (2000). Effects of plyometric exercise on muscle soreness and plasma creatine kinase levels and its comparison with eccentric and concentric exercise. *Journal of Strength and Conditioning Research*, *14*(1), 68. http://doi.org/10.1519/1533-4287(2000)014<0068:EOPEOM>2.0.CO;2

Järvinen, T. A. H. (2005). Muscle injuries: biology and treatment. *American Journal of Sports Medicine*, *33*(5), 745–764. http://doi.org/10.1177/0363546505274714

Järvinen, T. A. H., Järvinen, M., & Kalimo, H. (2013). Regeneration of injured skeletal muscle after the injury. *Muscles, Ligaments and Tendons Journal*, *3*(4), 337–45. http://doi.org/10.11138/mltj/2013.3.4.337

Jensen, M. P., & Karoly, P. (1992). Self-report scales and procedures for assessing pain in adults. In D. Turk & R. Melzack (Eds.), *Handbook of Pain Assessment* (pp. 135–151). New York, NY: Guilford Press. http://doi.org/10.1002/car.1158

Johnston, M. J., Cook, C. J., Drake, D., Costley, L., Johnston, J. P., & Kilduff, L. P. (2016). The neuromuscular, biochemical and endocrine responses to a single session verses double session training day in elite athletes. *Journal of Strength and Conditioning Research*. http://doi.org/10.1519/JSC.0000000000001423

Jung, U., Norman, K. E., Scharffetter-Kochanek, K., Beaudet, A. L., & Ley, K. (1998). Transit time of leukocytes rolling through venules controls cytokine- induced inflammatory cell recruitment in vivo. *Journal of Clinical Investigation*, *102*(8), 1526–1533. http://doi.org/10.1172/JCI119893

Kanda, K., Sugama, K., Hayashida, H., Sakuma, J., Kawakami, Y., Miuea, S., … Suzuki, K. (2013). Eccentric exercise-induced delayed-onset muscle soreness and changes in markers of muscle damage and inflammation. *Exercise Immunology Review*, *19*(1), 72–85.

Kanda, K., Sugama, K., Sakuma, J., Kawakami, Y., & Suzuki, K. (2014). Evaluation of serum leaking enzymes and investigation into new biomarkers for exercise induced muscle damage. *Exercise Immunology Review*, *20*, 39–54.

Kellmann, M. (2002). Underrecovery and overtraining: Different concepts - similar impact? In M. Kellmann (Ed.), *Enhancing Recovery: Preventing underperformance in athletes* (pp. 3–24). Champaign, IL: Human Kinetics.

Kelly, A. M. (2001). The minimum clinically significant difference in visual analogue scale pain score does not differ with severity of pain. *Emergency Medicine Journal : EMJ*, *18*(3), 205–207. http://doi.org/10.1136/emj.18.3.205

Khamwong, P., Pirunsan, U., & Paungmali, A. (2011). A prophylactic effect of proprioceptive neuromuscular facilitation (PNF) stretching on symptoms of muscle damage induced by eccentric exercise of the wrist extensors. *Journal of Bodywork and Movement Therapies*, *15*(4), 507–16. http://doi.org/10.1016/j.jbmt.2010.07.006

Kharraz, Y., Guerra, J., Mann, C. J., Serrano, A. L., & Muñoz-Cánoves, P. (2013). Macrophage plasticity and the role of inflammation in skeletal muscle repair. *Mediators of Inflammation*, *2013*, 1–9. http://doi.org/10.1155/2013/491497

Kleiner, G., Marcuzzi, A., Zanin, V., Monasta, L., & Zauli, G. (2013). Cytokine levels in the serum of healthy subjects. *Mediators of Inflammation*, *2013*, 1–6. http://doi.org/10.1155/2013/434010

Knight, K. L., & Londeree, B. R. (1980). Comparison of blood flow in the ankle of uninjured

subjects during therapeutic applications of heat, cold, and exercise. *Medicine and Science in Sports and Exercise*, *12*(1), 76–80.

Koch, A. J., Pereira, R., & Machado, M. (2014). The creatine kinase response to resistance exercise. *Journal of Musculoskeletal & Neuronal Interactions*, *14*(1), 68–77.

Kojima, A., Goto, K., Morioka, S., Naito, T., Akema, T., Fujiya, H., … Yoshioka, T. (2007). Heat stress facilitates the regeneration of injured skeletal muscle in rats. *Journal of Orthopaedic Science*, *12*(1), 74–82. http://doi.org/10.1007/s00776-006-1083-0

Kolaczkowska, E., & Kubes, P. (2013). Neutrophil recruitment and function in health and inflammation. *Nature Reviews. Immunology*, *13*(3), 159–175. http://doi.org/10.1038/nri3399

Kolari, P. J., Pekanmäki, K., & Pohjola, R. T. (1988). Transcutaneous oxygen tension in patients with post-thrombotic leg ulcers: treatment with intermittent pneumatic compression. *Cardiovascular Research*, *22*(2), 138–141. http://doi.org/10.1093/cvr/22.2.138

Kraemer, W. J., Bush, J. A., Wickham, R. B., Denegar, C. R., Gómez, A. L., Gotshalk, L. A., … Sebastianelli, W. (2001). Influence of compression therapy on symptoms following soft tissue injury from maximal eccentric exercise. *Journal of Orthopaedic & Sports Physical Therapy*, *31*(6), 282–290.

Kraemer, W. J., Spiering, B. a, Volek, J. S., Martin, G. J., Howard, R. L., Ratamess, N. a, … Maresh, C. M. (2009). Recovery from a national collegiate athletic association division I football game: muscle damage and hormonal status. *Journal of Strength and Conditioning Research / National Strength & Conditioning Association*, *23*(1), 2–10. http://doi.org/10.1519/JSC.0b013e31819306f2

Kreher, J. B., & Schwartz, J. B. (2012). Overtraining syndrome: a practical guide. *Sports Health*, *4*(2), 128–38. http://doi.org/10.1177/1941738111434406

Kuang, S., Gillespie, M. A., & Rudnicki, M. A. (2008). Niche regulation of muscle satellite cell self-renewal and differentiation. *Cell Stem Cell*, *2*(1), 22–31. http://doi.org/10.1016/j.stem.2007.12.012

Kuang, S., Kuroda, K., Le Grand, F., & Rudnicki, M. A. (2007). Asymmetric self-renewal and commitment of satellite stem cells in muscle. *Cell*, *129*(5), 999–1010. http://doi.org/10.1016/j.cell.2007.03.044

Labropoulos, N., Cunningham, J., Kang, S. S., Mansour, M. A., & Baker, W. H. (2010). Optimising the performance of intermittent pneumatic compression devices. *European Journal of Vascular & Endovascular Surgery*, *19*(6), 593–597. http://doi.org/10.1053/ejvs.2000.1067

Labropoulos, N., Wierks, C., & Suffoletto, B. (2002). Intermittent pneumatic compression for the treatment of lower extremity arterial disease: a systematic review. *Vascular Medicine*, *7*(2), 141–148. http://doi.org/10.1191/1358863x02vm423oa

Lapointe, B. M., Frenette, J., & Côté, C. H. (2002). Lengthening contraction-induced inflammation is linked to secondary damage but devoid of neutrophil invasion. *Journal of Applied Physiology*, *92*(5), 1995–2004. http://doi.org/10.1152/japplphysiol.00803.2001

Lau, W. Y., Muthalib, M., & Nosaka, K. (2013). Visual analog scale and pressure pain threshold for delayed onset muscle soreness assessment. *Journal of Musculoskeletal Pain*, *21*(4), 320–326. http://doi.org/10.3109/10582452.2013.848967

Lawrence, M. B., Kansas, G. S., Kunkel, E. J., & Ley, K. (1997). Threshold levels of fluid shear promote leukocyte adhesion through selectins (CD62L,P,E). *Journal of Cell Biology*, *136*(3), 717–727. http://doi.org/10.1083/jcb.136.3.717

Lee, J. S., Hobden, E., Stiell, I. G., & Wells, G. A. (2003). Clinically important change in the visual analog scale after adequate pain control. *Academic Emergency Medicine*, *10*(10), 1128–1130. http://doi.org/10.1197/S1069-6563(03)00372-5

Lees, A., Vanrenterghem, J., & De Clercq, D. (2004). Understanding how an arm swing enhances performance in the vertical jump. *Journal of Biomechanics*, *37*(12), 1929–40. http://doi.org/10.1016/j.jbiomech.2004.02.021

Lewis, P. B., Ruby, D., & Bush-Joseph, C. A. (2012). Muscle soreness and delayed-onset muscle soreness. *Clinics in Sports Medicine*, *31*(2), 255–262. http://doi.org/10.1016/j.csm.2011.09.009

Lieber, R. L., & Fridén, J. (1988). Selective damage of fast glycolytic muscle fibres with eccentric contraction of the rabbit tibialis anterior. *Acta Physiologica Scandinavica*, *133*(4), 587–588.

Lightfoot, J. T., Char, D., McDermott, J., & Goya, C. (1997). Immediate postexercise massage does not attenuate delayed onset muscle soreness. *Journal of Strength and Conditioning Research*, *11*(2), 119–124. http://doi.org/10.1519/1533-4287(1997)011<0119:IPMDNA>2.3.CO;2

Liu, K., Chen, L. E., Seaber, A. V., Johnson, G. W., & Urbaniak, J. R. (1999). Intermittent pneumatic compression of legs increases microcirculation in distant skeletal muscle. *Journal of Orthopaedic Research*, *17*(1), 88–95. http://doi.org/10.1002/jor.1100170114

Lockhart, N. C., & Brooks, S. V. (2008). Neutrophil accumulation following passive stretches contributes to adaptations that reduce contraction-induced skeletal muscle injury in mice. *Journal of Applied Physiology*, *104*(4), 1109–1115. http://doi.org/10.1152/japplphysiol.00850.2007

Loke, P., Gallagher, I. J., Nair, M. G., Zang, X., Brombacher, F., Mohrs, M., … Allen, J. E. (2007). Alternative activation is an innate response to injury that requires CD4+ T cells to be sustained during chronic infection. *Journal of Immunology*, *179*(6), 3926–3936. http://doi.org/10.4049/jimmunol.179.6.3926

Lurie, F., Awaya, D. J., Kistner, R. L., & Eklof, B. (2003). Hemodynamic effect of intermittent

pneumatic compression and the position of the body. *Journal of Vascular Surgery*, *37*(1), 137–142. http://doi.org/10.1067/mva.2002.24

Luster, A. D., Alon, R., & von Andrian, U. H. (2005). Immune cell migration in inflammation: present and future therapeutic targets. *Nature Immunology*, *6*(12), 1182–1190. http://doi.org/10.1038/ni1275

MacRae, B. A., Cotter, J. D., & Laing, R. M. (2011). Compression garments and exercise: Garment considerations, physiology and performance. *Sports Medicine*, *41*(10), 815–843.

Malm, C., Sjödin, B., Sjöberg, B., Lenkei, R., Renström, P., Lundberg, I. E., & Ekblom, B. (2004). Leukocytes, cytokines, growth factors and hormones in human skeletal muscle and blood after uphill or downhill running. *The Journal of Physiology*, *556*(3), 983–1000. http://doi.org/10.1113/jphysiol.2003.056598

Malone, M. D., Cisek, P. L., Comerota, A. J., Holland, B., Eid, I. G., & Comerota, A. J. (1999). High-pressure, rapid-inflation pneumatic compression improves venous hemodynamics in healthy volunteers and patients who are post-thrombotic. *Journal of Vascular Surgery*, *29*(4), 593–599. http://doi.org/S074152149900097X [pii]

Markworth, J. F., Vella, L. D., Figueiredo, V. C., & Cameron-Smith, D. (2014). Ibuprofen treatment blunts early translational signaling responses in human skeletal muscle following resistance exercise. *Journal of Applied Physiology*, *117*(1), 20–28. http://doi.org/10.1152/japplphysiol.01299.2013

Massimino, M. L., Rapizzi, E., Cantini, M., Libera, L. D., Mazzoleni, F., Arslan, P., & Carraro, U. (1997). ED2+ macrophages increase selectively myoblast proliferation in muscle cultures. *Biochemical and Biophysical Research Communications*, *235*(3), 754–759. http://doi.org/10.1006/bbrc.1997.6823

McBride, J. M., Blow, D., Kirby, T. J., Haines, T. L., Dayne, A. M., & Triplett, N. T. (2009). Relationship between maximal squat strength and five, ten, and forty yard sprint times. *Journal of Strength and Conditioning Research / National Strength & Conditioning Association*, *23*(6), 1633–1636. http://doi.org/10.1519/JSC.0b013e3181b2b8aa

McGee, K. J., & Burkett, L. N. (2003). The National Football League combine: a reliable predictor of draft status? *Journal of Strength and Conditioning Research / National Strength & Conditioning Association*, *17*(1), 6–11. http://doi.org/10.1519/1533-4287(2003)017<0006:TNFLCA>2.0.CO;2

McHugh, M. P. (2003). Recent advances in the understanding of the repeated bout effect: the protective effect against muscle damage from a single bout of eccentric exercise. *Scandinavian Journal of Medicine & Science in Sports*, *13*(2), 88–97. http://doi.org/2R477 [pii]

Medzhitov, R. (2008). Origin and physiological roles of inflammation. *Nature*, *454*(7203), 428–435. http://doi.org/10.1038/nature07201

Menth-Chiari, W. A., Curl, W. W., Smith, B. P., & Smith, T. L. (1999). Die mikrozirkulation der skelettmuskulatur nach stumpfem weichteiltrauma und kryotherapie: Einfluss auf gewebeperfusion und zellulare entzundungsreaktion. *Unfallchirurg*, *102*(9), 691–699. http://doi.org/10.1007/s001130050467

Mikkelsen, U., & Langberg, H. (2009). Local NSAID infusion inhibits satellite cell proliferation in human skeletal muscle after eccentric exercise. *Journal of Applied Physiology*, *107*(5), 1600–1611. http://doi.org/10.1152/japplphysiol.00707.2009.

Mishra, D. K., Friden, J., Schmitz, M. C., & Lieber, R. L. (1995). Anti-inflammatory medication after muscle injury. A treatment resulting in short-term improvement but subsequent loss of muscle function. *J Bone Joint Surg Am*, *77*(10), 1510–1519.

Moir, G., Shastri, P., & Connaboy, C. (2008). Intersession reliability of vertical jump height in women and men. *Journal of Strength and Conditioning Research*, *22*(6), 1779–1784. http://doi.org/10.1519/JSC.0b013e318185f0df

Molnar, G., Ho, M. L., & Schroedl, N. A. (1996). Evidence for multiple satellite cell populations and a non-myogenic cell type that is regulated differently in regenerating and growing skeletal muscle. *Tissue and Cell*, *28*(5), 547–556. http://doi.org/10.1016/S0040-8166(96)80057-7

Moriyama, T., Higashi, T., Togashi, K., Iida, T., Segi, E., Sugimoto, Y., … Tominaga, M. (2005). Sensitization of TRPV1 by EP1 and IP reveals peripheral nociceptive mechanism of prostaglandins. *Molecular Pain*, *1*(3), 1–13. http://doi.org/10.1186/1744-8069-1-3

Morris, R. J., Giddings, J. C., Ralis, H. M., Jennings, G. M., Davies, D. A., Woodcock, J. P., & Dunstan, F. D. J. (2006). The influence of inflation rate on the hematologic and hemodynamic effects of intermittent pneumatic calf compression for deep vein thrombosis prophylaxis. *Journal of Vascular Surgery*, *44*(11), 1039–1045. http://doi.org/10.1016/j.jvs.2006.06.016

Morris, R. J., & Woodcock, J. P. (2002). Effects of supine intermittent compression on arterial inflow to the lower limb. *Archives of Surgery*, *137*(11), 1269–1273. http://doi.org/10.1001/archsurg.137.11.1269

Mosser, D. M., & Edwards, J. P. (2008). Exploring the full spectrum of macrophage activation. *Nature Reviews. Immunology*, *8*(12), 958–969. http://doi.org/10.1038/nri2448.Exploring

Muluk, S. C., Hirsch, A. T., & Taffe, E. C. (2013). Pneumatic compression device treatment of lower extremity lymphedema elicits improved limb volume and patient-reported outcomes. *European Journal of Vascular & Endovascular Surgery*, *46*(4), 480–487. http://doi.org/10.1016/j.ejvs.2013.07.012

Munk, N., Symons, B., Shang, Y., Cheng, R., & Yu, G. (2012). Noninvasively measuring the hemodynamic effects of massage on skeletal muscle: A novel hybrid near-infrared diffuse optical instrument. *Journal of Bodywork and Movement Therapies*, *16*(1), 22–28. http://doi.org/10.1016/j.jbmt.2011.01.018

Murase, S., Terazawa, E., Queme, F., Ota, H., Matsuda, T., Hirate, K., … Mizumura, K. (2010). Bradykinin and nerve growth factor play pivotal roles in muscular mechanical hyperalgesia after exercise (delayed-onset muscle soreness). *Journal of Neuroscience*, *30*(10), 3752–3761. http://doi.org/10.1523/JNEUROSCI.3803-09.2010

Musarò, A. (2005). Growth factor enhancement of muscle regeneration: A central role of IGF-1. *Archives Italiennes de Biologie*, *143*(1), 243–248.

Neish, A. S., Williams, A. J., Palmer, H. J., Whitley, M. Z., & Collins, T. (1992). Functional analysis of the human vascular cell adhesion molecule 1 promoter. *Journal of Experimental Medicine*, *176*(6), 1583–1593. http://doi.org/10.1084/jem.176.6.1583

Newham, D. J., Mills, K. R., Quigley, B. M., & Edwards, R. H. (1983). Pain and fatigue after concentric and eccentric muscle contractions. *Clinical Science*, *64*(1), 55–62.

Newton, M. J., Morgan, G. T., Sacco, P., Chapman, D. W., & Nosaka, K. (2008). Comparison of responses to strenuous eccentric exercise of the elbow flexors between resistance-trained and untrained men. *Journal of Strength and Conditioning Research*, *22*(2), 597–607. http://doi.org/10.1519/JSC.0b013e3181660003

Nie, J., Tong, T. K., George, K., Fu, F. H., Lin, H., & Shi, Q. (2011). Resting and post-exercise serum biomarkers of cardiac and skeletal muscle damage in adolescent runners. *Scandinavian Journal of Medicine and Science in Sports*, *21*(5), 625–629. http://doi.org/10.1111/j.1600-0838.2010.01096.x

NormaTec. (n.d.-a). NormaTec athlete community page. Retrieved October 29, 2014, from http://www.normatecrecovery.com/athletes.aspx

NormaTec. (n.d.-b). NormaTec Pro Recovery System product page. Retrieved January 10, 2015, from http://www.normatecrecovery.com/mvp-pro.aspx

NormaTec. (n.d.-c). The science behind NormaTec recovery. Retrieved January 29, 2015, from http://www.normatecrecovery.com/science.shtml

Nosaka, K., Sakamoto, K., Newton, M., & Sacco, P. (2001). How long does the protective effect on eccentric exercise-induced muscle damage last? *Med Sci Sports Exerc*, *33*(9), 1490–1495. http://doi.org/10.1097/00005768-199605001-00674

Ode, J. J., Pivarnik, J. M., Reeves, M. J., & Knous, J. L. (2007). Body mass index as a predictor of percent fat in college athletes and nonathletes. *Medicine and Science in Sports and Exercise*, *39*(3), 403–409. http://doi.org/10.1249/01.mss.0000247008.19127.3e

Palisano, R. J. (2011). Beyond $p < .05$: what is the effect size? *Physical & Occupational Therapy in Pediatrics*, *31*(4), 341–344. http://doi.org/10.3109/01942638.2011.622649

Palmer, R. M. J., Ferrige, A. G., & Moncada, S. (1987). Nitric oxide release accounts for the biological activity of endothelium-derived relaxing factor. *Nature*, *327*(6122), 524–526.

Paschalis, V., Giakas, G., Baltzopoulos, V., Jamurtas, A. Z., Theoharis, V., Kotzamanidis, C., & Koutedakis, Y. (2007). The effects of muscle damage following eccentric exercise on gait biomechanics. *Gait & Posture*, *25*(2), 236–242. http://doi.org/10.1016/j.gaitpost.2006.04.002

Paulsen, G., Mikkelsen, U. R., Raastad, T., & Peake, J. M. (2012). Leucocytes, cytokines and satellite cells: what role do they play in muscle damage and regeneration following eccentric exercise. *Exercise Immunology Reviews*, *18*(1), 42–97.

Peake, J. M., Della Gatta, P. A., Suzuki, K., & Nieman, D. C. (2015). Cytokine expression and secretion by skeletal muscle cells: regulatory mechanisms and exercise effects. *Exercise Immunology Reviews*, *21*(1), 8–25.

Peake, J. M., Nosaka, K., & Suzuki, K. (2005). Characterization of inflammatory responses to eccentric exercise in humans. *Exerc Immunol Rev*, *11*(2005), 64–85.

Peake, J. M., Suzuki, K., Wilson, G., Hordern, M., Nosaka, K., Mackinnon, L., & Coombes, J. S. (2005). Exercise-induced muscle damage, plasma cytokines, and markers of neutrophil activation. *Medicine & Science in Sports & Exercise*, *37*(5), 737–745. http://doi.org/10.1249/01.MSS.0000161804.05399.3B

Peake, J. M., Wilson, G., Mackinnon, L., & Coombes, J. S. (2005). Carbohydrate supplementation and alterations in neutrophils, and plasma cortisol and myoglobin concentration after intense exercise. *European Journal of Applied Physiology*, *93*(5), 672–678. http://doi.org/10.1007/s00421-004-1248-5

Pearson, J., Low, D. A., Stöhr, E., Kalsi, K., Ali, L., Barker, H., & González-Alonso, J. (2011). Hemodynamic responses to heat stress in the resting and exercising human leg: insight into the effect of temperature on skeletal muscle blood flow. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology*, *300*(3), R663–R673. http://doi.org/10.1152/ajpregu.00662.2010

Pedersen, B. K., & Febbraio, M. A. (2008). Muscle as an endocrine organ: Focus on muscle-derived interleukin-6. *Physiological Reviews*, *88*(4), 1379–1406. http://doi.org/10.1152/physrev.90100.2007.

Pedersen, B. K., & Hoffman-Goetz, L. (2000). Exercise and the immune system: Regulation, integration, and adaptation. *Physiological Reviews*, *80*(3), 1055–1081.

Pen, L. J., & Fisher, C. A. (1994). Athletes and Pain Tolerance. *Sports Medicine*, *18*(5), 319–329. http://doi.org/10.2165/00007256-199418050-00004

Peterson, J. M., Trappe, T. A., Mylona, E., White, F., Lambert, C. P., Evans, W. J., & Pizza, F. X. (2003). Ibuprofen and acetaminophen: effect on muscle inflammation after eccentric exercise. *Medicine and Science in Sports and Exercise*, *35*(6), 892–896. http://doi.org/10.1249/01.MSS.0000069917.51742.98

Philippou, A., Bogdanis, G., Maridaki, M., Halapas, A., Sourla, A., & Koutsilieris, M. (2009).

Systemic cytokine response following exercise-induced muscle damage in humans. *Clinical Chemistry and Laboratory Medicine : CCLM / FESCC*, *47*(6), 777–782. http://doi.org/10.1515/CCLM.2009.163

Pincivero, D. M., & Bompa, T. O. (1997). A physiological review of American football. *Sports Med*, *23*(4), 247–260.

Pizza, F. X. (2008). Neutrophils and macrophages in muscle damage and repair. In P. M. Tiidus (Ed.), *Skeletal Muscle Damage and Repair* (pp. 49–58). Windsor, ON: Human Kinetics.

Pizza, F. X., Peterson, J. M., Baas, J. H., & Koh, T. J. (2005). Neutrophils contribute to muscle injury and impair its resolution after lengthening contractions in mice. *The Journal of Physiology*, *562*(3), 899–913. http://doi.org/10.1113/jphysiol.2004.073965

Pokora, I., Kempa, K., Chrapusta, S. J., & Langfort, J. (2014). Effects of downhill and uphill exercises of equivalent submaximal intensities on selected blood cytokine levels and blood creatine kinase. *Biology of Sport*, *31*(3), 173–178. http://doi.org/10.5604/20831862.1111434

Poprzęcki, S., Staszkiewicz, A., & Hübner-Woźniak, E. (2004). Effect of eccentric and concentric exercise on plasma creatine kinase (CK) and lactate dehydrogenase (LDH) activity in healthy adults. *Biology of Sport*, *21*(2), 193–203.

Portillo-Soto, A., Eberman, L. E., Demchak, T. J., & Peebles, C. (2014). Comparison of blood flow changes with soft tissue mobilization and massage therapy. *Journal of Alternative and Complementary Medicine*, *20*(12), 932–6. http://doi.org/10.1089/acm.2014.0160

Rantanen, J., Thorsson, O., Wollmer, P., Hurme, T., & Kalimo, H. (1999). Effects of therapeutic ultrasound on the regeneration of skeletal myofibers after experimental muscle injury. *The American Journal of Sports Medicine*, *27*(1), 54–59.

Reilly, T., & Waterhouse, J. (2009). Sports performance: Is there evidence that the body clock plays a role? *European Journal of Applied Physiology*, *106*(3), 321–332. http://doi.org/10.1007/s00421-009-1066-x

Rifkind, J. M., Nagababu, E., Dobrosielski, D. A., Salgado, M. T., Lima, M., Ouyang, P., & Silber, H. A. (2014). The effect of intermittent pneumatic compression of legs on the levels of nitric oxide related species in blood and on arterial function in the arm. *Nitric Oxide*, *40*(2014), 117–122. http://doi.org/10.1016/j.niox.2014.06.007

Rigamonti, E., Zordan, P., Sciorati, C., Rovere-Querini, P., & Brunelli, S. (2014). Macrophage plasticity in skeletal muscle repair. *BioMed Research International*, *2014*, 1–9. http://doi.org/10.1155/2014/560629

Robert, L. (2012). Physiological profile of national-level junior American football players in Australia. *Serbian Journal of Sports Sciences*, *2012*(4), 127–136.

Roberts, L. A., Muthalib, M., Stanley, J., Lichtwark, G. A., Nosaka, K., Coombes, J. S., & Peake, J. M. (2015). Effects of cold water immersion and active recovery on hemodynamics

and recovery of muscle strength following resistance exercise. *American Journal of Physiology - Regulatory, Integrative and Comparative Physiology*, *309*(4), R389–R398. http://doi.org/10.1152/ajpregu.00151.2015

Roberts, L. A., Raastad, T., Markworth, J. F., Figueiredo, V. C., Egner, I. M., Shield, A., … Peake, J. M. (2015). Post-exercise cold water immersion attenuates acute anabolic signalling and long-term adaptations in muscle to strength training. *The Journal of Physiology*, *593*(18), 4285–4300. http://doi.org/10.1113/JP270570

Robson-Ansley, P., Cockburn, E., Walshe, I., Stevenson, E., & Nimmo, M. (2010). The effect of exercise on plasma soluble IL-6 receptor concentration: A dichotomous response. *Exercise Immunology Review*, *16*, 56–76.

Roseguini, B. T., Mehmet Soylu, S., Whyte, J. J., Yang, H. T., Newcomer, S., & Laughlin, M. H. (2010). Intermittent pneumatic leg compressions acutely upregulate VEGF and MCP-1 expression in skeletal muscle. *American Journal of Physiology. Heart and Circulatory Physiology*, *298*(6), H1991–H2000. http://doi.org/10.1152/ajpheart.00006.2010

Rowlands, D. S., Rössler, K., Thorp, R. M., Graham, D. F., Timmons, B. W., Stannard, S. R., & Tarnopolsky, M. A. (2008). Effect of dietary protein content during recovery from high-intensity cycling on subsequent performance and markers of stress, inflammation, and muscle damage in well-trained men. *Applied Physiology, Nutrition, and Metabolism*, *33*(1), 39–51. http://doi.org/10.1139/H07-136

Sawyer, D. T., Ostarello, J. Z., Suess, E. a, & Dempsey, M. (2002). Relationship between football playing ability and selected performance measures. *J Strength Cond Res*, *16*(4), 611–616.

Saxton, J. M., Clarkson, P. M., James, R., Miles, M., Westerfer, M., Clark, S., & Donnelly, A. E. (1995). Neuromuscular dysfunction following eccentric exercise. *Medicine and Science in Sports and Exercise*, *27*(8), 1185–1193.

Schoenfeld, B. J. (2012). The use of nonsteroidal anti-inflammatory drugs for exercise-induced muscle damage: Implications for skeletal muscle development. *Sports Medicine*, *42*(12), 1017–1028. http://doi.org/10.2165/11635190-000000000-00000

Schultz, E. (1996). Satellite cell proliferative compartments in growing skeletal muscles. *Developmental Biology*, *175*(97), 84–94.

Selsby, J. T., Rother, S., Tsuda, S., Pracash, O., Quindry, J., & Dodd, S. L. (2007). Intermittent hyperthermia enhances skeletal muscle regrowth and attenuates oxidative damage following reloading. *Journal of Applied Physiology*, *102*(4), 1702–1707. http://doi.org/10.1152/japplphysiol.00722.2006

Shah, A., Unger, E., Bain, M. D., Bruce, R., Bodkin, J., Ginnetti, J., … Evans, S. S. (2002). Cytokine and adhesion molecule expression in primary human endothelial cells stimulated with fever-range hyperthermia. *International Journal of Hyperthermia*, *18*(6), 534–551. http://doi.org/10.1080/0265673021015784

Shortreed, K., Johnston, A., & Hawke, T. J. (2008). Satellite cells and muscle repair. In P. M. Tiidus (Ed.), *Skeletal Muscle Damage and Repair* (pp. 77–102). Windsor, ON: Human Kinetics.

Sierer, S. P., Battaglini, C. L., Mihalik, J. P., Shields, E. W., & Tomasini, N. T. (2008). The National Football League combine: Performance differences between drafted and nondrafted players entering the 2004 and 2005 drafts. *Journal of Strength & Conditioning Research*, *22*(1), 6–12.

Smith, E. S. J., & Lewin, G. R. (2009). Nociceptors: a phylogenetic view. *Journal of Comparative Physiology A*, *195*(12), 1089–1106. http://doi.org/10.1007/s00359-009-0482-z

Smith, L. L. (1991). Acute inflammation: the underlying mechanism in delayed onset muscle soreness? *Medicine and Science in Sports and Exercise*, *23*(5), 542–551.

Smith, L. L., Anwar, A., Fragen, M., Rananto, C., Johnson, R., & Holbert, D. (2000). Cytokines and cell adhesion molecules associated with high-intensity eccentric exercise. *European Journal of Applied Physiology*, *82*(1), 61–67. http://doi.org/10.1007/s004210050652

Smith, L. L., Fulmer, M. G., Holbert, D., McCammon, M. R., Houmard, J. A., Frazer, D., … Israel, R. G. (1994). The impact of a repeated bout of eccentric exercise on muscular strength, muscle soreness and creatine kinase. *British Journal of Sports Medicine*, *28*(4), 267–271. http://doi.org/10.1136/bjsm.28.4.267

Smith, L. L., Keating, M. N., Holbert, D., Spratt, D. J., McCammon, M. R., Smith, S. S., & Israel, R. C. (1994). The effects of athletic massage on delayed onset muscle soreness, creatine kinase, and neutrophil count: A preliminary report. *Journal of Orthopaedic & Sports Physical Therapy*, *19*(2), 93–99.

Smith, L. L., McKune, A. J., Semple, S. J., Sibanda, E., Steel, H., & Anderson, R. (2007). Changes in serum cytokines after repeated bouts of downhill running. *Applied Physiology, Nutrition, and Metabolism*, *32*(2), 233–240. http://doi.org/10.1139/h06-106

Starkie, R., Ostrowski, S. R., Jauffred, S., Febbraio, M., & Pedersen, B. K. (2003). Exercise and IL-6 infusion inhibit endotoxin-induced TNF-alpha production in humans. *The FASEB Journal : Official Publication of the Federation of American Societies for Experimental Biology*, *17*(8), 884–886. http://doi.org/10.1096/fj.02-0670fje

Staron, R. S., Hikida, R. S., Murray, T. F., Nelson, M. M., Johnson, P., & Hagerman, F. (1992). Assessment of skeletal muscle damage in successive biopsies from strength-trained and untrained men and women. *European Journal of Applied Physiology and Occupational Physiology*, *65*(3), 258–264. http://doi.org/10.1007/BF00705091

Steensberg, A., Fischer, C. P., Keller, C., Møller, K., & Pedersen, B. K. (2003). IL-6 enhances plasma IL-1ra, IL-10, and cortisol in humans. *American Journal of Physiology. Endocrinology and Metabolism*, *285*(2), E433–E437. http://doi.org/10.1152/ajpendo.00074.2003

Stone, M. J., Waterman, M. R., Harimoto, D., & Murray, G. (1977). Serum myoglobin level as diagnostic test in patients with acute myocardial infarction. *British Heart Journal*, *39*(10), 375–380.

Summan, M., Warren, G. L., Mercer, R. R., Chapman, R., Hulderman, T., Van Rooijen, N., & Simeonova, P. P. (2006). Macrophages and skeletal muscle regeneration: a clodronate-containing liposome depletion study. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology*, *290*(6), R1488–R1495. http://doi.org/10.1152/ajpregu.00465.2005

Suzuki, K., Totsuka, M., Nakaji, S., Yamada, M., Liu, Q., Sugawara, K., … Möhlenkamp, S. (1999). Endurance exercise causes interaction among stress hormones, cytokines, neutrophil dynamics, and muscle damage. *Journal of Applied Physiology*, *87*(4), 1360–1367.

Takagi, R., Fujita, N., Arakawa, T., Kawada, S., Ishii, N., & Miki, A. (2011). Influence of icing on muscle regeneration after crush injury to skeletal muscles in rats. *Journal of Applied Physiology (Bethesda, Md. : 1985)*, *110*(2), 382–8. http://doi.org/10.1152/japplphysiol.01187.2010

Talag, T. S. (1973). Residual muscular soreness as influenced by concentric, eccentric, and static contractions. *Research Quarterly*, *44*(4), 458–469. http://doi.org/10.1080/10671188.1973.10615226

Tang, N. K. Y., Salkovskis, P. M., Hodges, A., Wright, K. J., Hanna, M., & Hester, J. (2008). Effects of mood on pain responses and pain tolerance: An experimental study in chronic back pain patients. *Pain*, *138*(2), 392–401. http://doi.org/10.1016/j.pain.2008.01.018

Tatsumi, R., Anderson, J. E., Nevoret, C. J., Halevy, O., & Allen, R. E. (1998). HGF/SF is present in normal adult skeletal muscle and is capable of activating satellite cells. *Developmental Biology*, *194*(1), 114–128. http://doi.org/10.1006/dbio.1997.8803

Tedesco, F. S., Dellavalle, A., Diaz-Manera, J., Messina, G., & Cossu, G. (2010). Repairing skeletal muscle: regenerative potential of skeletal muscle stem cells. *Journal of Clinical Investigation*, *120*(1), 11–19. http://doi.org/10.1172/JCI40373.and

Tesarz, J., Schuster, A. K., Hartmann, M., Gerhardt, A., & Eich, W. (2012). Pain perception in athletes compared to normally active controls: A systematic review with meta-analysis. *Pain*, *153*(6), 1253–1262. http://doi.org/10.1016/j.pain.2012.03.005

Thorsson, O., Lilja, B., Ahlgren, L., Hemdal, B., & Westlin, N. (1985). The effect of local cold application on intramuscular blood flow at rest and after running. *Medicine & Science in Sports & Exercise*, *17*(6), 710–713. http://doi.org/http://dx.doi.org/10.1249/00005768-198512000-00016

Tidball, J. G. (2005). Inflammatory processes in muscle injury and repair. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology*, *288*(2), R345–R353. http://doi.org/10.1152/ajpregu.00454.2004

Tiidus, P. M. (1998). Radical species in inflammation and overtraining. *Canadian Journal of Physiology and Pharmacology*, *76*(5), 533–538. http://doi.org/10.1139/y98-047

Tiidus, P. M. (2010). Skeletal muscle damage and repair: Classic paradigms and recent developments. *Journal of Musculoskeletal Pain*, *18*(4), 396–402. http://doi.org/10.3109/10582452.2010.502620

Tiidus, P. M., & Ianuzzo, C. D. (1982). Effects of intensity and duration of muscular exercise on delayed soreness and serum enzyme activities. *Medicine and Science in Sports and Exercise*, *15*(6), 461–465.

Timmins, R. G., Shield, A. J., Williams, M. D., Lorenzen, C., & Opar, D. A. (2016). Architectural adaptations of muscle to training and injury: a narrative review outlining the contributions by fascicle length, pennation angle and muscle thickness. *British Journal of Sports Medicine*, bjsports-2015-094881. http://doi.org/10.1136/bjsports-2015-094881

Tipton, K. D., Elliott, T. A., Cree, M. G., Aarsland, A. A., Sanford, A. P., & Wolfe, R. R. (2007). Stimulation of net muscle protein synthesis by whey protein ingestion before and after exercise. *American Journal of Physiology. Endocrinology and Metabolism*, *292*(1), E71–E76. http://doi.org/10.1152/ajpendo.00166.2006

Todd, K. H., Funk, K. G., Funk, J. P., & Bonacci, R. (1996). Clinical significance of reported changes in pain severity. *Annals of Emergency Medicine*, *27*(4), 485–489. http://doi.org/10.1016/S0196-0644(96)70238-X

Tomlin, D. L., & Wenger, H. A. (2001). The relationship between aerobic fitness and recovery from high intensity intermittent exercise. *Sports Medicine*, *31*(1), 1–11.

Townsend, C. M., Kahanov, L., & Eberman, L. E. (2014). Creatine kinase and myoglobin as markers of muscle damage in division-1 collegiate football players. *Asian Journal of Sports Medicine*, *5*(2), 1–18.

Trappe, T. A., White, F., Lambert, C. P., Cesar, D., Hellerstein, M., & Evans, W. J. (2002). Effect of ibuprofen and acetaminophen on postexercise muscle protein synthesis. *American Journal of Physiology. Endocrinology and Metabolism*, *282*, E551–E556. http://doi.org/10.1152/ajpendo.00352.2001

Trost, Z., France, C. R., & Thomas, J. S. (2011). Pain-related fear and avoidance of physical exertion following delayed-onset muscle soreness. *Pain*, *152*(7), 1540–1547. http://doi.org/10.1016/j.pain.2011.02.038

Tschakovsky, M. E., Rogers, A. M., Pyke, K. E., Saunders, N. R., Glenn, N., Lee, S. J., … Dwyer, E. M. (2004). Immediate exercise hyperemia in humans is contraction intensity dependent: evidence for rapid vasodilation. *Journal of Applied Physiology (Bethesda, Md. : 1985)*, *96*(2), 639–644. http://doi.org/10.1152/japplphysiol.00769.2003

Tulapurkar, M. E., Almutairy, E. A., Shah, N. G., He, J. R., Puche, A. C., Shapiro, P., … Hasday, J. D. (2012). Febrile-range hyperthermia modifies endothelial and neutrophilic

functions to promote extravasation. *American Journal of Respiratory Cell and Molecular Biology*, *46*(6), 807–814. http://doi.org/10.1165/rcmb.2011-0378OC

Twist, C., & Eston, R. (2005). The effects of exercise-induced muscle damage on maximal intensity intermittent exercise performance. *European Journal of Applied Physiology*, *94*(5), 652–658. http://doi.org/10.1007/s00421-005-1357-9

Unver, S., & Atan, T. (2015). Investigation of the changes in performance measurements based on circadian rhythm. *Anthropologist*, *19*(2), 423–430.

Vaile, J., Halson, S., Gill, N., & Dawson, B. (2008). Effect of hydrotherapy on the signs and symptoms of delayed onset muscle soreness. *European Journal of Applied Physiology*, *102*(4), 447–455. http://doi.org/10.1007/s00421-007-0605-6

Vieira Ramos, G., Pinheiro, C. M., Messa, S. P., Delfino, G. B., Marqueti, R. de C., Salvini, T. de F., & Durigan, J. L. Q. (2016). Cryotherapy reduces inflammatory response without altering muscle regeneration process and extracellular matrix remodeling of rat muscle. *Scientific Reports*, *6*(18525), 1–12. http://doi.org/10.1038/srep18525

Vincent, H. K., & Vincent, K. R. (1997). The effect of training status on the serum creatine kinase response, soreness and muscle function following resistance exercise. *International Journal of Sports Medicine*, *18*(6), 431–437. http://doi.org/10.1055/s-2007-972660

Virgen-Ortiz, A., Marin, J. L., Trujillo, X., Huerta, M., & Muñiz, J. (2008). Sprint training attenuates the deficits of force and dynamic stiffness in rat soleus muscle caused by eccentric contractions. *Journal of Biomechanics*, *41*(11), 2533–2538. http://doi.org/10.1016/j.jbiomech.2008.05.003

Visconti, L., Capra, G., Carta, G., Forni, C., & Janin, D. (2015). Effect of massage on DOMS in ultramarathon runners: A pilot study. *Journal of Bodywork and Movement Therapies*, *19*(3), 458–463. http://doi.org/10.1016/j.jbmt.2014.11.008

Waller, T., Caine, M., & Morris, R. (2006). Intermittent pneumatic compression technology for sports recovery. *The Engineering of Sport*, *3*, 391–396. http://doi.org/10.1007/978-0-387-45951-6_70

Walsh, N. P., Gleeson, M., Pyne, D. B., Nieman, D. C., Dhabhar, S., Shephard, R. J., … Bermon, S. (2011). Position statement part two: Maintaining immune health. *Exercise Immunology Review*, *17*, 64–103.

Walsh, N. P., Gleeson, M., Shephard, R. J., Jeffrey, M. G., Woods, A., Bishop, N. C., … Rogers, C. J. (2011). Position statement part one : Immune function and exercise. *Exercise Immunology Review*, *17*(1), 6–63.

Wang, H., Melton, D. W., Porter, L., Sarwar, Z. U., McManus, L. M., & Shireman, P. K. (2014). Altered macrophage phenotype transition impairs skeletal muscle regeneration. *American Journal of Pathology*, *184*(4), 1167–1184. http://doi.org/10.1016/j.ajpath.2013.12.020

Warren, G. L., Lowe, D. A., & Armstrong, R. B. (1999). Measurement tools used in the study of eccentric contraction-induced injury. *Sports Medicine*, *27*(1), 43–59.

Westermann, S., Vollmar, B., Thorlacius, H., & Menger, M. D. (2000). Surface cooling inhibits tumor necrosis factor-α – induced microvascular perfusion failure , leukocyte adhesion , and apoptosis in the striated muscle. *Surgery*, 881–889.

White, G. E., & Wells, G. D. (2013). Cold-water immersion and other forms of cryotherapy : physiological changes potentially affecting recovery from high-intensity exercise. *Extreme Physiology and Medicine*, *2*(26), 1–11. http://doi.org/10.1186/2046-7648-2-26

Wiener, A., Mizrahi, J., & Verbitsky, O. (2001). Enhancement of tibialis anterior recovery by intermittent sequential pneumatic compression of the legs. *BAM-PADOVA*, *11*(1), 87–90.

Wilcock, I. M., Cronin, J. B., & Hing, W. A. (2006). Physiological response to water immersion: A method for sport recovery? *Sports Medicine*, *36*(9), 747–765. http://doi.org/10.2165/00007256-200636090-00003

Witard, O. C., Turner, J. E., Jackman, S. R., Kies, A. K., Jeukendrup, A. E., Bosch, J. A., & Tipton, K. D. (2014). High dietary protein restores overreaching induced impairments in leukocyte trafficking and reduces the incidence of upper respiratory tract infection in elite cyclists. *Brain, Behavior, and Immunity*, *39*(2014), 211–219. http://doi.org/10.1016/j.bbi.2013.10.002

Wozniak, A. C., & Anderson, J. E. (2007). Nitric oxide-dependence of satellite stem cell activation and quiescence on normal skeletal muscle fibers. *Developmental Dynamics*, *236*(1), 240–250. http://doi.org/10.1002/dvdy.21012

Wu, H.-C., Hsu, W.-H., & Chen, T. (2005). Complete recovery time after exhaustion in high-intensity work. *Ergonomics*, *48*(6), 668–679. http://doi.org/10.1080/00140130500070871

Yago, T., Wu, J., Wey, C. D., Klopocki, A. G., Zhu, C., & McEver, R. P. (2004). Catch bonds govern adhesion through L-selectin at threshold shear. *Journal of Cell Biology*, *166*(6), 913–923. http://doi.org/10.1083/jcb.200403144

Zainuddin, Z., Newton, M., Sacco, P., & Nosaka, K. (2005). Effects of massage on delayed-onset muscle soreness, swelling and recovery of muscle function. *Journal of Athletic Training*, *40*(3), 174–180.

Zaleska, M., Olszewski, W. L., Jain, P., Gogia, S., Rekha, A., Mishra, S., & Durlik, M. (2013). Pressures and timing of intermittent pneumatic compression devices for efficient tissue fluid and lymph flow in limbs with lymphedema. *Lymphatic Research and Biology*, *11*(4), 227–232. http://doi.org/10.1089/lrb.2013.0016

Zareba, P., Wu, C., Agzarian, J., Rodriguez, D., & Kearon, C. (2014). Meta-analysis of randomized trials comparing combined compression and anticoagulation with either modality alone for prevention of venous thromboembolism after surgery. *British Journal of Surgery*, *101*(9), 1053–1062. http://doi.org/10.1002/bjs.9527

Zelikovski, A., Kaye, C. L., Fink, G., Spitzer, S. A., & Shapiro, Y. (1993). The effects of the modified intermittent sequential pneumatic device (MISPD) on exercise performance following an exhaustive exercise bout. *British Journal of Sports Medicine*, *27*(4), 255–259. http://doi.org/10.1136/bjsm.27.4.255

Zhang, C., Li, Y., Wu, Y., Wang, L., Wang, X., & Du, J. (2013). Interleukin-6/signal transducer and activator of transcription 3 (STAT3) pathway is essential for macrophage infiltration and myoblast proliferation during muscle regeneration. *Journal of Biological Chemistry*, *288*(3), 1489–1499. http://doi.org/10.1074/jbc.M112.419788

Zhu, C., Yago, T., Lou, J., Zarnitsyna, V. I., & Rodger, P. (2009). Mechanisms for flow-enhanced cell adhesion. *Annals of Biomedical Engineering*, *36*(4), 604–621. http://doi.org/10.1007/s10439-008-9464-5.Mechanisms

Zuchtriegel, G., Uhl, B., Hessenauer, M. E. T., Kurz, A. R. M., Rehberg, M., Lauber, K., … Reichel, C. A. (2015). Spatiotemporal expression dynamics of selectins govern the sequential extravasation of neutrophils and monocytes in the acute inflammatory response. *Arteriosclerosis, Thrombosis, and Vascular Biology*, *35*(4), 899–910. http://doi.org/10.1161/ATVBAHA.114.305143

APPENDICES

APPENDIX A – Participant Informed Consent Form

UNIVERSITY OF MANITOBA | Faculty of Kinesiology and Recreation Management

102 Frank Kennedy Centre
Winnipeg, Manitoba
Canada R3T 2N2

Consent Form

Title: Intermittent Pneumatic Compression on the Recovery of Performance, Inflammation and Muscle Damage in Collegiate Athletes

Names of Researchers: Jérémie Chase (Primary investigator), Faculty of Kinesiology and Recreation Management, University of Manitoba, phone [redacted] email: [redacted]

Stephen Cornish, Ph.D. (Assistant Professor – Thesis Advisor), Faculty of Kinesiology and Recreation Management, University of Manitoba, phone: [redacted] or [redacted], email: [redacted]

You are being asked to participate in a research study to investigate the effectiveness of intermittent pneumatic compression devices for recovery in sport. This study is being done to compare if there is a difference in performance, soreness, exertion, and blood markers of inflammation and muscle damage between those using, or not using, the devices. For this study you will be randomized to either use the devices, or be a control. After both team practices and strength and conditioning sessions, regardless of group assignment, you will be asked to undergo a 30 minute recovery session.

A total of 30 subjects are expected to participate in this trial.

Before you decide, it is important for you to understand what the research involves. This consent form will tell you about the study, why the research is being done, what will happen to you during the study and the possible benefits, risks and discomforts. If you wish to participate, you will be asked to sign this form. Your participation is entirely voluntary, so it is up to you to decide whether or not to take part in this study. If you do decide to take part in this study, you are free to withdraw at any time without giving any reasons for your decision nor will you

lose the benefit of any medical care to which you are entitled or are presently receiving nor will there be prejudice, penalty or loss of benefits, and if you choose to withdraw. Choosing or refusing to participate in this study will also have no impact on the decisions regarding your participation in competitive games by the coaching staff of your team. Please take time to read the following information carefully and to discuss it with your family, friends, and doctor before you decide.

Purpose of the study: The purpose of this research is to evaluate if there are benefits to using the intermittent pneumatic compression pants on recovery in sport following football training. Specifically, the study will evaluate changes in performance on vertical jump and speed tests, soreness, and on blood markers of inflammation and muscle damage.

Possible benefits of the study: You may see lower soreness and better performance following the recovery session. These results are not guaranteed.

Procedures: You will initially be given a questionnaire that asks whether you are at risk for performing exercise. This will take several minutes to complete. If it is determined you are at risk, we will ask you to obtain permission from your family physician before entering the study.

You will be assigned to one of two groups. The first group will be using the intermittent pneumatic compression pants after a day of training session. The second group will be the control group, and asked to remain seated following the training session. The study uses a randomized crossover design, meaning this intervention will be repeated using the opposite recovery mode a week later.

Each of the recovery treatments will last 30 minutes after the last training session of the day. During this time, you will be welcome to bring some reading material (study notes, books) or electronic devices to keep busy during the treatment session. If you agree to be in the study, we will do the following measurements on you:

1. Body mass, height, resting blood pressure and heart rate will be assessed at baseline one week before participating in the study. You will also be familiarized with the performance tests and the compression devices. This procedure takes about 1 hour.
2. Blood (to assess markers of inflammation and muscle damage) will be taken at baseline at the start of the training day, after the last training session,

immediately after recovery, and at 3 hours and 24 hours after recovery. Approximately 16 mL of (about 3 teaspoons) blood will be collected at each time point. This procedure will take about 5 minutes each time for a total of 20 minutes. Blood sampling will be done by a certified phlebotomist (an individual certified and trained to draw blood) under sterile conditions at each time point.

3. Maximum jump height will be measured using a Quattro force platform. Your time to complete a 10 metre sprint will also be measured, and timed using laser gate systems. This will be done at baseline, and at 3 and 24 hours after the last training session. This should take approximately 2 hours total.
4. Delayed onset muscle soreness will be assessed on visual analog scales. The visual analog scales are 100 mm long lines with the words "no soreness" on the left hand side and "extreme soreness" on the right hand side of the line. You will be asked to draw a vertical line on the scale to rate your current level of pain and exertion. This form will also ask if you have consumed any medication that might influence the results. This procedure will take about 1 minute at each blood draw.

Familiarization (one week prior to the intervention week)

-Resting blood pressure
-Resting heart rate
-Height
-Weight
-PAR-Q+ (pre-screening form)
-Familiarization (Vertical jump, 10m sprint, and IPC device)

Day 1 (Steps in sequential order)

1-Blood draw and DOMS measurement
2-Performance testing
3- Sprint and speed training (afternoon)
4- Strength training (evening)
5-Blood draw and DOMS measurement
6-Recovery session (IPC or control)*
7-Blood draw and DOMS measurement immediately after recovery
8-Blood draw and DOMS measurement 3 hours after step 4
9-Performance testing 3 hours after step 4

Day 2

10-Blood draw and DOMS measurement 24 hours after step 4

11-Performance testing 24 hours after step 4

Crossover – Days 1 and 2 are repeated the following week in a randomized crossover fashion (opposite recovery intervention)

*Recovery sessions will consist of measurements of heart rate and blood pressure, and the 30 minute IPC or control condition.

In addition to the above procedures, you will be asked to fill out a food log that would be started a day before the interventions. You will also be asked to report if you are taking any nutritional supplements during the trial that might influence inflammation. This log should take approximately 15 minutes.

Foreseeable risks, side effects or discomfort: The intermittent pneumatic compression devices might create mild discomfort; however, a familiarization session will occur for you to get use to the devices.

There is a risk of injury and discomfort during exercise testing, but the tests are no more strenuous than if you were to complete them on your own or as part of your training program. This risk will also be minimized with supervision by certified exercise trainers and the familiarization session to the testing protocols.

There might also be a risk of injury occurring during a training session, however, this risk is just as prevalent whether you join the study or not, as it will be the training sessions you would be expected to participate in as part of your role as an athlete.

There will be some discomfort when blood is drawn for testing inflammation levels. Bruising or infection at the sight of the blood draw is a possibility, but care will be taken to minimize these risks by using sterile equipment and procedures.

There may be unforeseen risks during the study or after the study has been completed.

Alternatives to this study: You do not have to participate in this study to use the intermittent pneumatic compression devices. Pending on availability of the intermittent pneumatic compression devices, there might be the option to try them out for a few weeks after the study is complete if you decide not to participate.

They can also be purchased online at the NormaTec website, or local companies Recovery Spot and Massage Athletica offer them on a pay-per use basis. There are also many other alternatives that you can use that might help post-exercise recovery, which you can discuss with the team athletic therapists.

Research-Related Injury: There will be no cost to you for participation in this study. You will not be charged for the research procedures. If you are making a car trip to the University of Manitoba exclusively for this research project, you will be reimbursed for parking fees.

In the event you become ill or injured as a result of participating in this study, acute medical treatment will be made available at no additional cost to you by an individual trained in first aid, CPR-C and AED and, if needed, emergency services for the University of Manitoba and City of Winnipeg will be called in the case of an emergency. By signing this document you do not waive any of your legal rights and it will not initiate prejudice, penalty or loss of benefits to which you are otherwise entitled.

Confidentiality: Precautions will be taken to protect your anonymity during the study. All data collected will be stored in a locked office in the Faculty of Kinesiology and Recreation Management. While absolute confidentiality cannot be guaranteed, every effort will be made to ensure that the information you provide for this study is kept entirely confidential. A participant code will be assigned to identify you on any document or forms used to collect the data. Your name or other identifying traits will not be attached to any information, nor mentioned in any study report, nor be made available to anyone except the research team. To keep your participation confidential to your teammates and coaches, you will not be asked to leave early or in the middle of any training sessions. All study procedures will occur before or after training.

It is the intention of the research team to publish results of this research in scientific journals and to present the findings at related conferences and workshops, but your identity will not be revealed. All results will be presented as averages. Should the coaches or media request the results of the study, only averages will be presented.

If you have questions concerning the study you can contact Jérémie Chase at [redacted] or at [redacted]. Any adverse events you experience during the study should be reported immediately by contacting the primary investigator.

If you have any questions about your rights as a research subject or concerns about the study, please direct questions regarding your rights as a participant in this research to the Human Ethics Coordinator at the Office of Research Ethics and Compliance at the University of Manitoba (Phone: 204-474-7122 or Email: humanethics@umanitoba.ca).

The University of Manitoba may look at the research records to ensure that the study was carried out in a safe and proper manner.

If, during the course of this study, new information becomes available that may be related to your willingness to continue to participate, this information will be provided to you by the investigator. That is, if new research information about intermittent pneumatic compression indicates some negative health affects you will be notified immediately. Also, if you desire to have a copy of your personal results from the study this will be made available to you by adding your email address at the bottom of this form. Further, the complete results of this research will be made available to you if you so desire.

Your participation in the research is entirely voluntary. You are free to withdraw from this study at any time and this withdrawal will not affect your access to health care, team playing time, or other services. This can be done by contacting the primary investigator (Jérémie Chase) at the phone number or email address listed above, or in person. If you choose to enter the study and then decide to withdraw at a later time, a discussion between yourself and the primary investigator will occur to decide what will be done with the data collected during your enrolment in the study.

Please read the following before signing this consent form:

- I have read or have had this read to me and understood the research subject information and consent form.
- I have had sufficient time to consider the information provided and to ask for advice if necessary.
- I have had the opportunity to ask questions and have had satisfactory responses to my questions.
- I understand that all of the information collected will be kept confidential and that the result will only be used for scientific objectives.
- I understand that my participation in this study is voluntary and that I am completely free to refuse to participate or to withdraw from this study at any time without changing in any way the quality of care that I receive.
- I understand that I am not waiving any of my legal rights as a result of signing this consent form.
- I understand that there is no guarantee that this study will provide any benefits to me (if applicable).
- I have read this form and I freely consent to participate in this study.
- I have been told that I will receive a dated and signed copy of this form.

Research Subject's Signature: _____

Date: _____

Primary Investigator Signature: _____

Date: _____

Email address for results: _____

APPENDIX B – Coach Consent Form

102 Frank Kennedy Centre
Winnipeg, Manitoba
Canada R3T 2N2

UNIVERSITY OF MANITOBA | Faculty of Kinesiology and Recreation Management

Consent Form

Title: Intermittent Pneumatic Compression on the Recovery of Performance, Inflammation and Muscle Damage in Football Athletes

Names of Researchers: Jérémie Chase (Primary investigator), Faculty of Kinesiology and Recreation Management, University of Manitoba, phone [redacted], email: [redacted]

Stephen Cornish, Ph.D. (Assistant Professor – Thesis Advisor), Faculty of Kinesiology and Recreation Management, University of Manitoba, phone: [redacted] or [redacted], email: [redacted]

You are being asked permission to allow your team's athletes to be recruited for an off-season research project. The purpose of this study is to investigate the effectiveness of intermittent pneumatic compression devices for recovery in sport. This study is being done to compare if there is a difference in performance, soreness, and blood markers of inflammation and muscle damage between those using the devices and not. Participants will be asked to undergo a 30 minute recovery session after a day of speed and strength training, and physical testing as well as blood draws at various time points following the session.

A total of 30 participants are expected to participate in this trial.

Confidentiality: Precautions will be taken to protect participant anonymity during the study. All data collected will be stored in a locked office in the Faculty of Kinesiology and Recreation Management. Your name or other identifying traits will not be attached to any information, nor mentioned in any study report, nor be made available to anyone except the research team. To protect the participant confidentiality from the coaching staff and non-participating teammates, all study

procedures will occur outside of scheduled training times, and at no point will they be asked to leave early or in the middle of a session.

It is the intention of the research team to publish results of this research in scientific journals and to present the findings at related conferences and workshops, but your identity or information will not be revealed. All results will be presented as averages. Likewise, should you as a coach wish to see the results of the study, all results will be presented as averages to maintain participant anonymity after the study is done.

If you have questions concerning the study you can contact Jérémie Chase at [redacted] or at [redacted].
If you have any questions about your rights, or concerns about the study, please direct questions regarding your rights to the Human Ethics Coordinator at the Office of Research Ethics and Compliance at the University of Manitoba (Phone: 204-474-7122 or Email: humanethics@umanitoba.ca).

Please read the following before signing this consent form:

- I have read or have had this read to me and understood the consent form.
- I have had sufficient time to consider the information provided and to ask for advice if necessary.
- I have had the opportunity to ask questions and have had satisfactory responses to my questions.
- I understand that all of the information collected will be kept confidential and that the result will only be used for scientific objectives.
- I understand that I am not waiving any of my legal rights as a result of signing this consent form.
- I have been told that I will receive a dated and signed copy of this form.

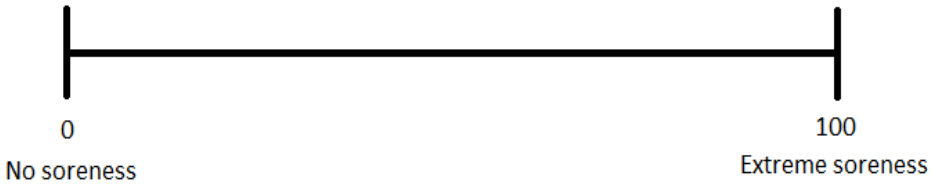
Coach's Signature: _____

Date: _____

Primary Investigator Signature: _____

Date: _____

APPENDIX C – Visual Analogue Scale


0
No soreness
100
Extreme soreness

APPENDIX D – Dietary Tracking Sheet

Three day food log

The food log is meant for you to record your daily food intake for the study before the first intervention. This also includes drinks (alcoholic as well), snacks, and supplements. Not only does it allow the investigators to get an idea of your normal diet, it also makes it easier to replicate the diet for the second intervention. Do not let the diet tracking influence your choice of foods that you eat. All information is confidential and no participant will be identified.

Since diet has an influence on some of the measures taken as part of the study, we ask that you replicate what you had eaten to the best of your ability. This will mean both interventions will be done under the same state. The more detail (time of day, quantity, etc…) you put the easier it will be for you on the second week.

-When logging a meal you ate, always break down each element of it. For example, don't write chicken sandwich. Log two slices of bread, chicken, mayo etc…

-The best way to do it is to log as you go, rather than to remember everything at the end of the night. Keeping quick notes on your phone if the logging sheet is not handy is also an option.

Sample

Date:_____

Time of Day	Food/Drink	Quantity
8:30 AM	Oatmeal	1 cup
	Orange Juice	1 cup
	Scrambled Eggs	2 eggs
	Shredded cheese for eggs	1/4 cup
9:45	Protein Shake, Mixed with water	2 scoops
11:30	Coffee, 1 milk 1 sugar	1 cup
12:15	Sub Sandwich	
	2 slices white bread	2 slices
	Sliced ham -1/4" cut	3 slices
	Lettuce	2 Leaves
	Tomato	2 slices
	Mustard	1 tablespoon

Day 1

Date:______________________________

Time of Day	Food/Drink	Quantity

Day 2

Date:______________________________

Time of Day	Food/Drink	Quantity

APPENDIX E – Ethical Approval Certificate

UNIVERSITY OF MANITOBA | Research Ethics and Compliance
Office of the Vice-President (Research and International)

Human Ethics
208-194 Dafoe Road
Winnipeg, MB
Canada R3T 2N2
Phone +204-474-7122
Fax +204-269-7173

APPROVAL CERTIFICATE

February 25, 2016

TO: **Jeremie Chase** (Supervisor: Stephen Cornish)
Principal Investigator

FROM: **Zana Lutfiyya, Chair**
Education/Nursing Research Ethics Board (ENREB)

Re: **Protocol #E2016:010 (HS19321)**
"Intermittent Pneumatic Compression on the Recovery of Performance, Inflammation and Muscle Damage in Football Athletes"

Please be advised that your above-referenced protocol has received human ethics approval by the **Education/Nursing Research Ethics Board**, which is organized and operates according to the Tri-Council Policy Statement (2). **This approval is valid for one year only and will expire on February 25, 2017**.

Any significant changes of the protocol and/or informed consent form should be reported to the Human Ethics Secretariat in advance of implementation of such changes.

Please note:

- **If you have funds pending human ethics approval, please mail/e-mail/fax (261-0325) a copy of this Approval (identifying the related UM Project Number) to the Research Grants Officer in ORS in order to initiate fund setup. (How to find your UM Project Number: http://umanitoba.ca/research/ors/mrt-faq.html#pr0)**

- **if you have received multi-year funding for this research, responsibility lies with you to apply for and obtain Renewal Approval at the expiry of the initial one-year approval; otherwise the account will be locked.**

The Research Quality Management Office may request to review research documentation from this project to demonstrate compliance with this approved protocol and the University of Manitoba *Ethics of Research Involving Humans*.

The Research Ethics Board requests a final report for your study (available at: http://umanitoba.ca/research/orec/ethics/human_ethics_REB_forms_guidelines.html) **in order to be in compliance with Tri-Council Guidelines.**